“Non-Diabetic Retinal Vascular Disease”

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Retinal Vascular Disease

- No financial disclosures

Hypoperfusion Syndrome

- Occurs when the eye lacks blood perfusion secondary to carotid artery blockage or ophthalmic artery blockage.
- Terminology debate: venous stasis retinopathy vs. hypoperfusion syndrome
- Why is venous stasis retinopathy a poor term for this condition? Because it is an arterial problem, not a venous problem. The venous stasis is the result of poor flow into the arteries

Hypoperfusion Syndrome

- Patient may complain of dull, chronic ache in the affected eye
- Photostress issues / dazzle
- TIA symptoms may or may not be present (amaurosis fugax)
- Possible bruit / decreased pulse strength in carotid

Hypoperfusion Syndrome

- Bruit at 30-85% blockage; swishing sound
- Bell vs. diaphragm
- Definitive diagnosis requires carotid imaging

Hypoperfusion Syndrome

- Peripheral dot / blot hemorrhages
- Dilated veins
- Relatively spares the posterior pole

Ocular Ischemic Syndrome

- With ocular ischemic syndrome same findings plus……..
  - NVD / NVE / NVI
  - Iritis
  - Sluggish pupil
  - Conjunctival congestion
  - Corneal Edema
  - 80% unilateral / 20% bilateral

Ocular Ischemic Syndrome

- Rare! Only 10% of eyes with 70+% blocked carotids
- 60% CF or worse VA by one year: 82% if NVI is present
- Teichopsia: colored afterimages after viewing lights

Ocular Ischemic Syndrome (OIS)

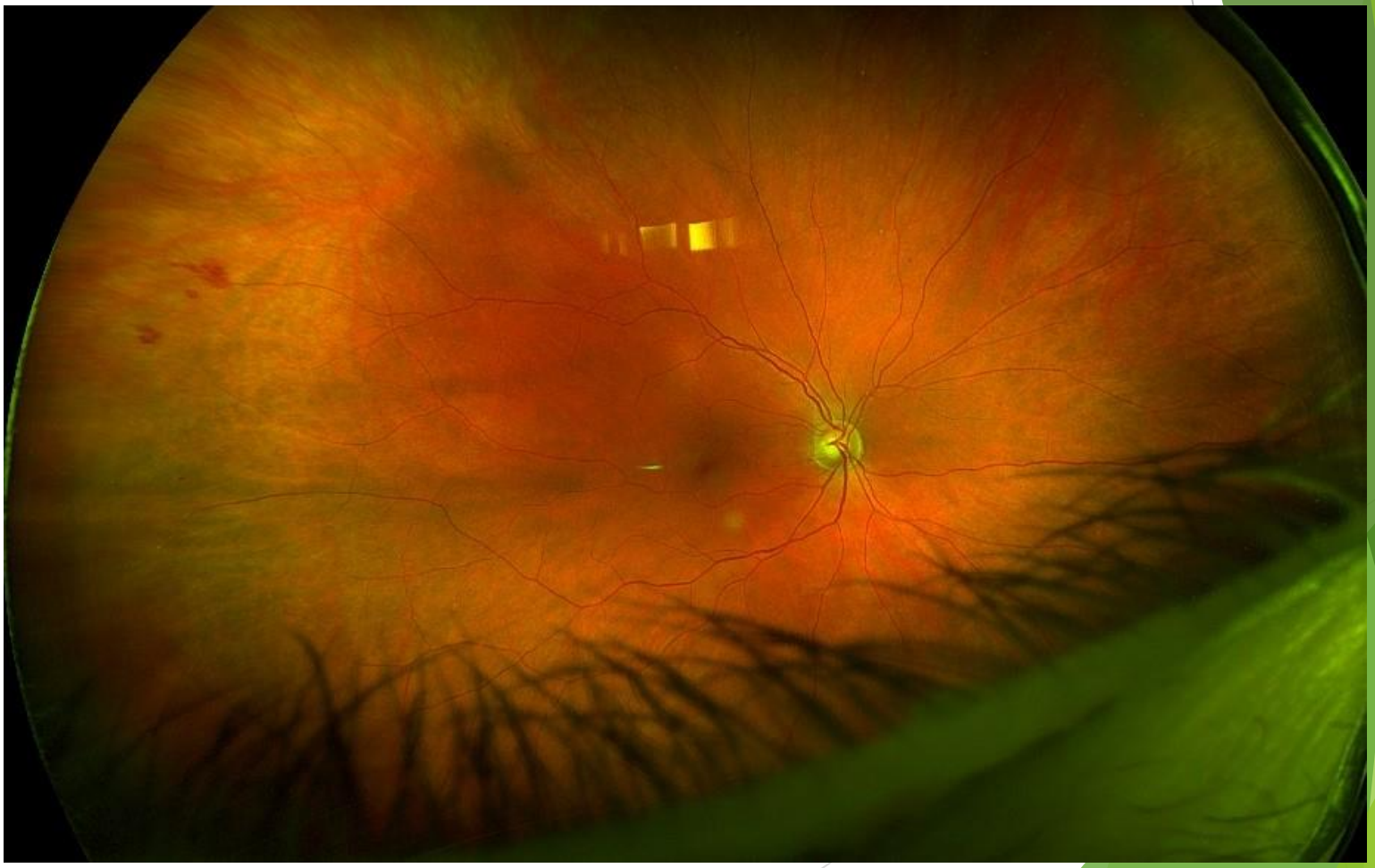
- When presented with these ocular findings…….

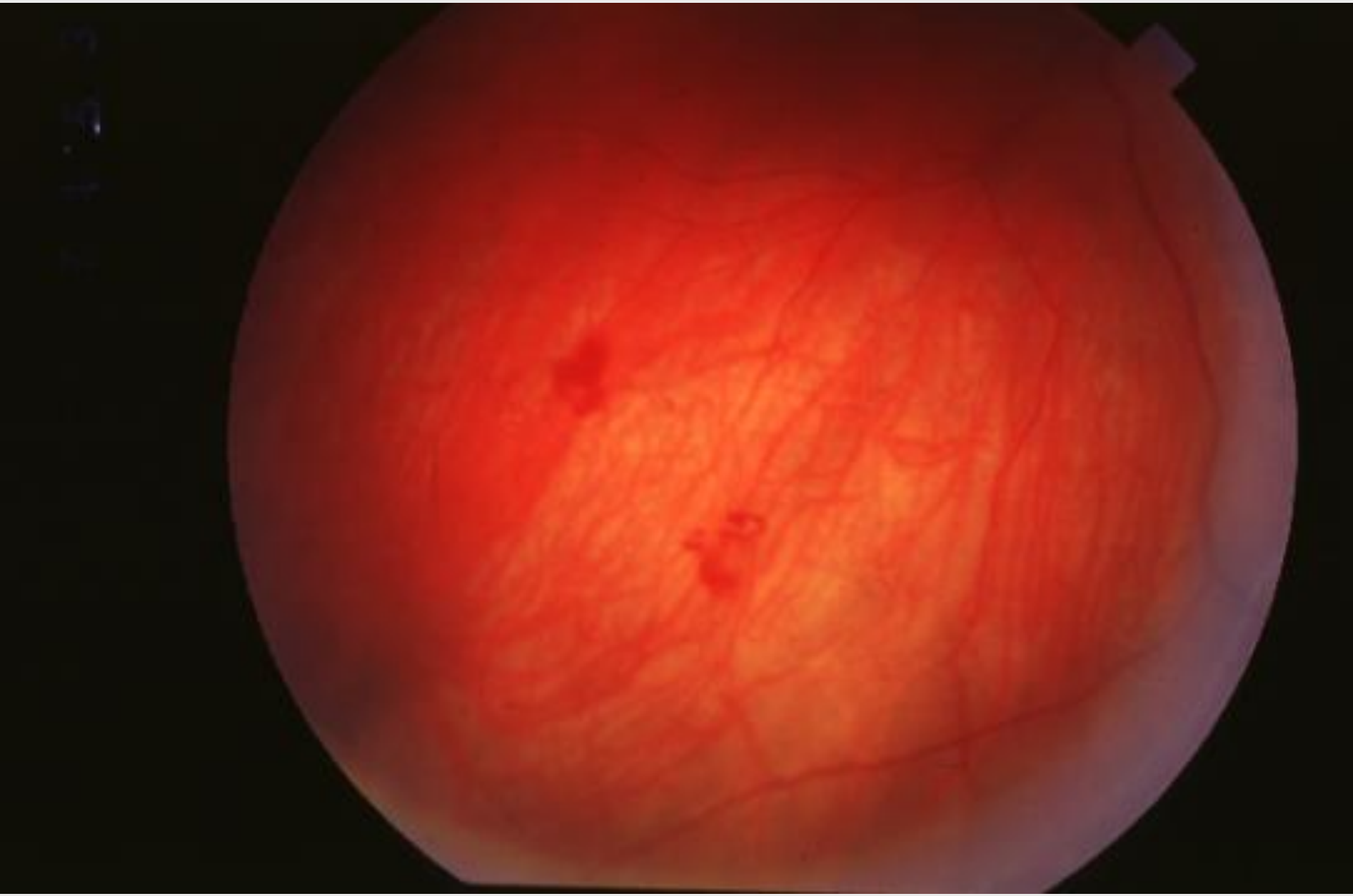
- Question about TIA
- Check carotids
- Arrange for carotid testing (Doppler has limits)
- ESR
- C-reactive protein
- CBC
- Lipid panel

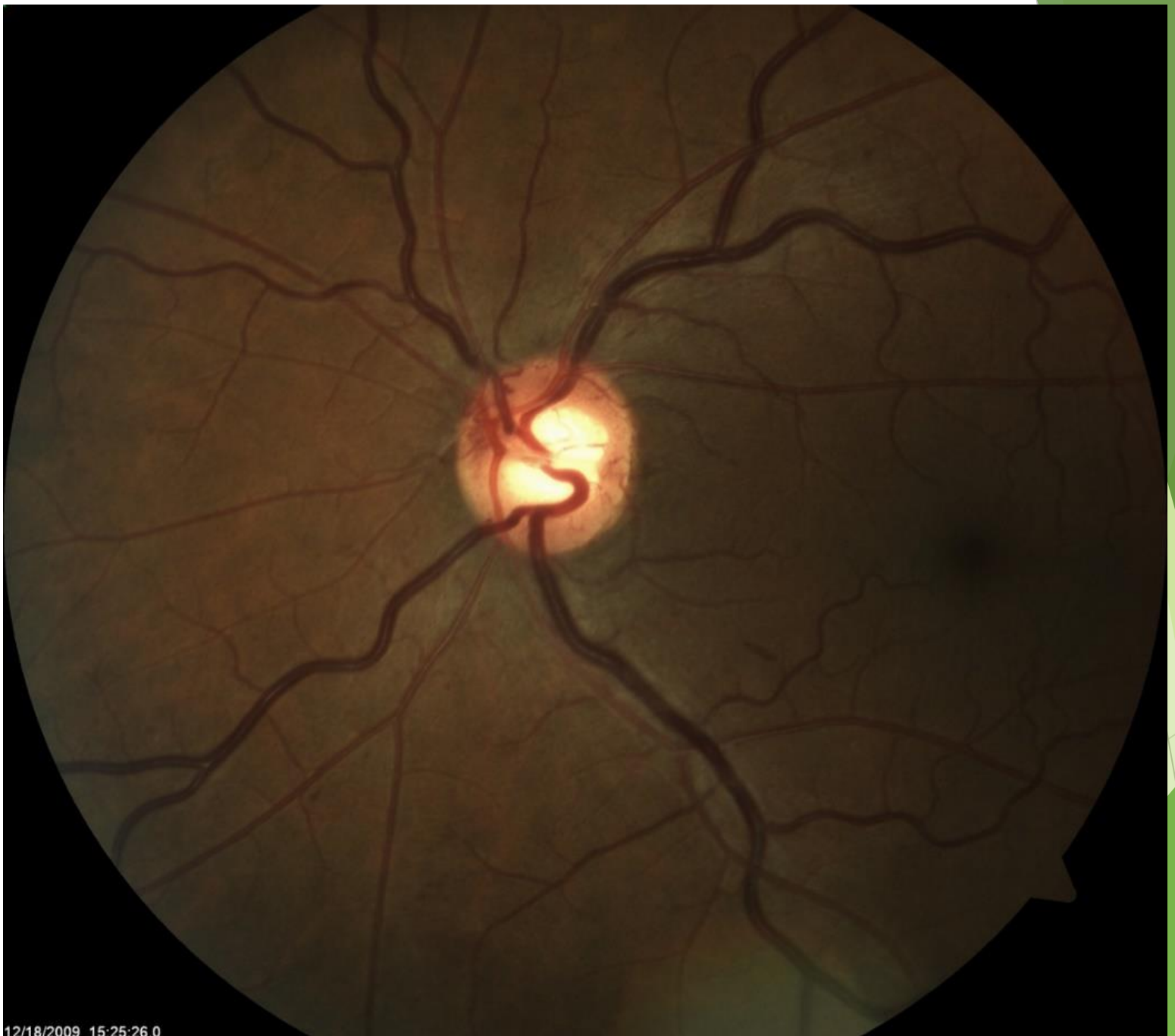
Ocular Ischemic Syndrome

- Treatment:
  - Systemic management (diet, drugs, endarterectomy)
  - PRP / cryotherapy / anti-VEGF injections
  - Five-year mortality rate of 40%

Hypoperfusion syndrome



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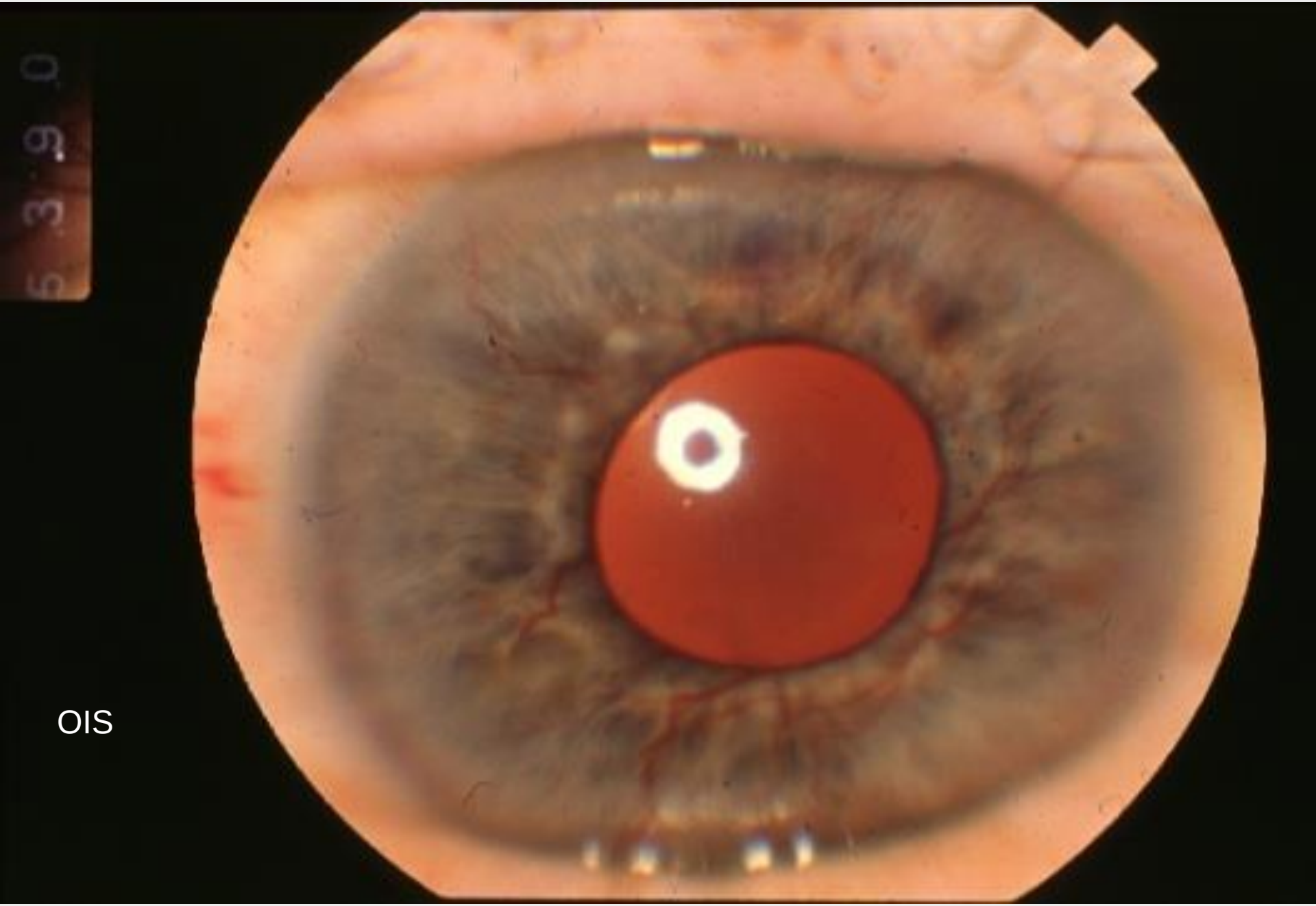


OIS

Sickle Cell Retinopathy

- Hemoglobinopathy affecting mostly AA (8% in US carry trait, .27% have sickle cell disease.)
- About 100,000 people in the US have the disease
- Malaria and natural selection (sickle trait carriers are resistant to malaria)
- AC, SA, SS, Sthal, SC. All forms autosomal recessive. A is normal hemoglobin
- Improper amino acid substitutions

Sickle Cell

- RBC's become sickle shaped
- RBC's get trapped > hypoxia > ischemia > more sickling =cycle (also sickle due to acidosis, hyperosmolarity)
- Splenic crisis common, can be fatal. Many patients need spleenectomy
- SS patients tend to have the worst systemic complications while SC and Sthal patients tend to have the most severe ocular problems. Possibly due to decreased life expectancy with SS

Sickle Cell Retinopathy Stages

- Stage I :peripheral arteriolar occlusion
- Stage II:peripheral arterio-venular anastomoses
- Stage III: Neovascularization
- Stage IV: Vitreous Hemorrhage
- Stage V: Tractional retinal detachment

Sickle Cell Retinopathy

- Pre-proliferative findings…
- Salmon-patch hemes
- Sunburst pigment
- Refractile bodies
- Sclerosis of arterioles
- ERM
- Conjunctival comma sign

- Proliferative findings and others:
- Sea-fan neovascularization
- Vitreous Hemorrhage
- Tractional retinal detachment

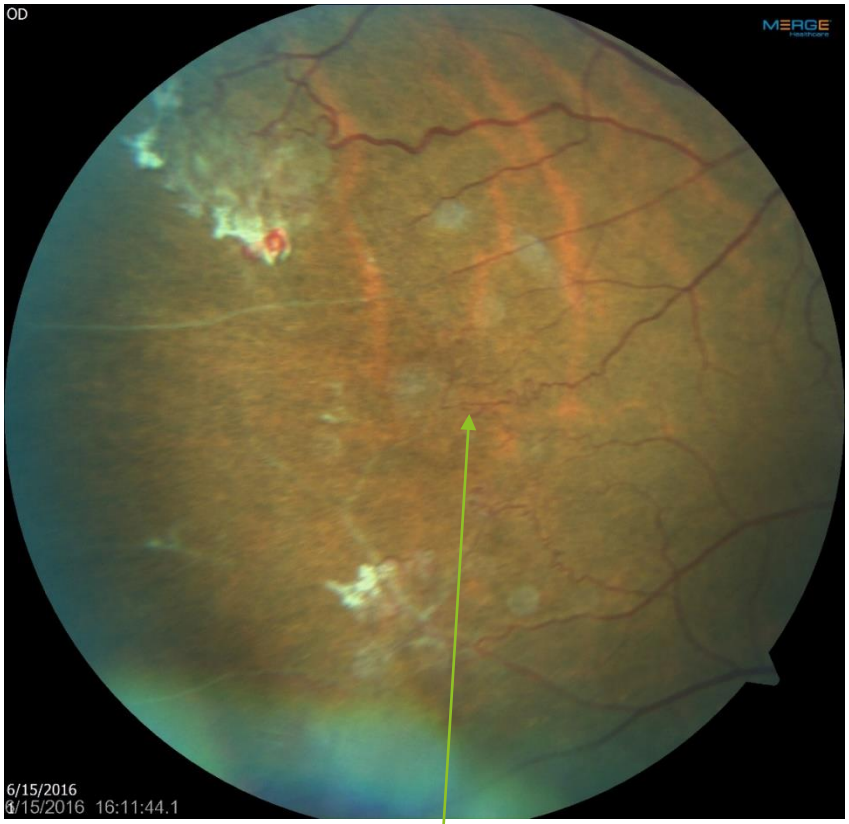
Other considerations:

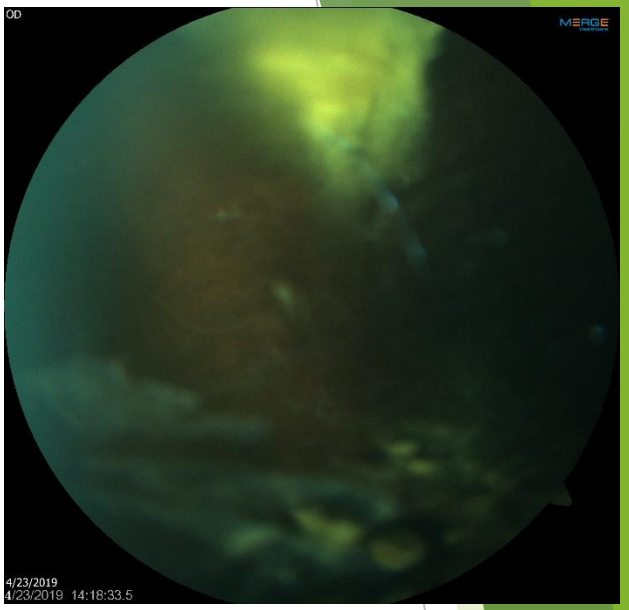
- Angioid streaks
- CRAO / BRAO
- Hyphema issues (24-24 rule)

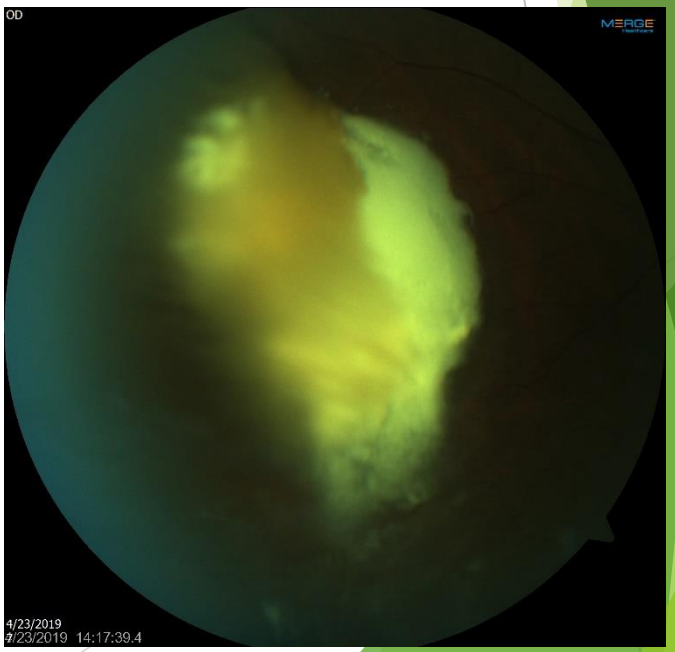
Sickle Cell Retinopathy

- PRP or cryo anterior to sea fans
- Anti-VEGF injections
- Is treatment always necessary? No! The sickled RBC's can occlude the sea fan NV, and cause it to regress

Sea fans: S-Thal





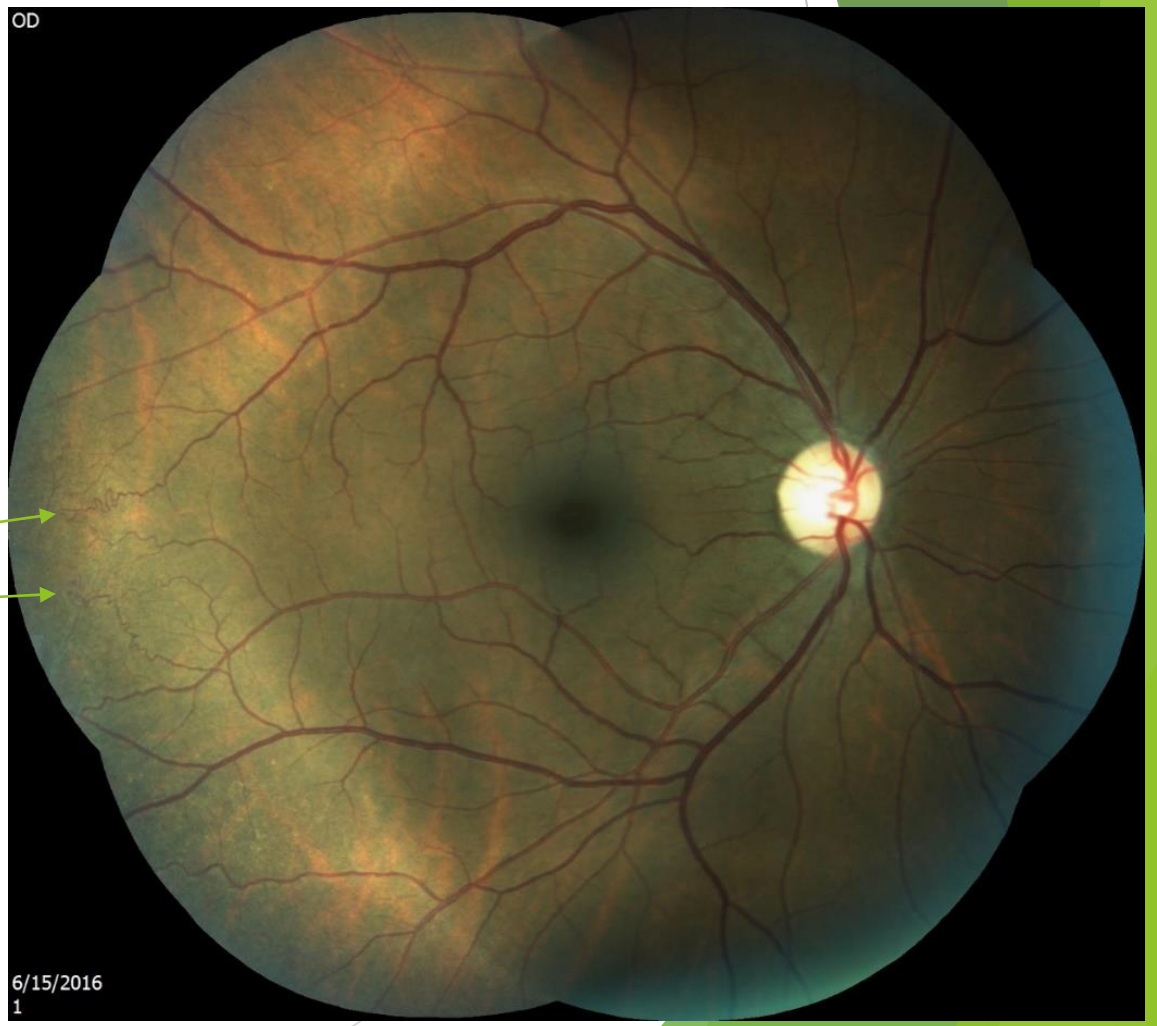


2016

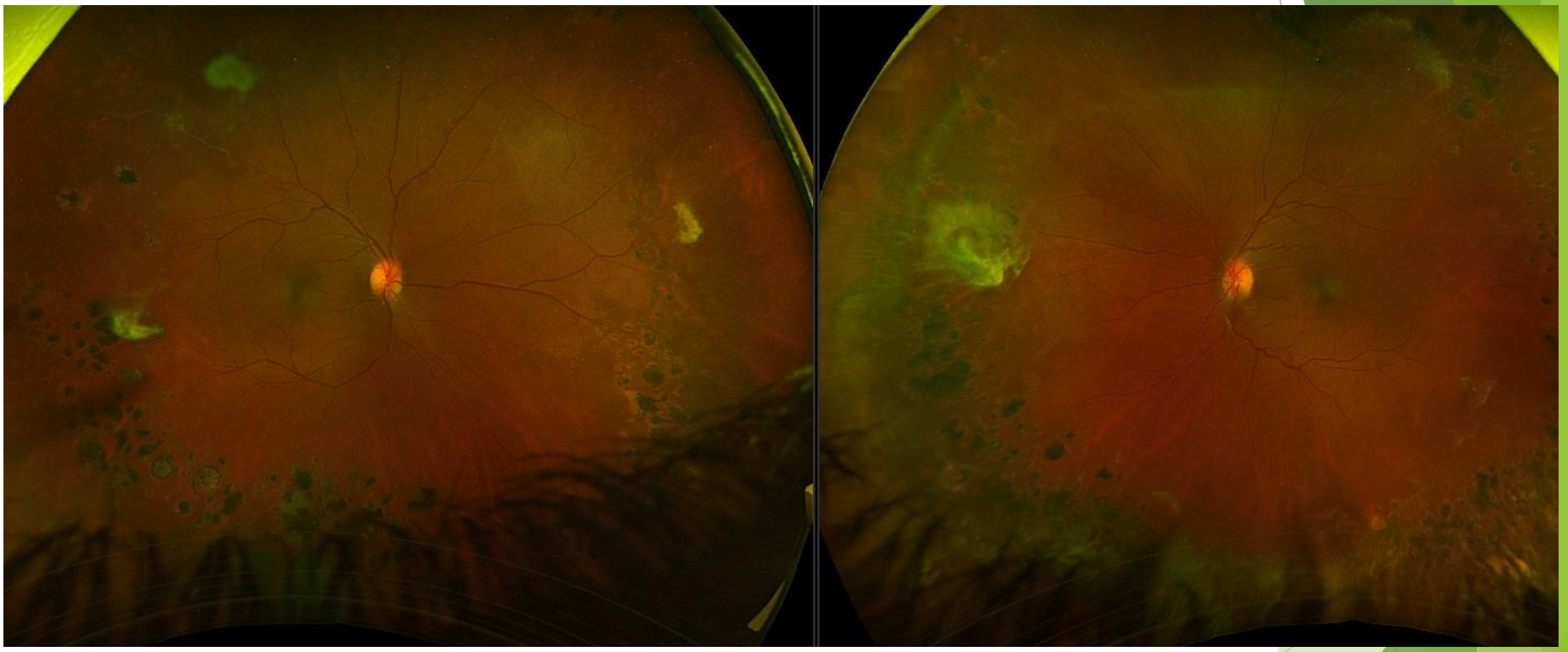
anastomoses

2019

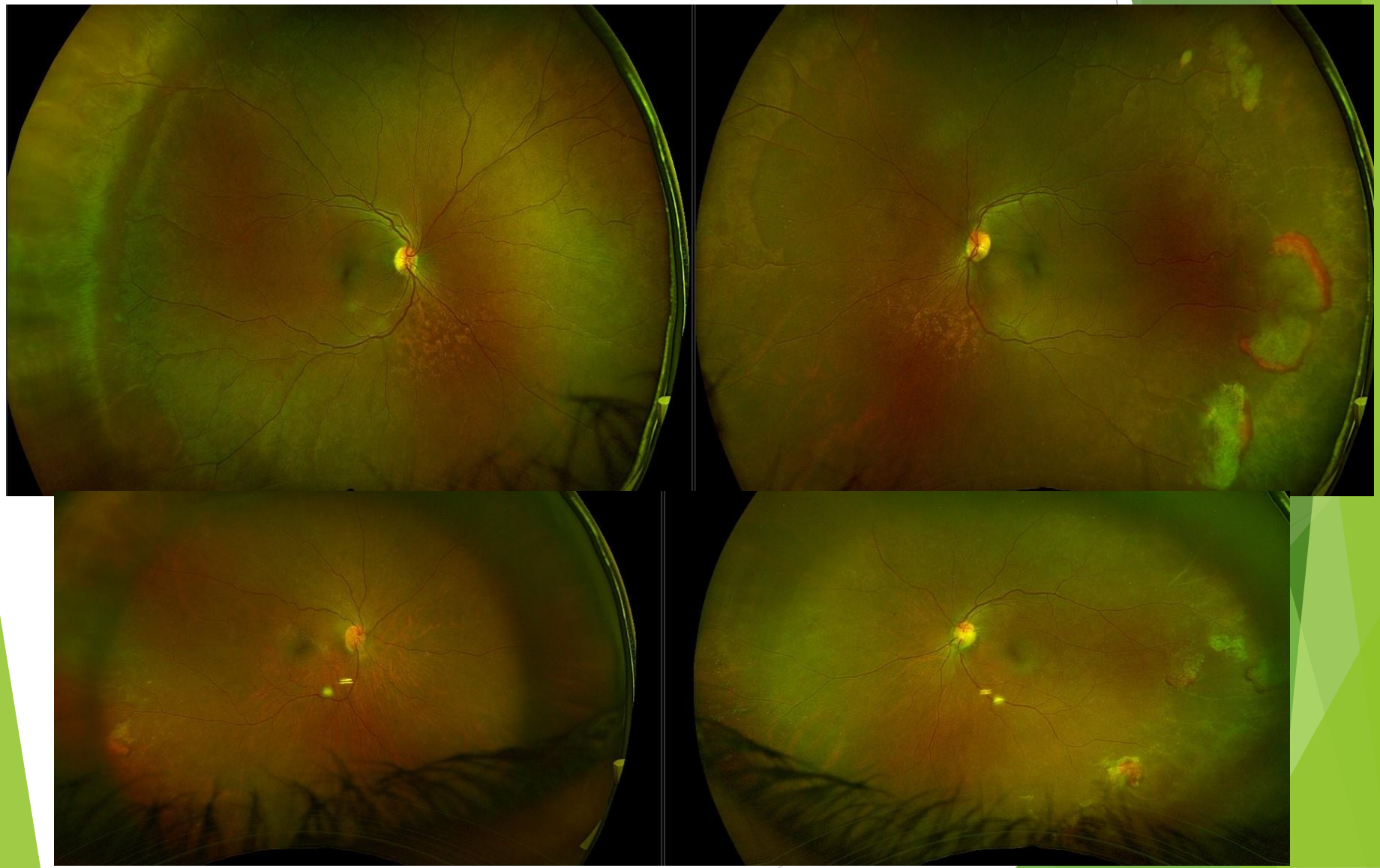
Peripheral anastomoses: S-Thalasemmia

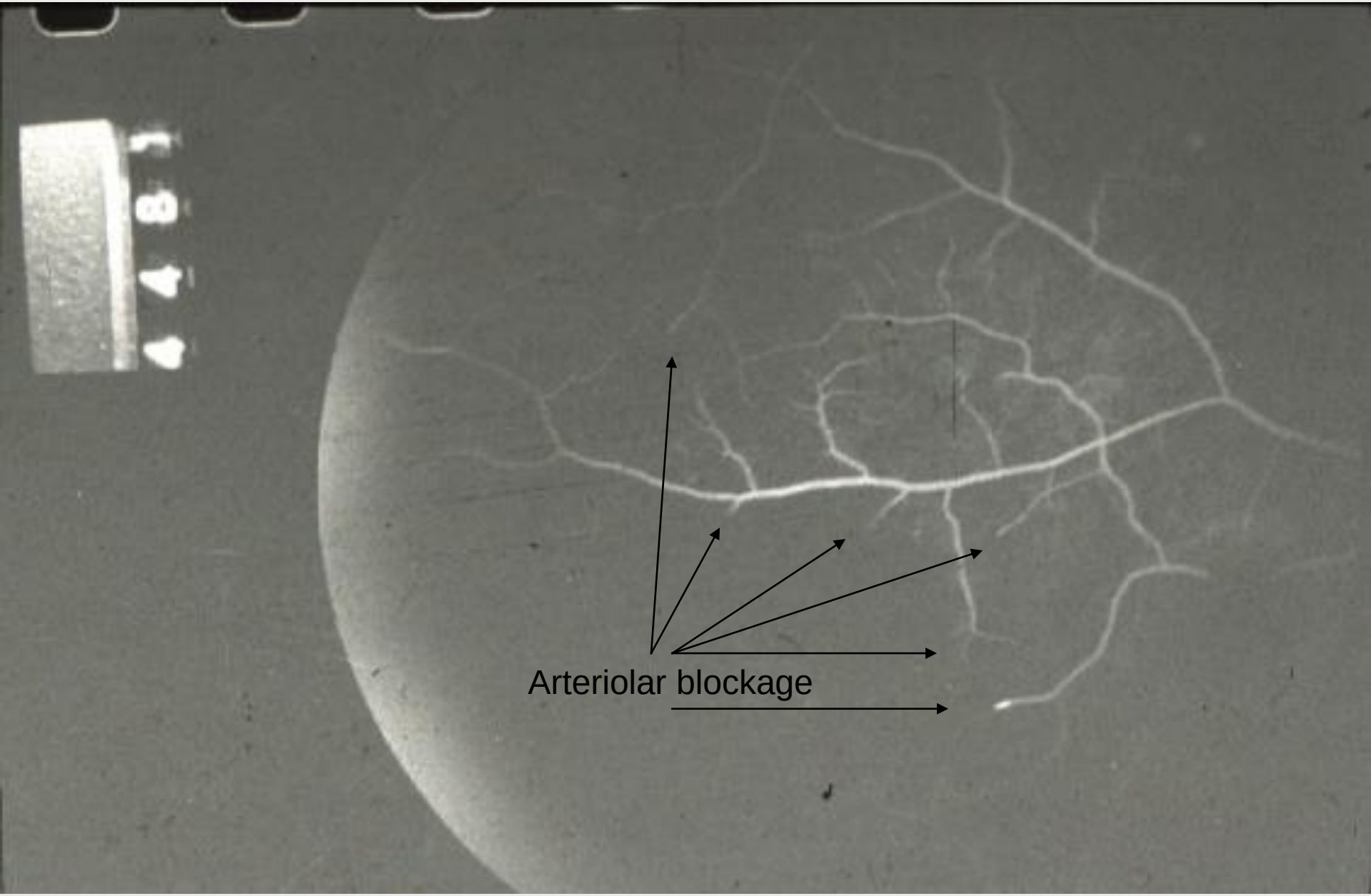


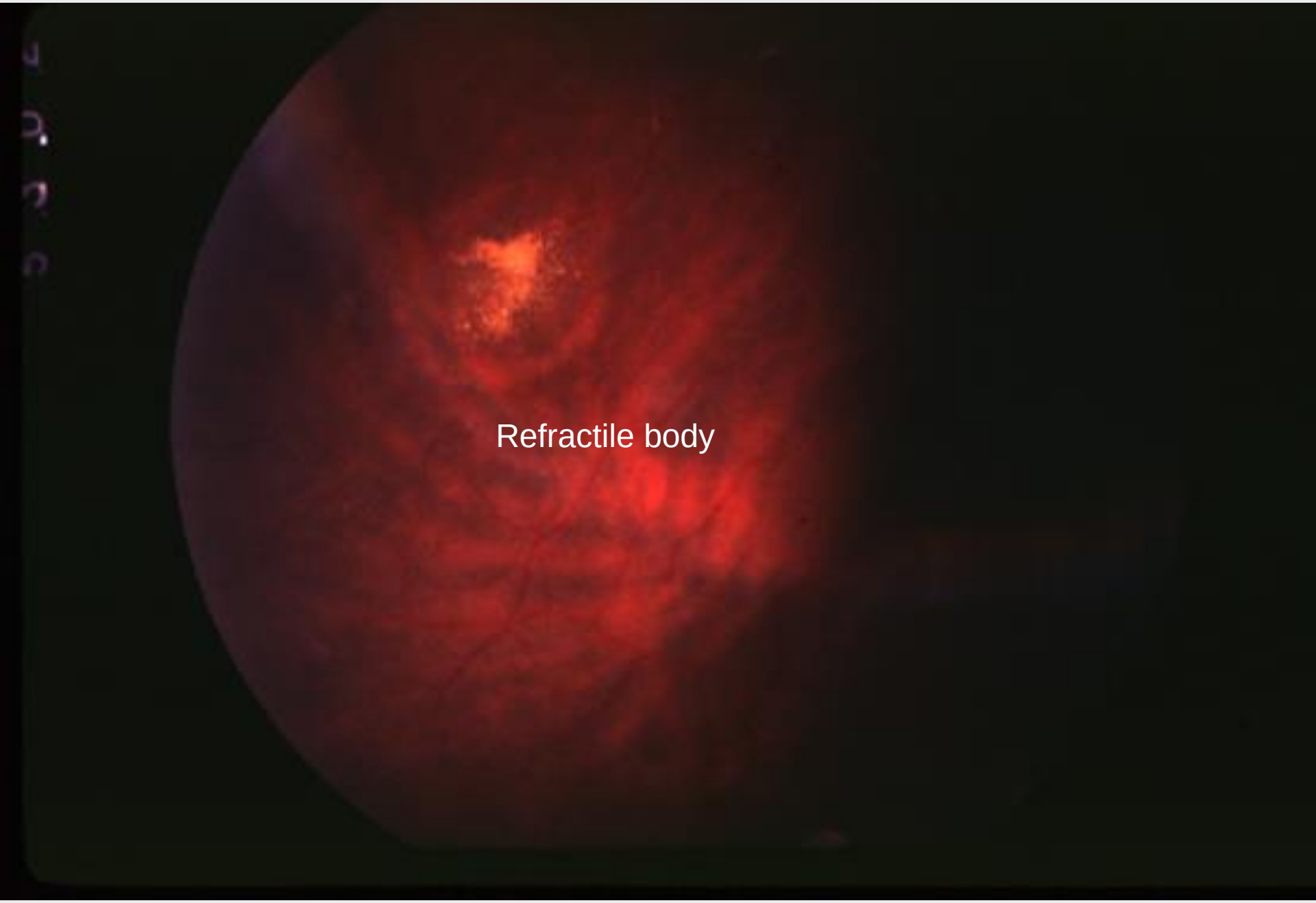
Sickle cell sea fans post PRP



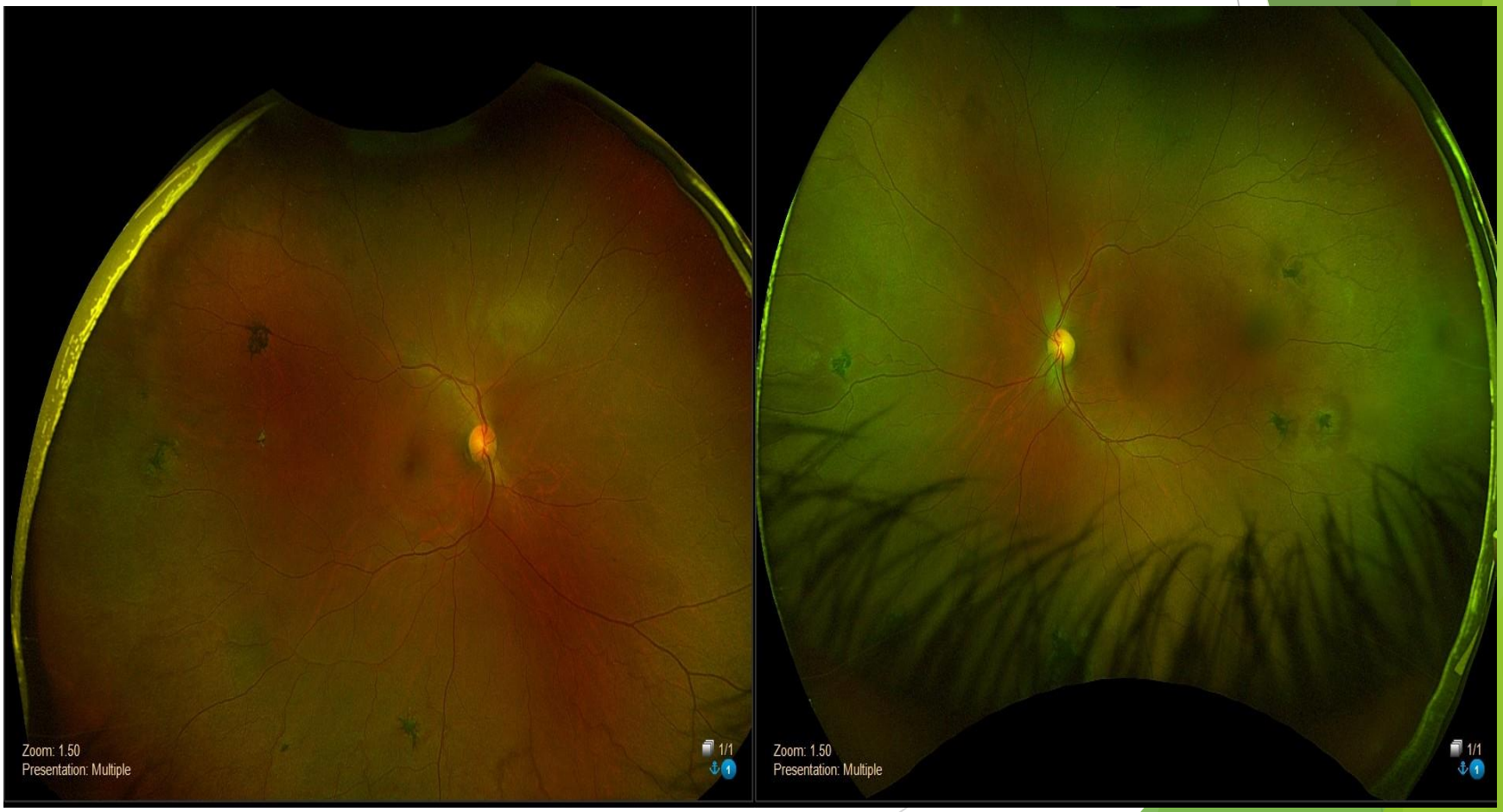
Not sickle cell, but……

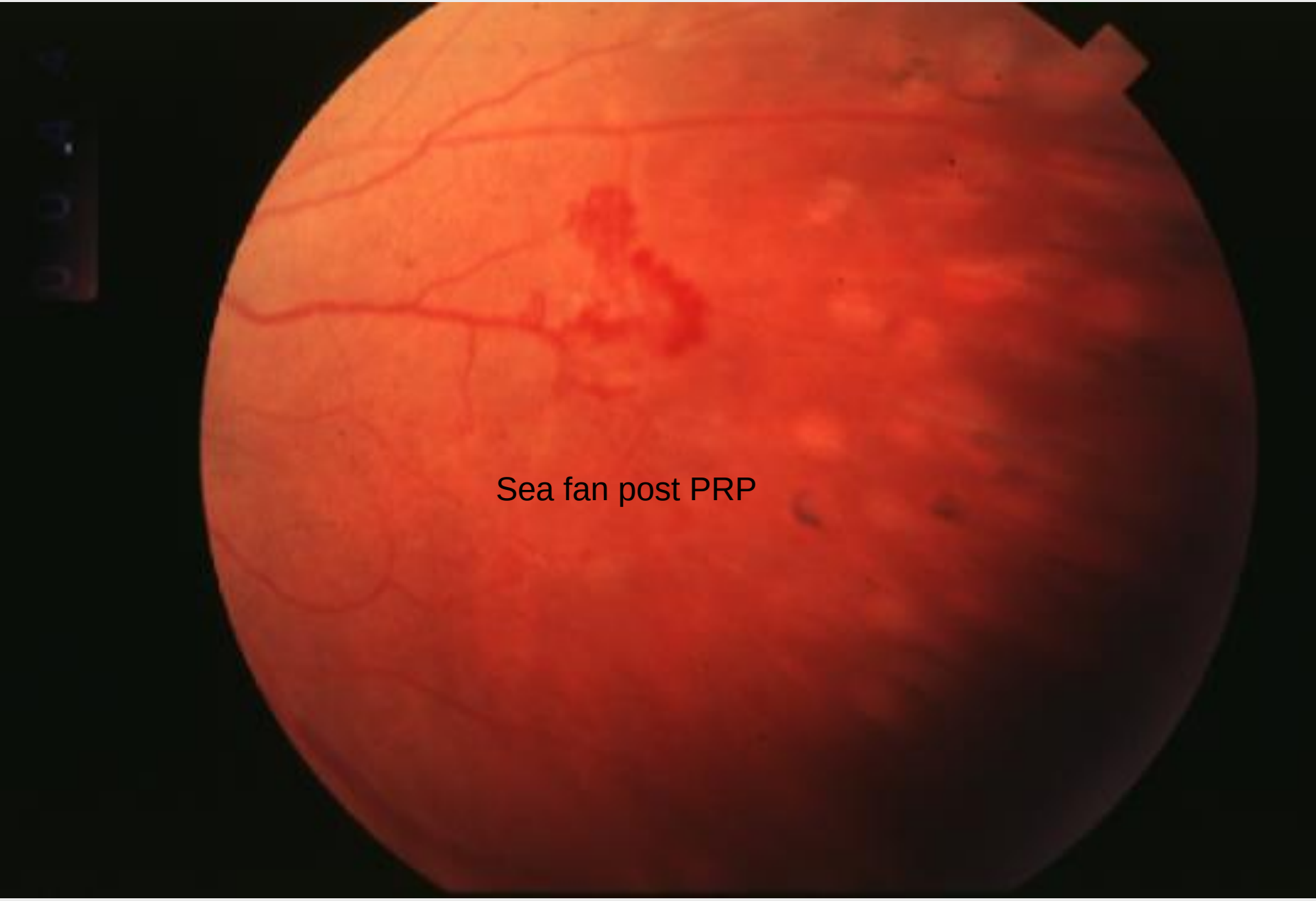


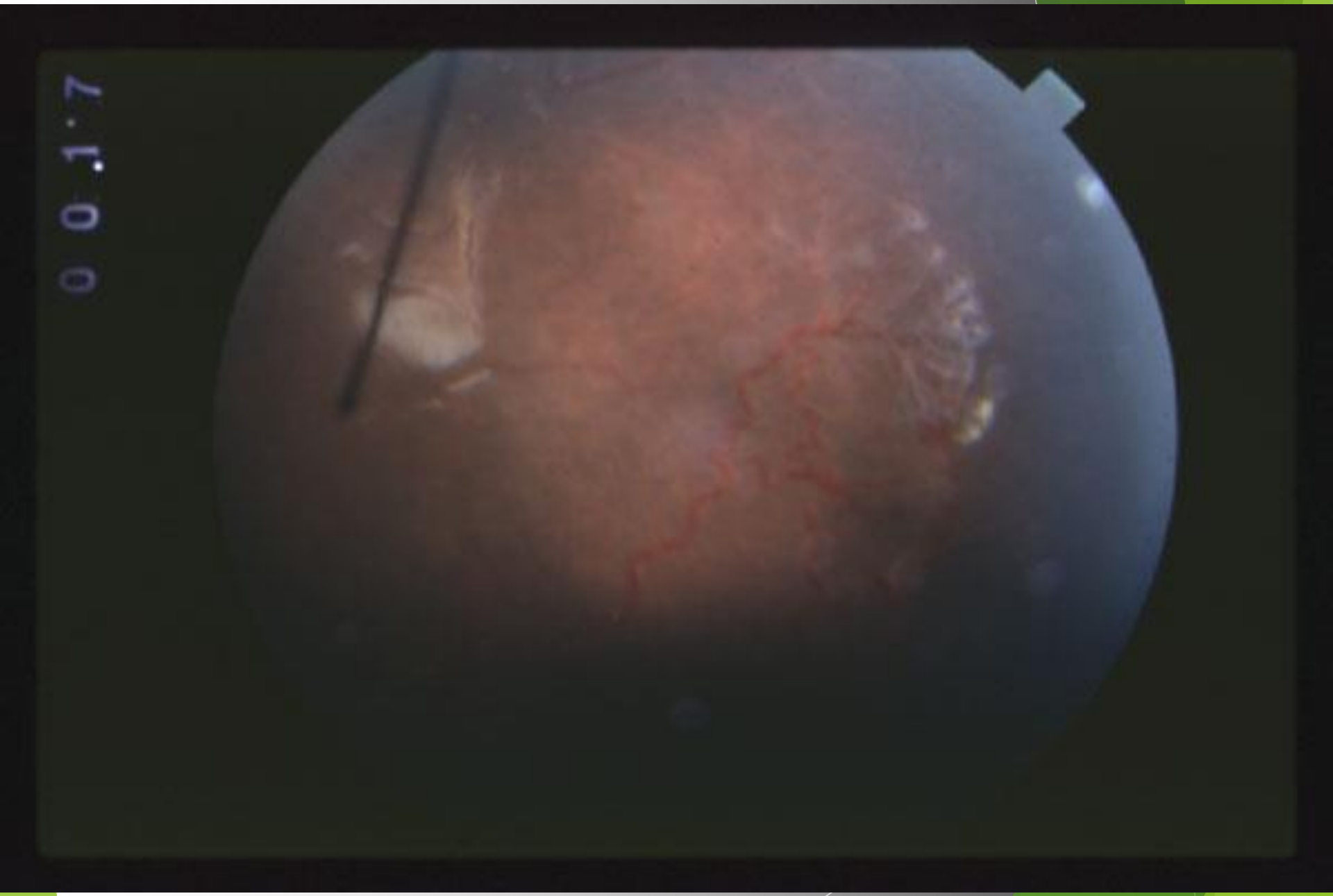

Arteriolar blockage


Refractile body

Sunburst pigment




Sea fan post PRP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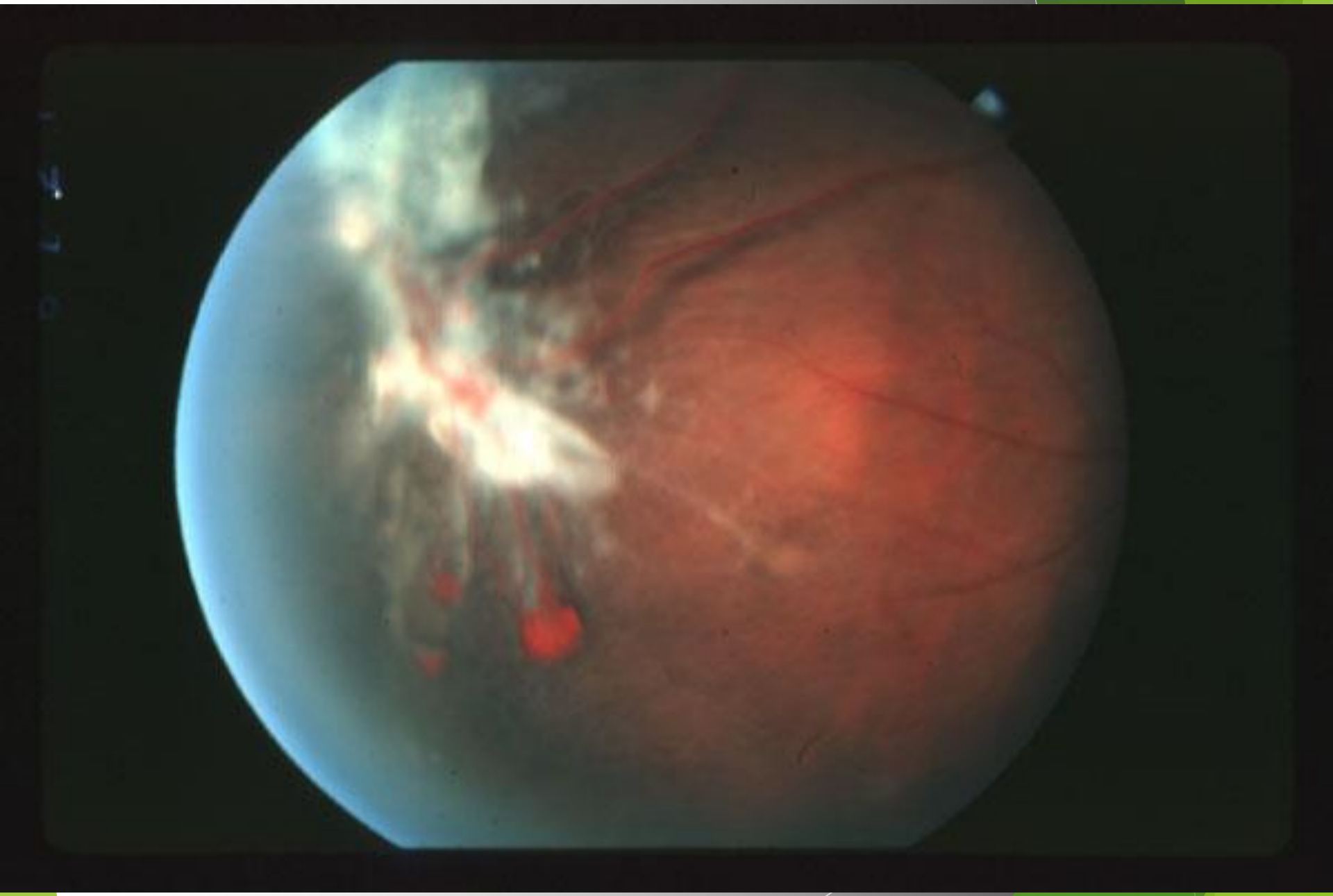


Hypertensive Retinopathy

- Over 100 million Americans have HTN, almost half of US adults
- 56% of AA adults, 48% of C, 46% of Asian, 39% of Hispanic
- Only one in four people with HTN are well controlled

- Many associated retinal findings-window to the body
- Arteriosclerosis, or arterial hardening, is an early finding
- Narrowing of the arteries

Hypertensive Retinopathy Grading

- Grade I : arteries ¾ normal caliber
- Grade II : arteries ½ normal caliber
- Grade III : arteries 1/3 normal caliber
- Grade IV : arteries thread-like or invisible

Study

- *Hypertension* Journal 2013 (October issue)
- 2907 Hypertensive patients followed for 13 years…….
- Mild HTN retinopathy = 35% increase in stroke risk
- Moderate to severe HTN retinopathy = 137% increase in stroke risk

Hypertensive Retinopathy

- Sclerosis > increased ALR
- Flame hemes (NFL)
- CWS (diastolic above 110)
- Gunn’s sign
- Papilledema
- Vein occlusions

Malignant Hypertension

- Ocular findings are severe and include exudative edema and papilledema
- Mortality rate:
- 80% @ 1 year
- 95% @ 2 years

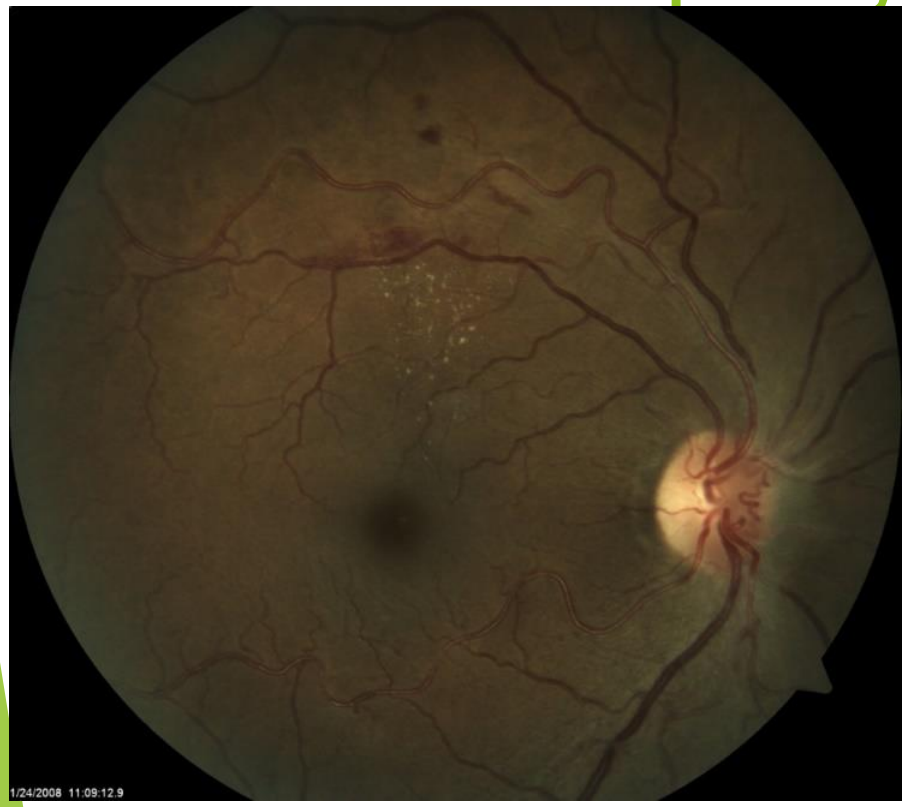
Hypertensive Retinopathy

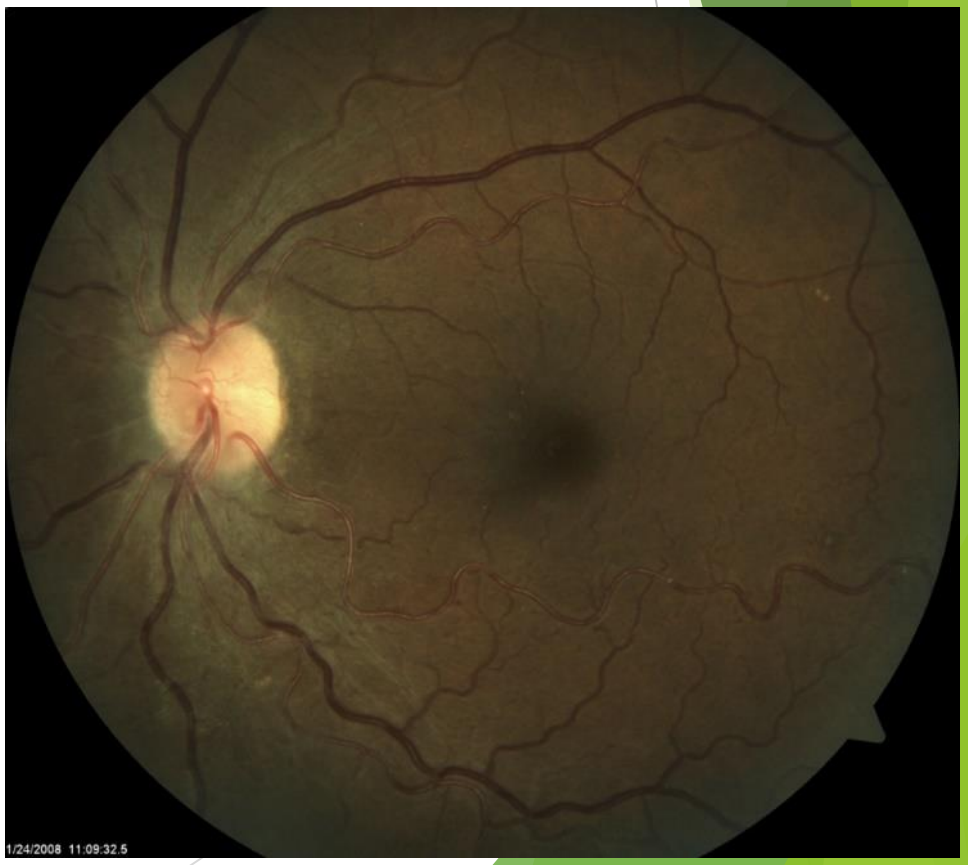
- The choroid is commonly affected: more vascular than the retina
- Elschnig's spots and Siegrist's streaks
- Watch substantial asymmetry: possible carotid issues

Hypertensive Retinopathy

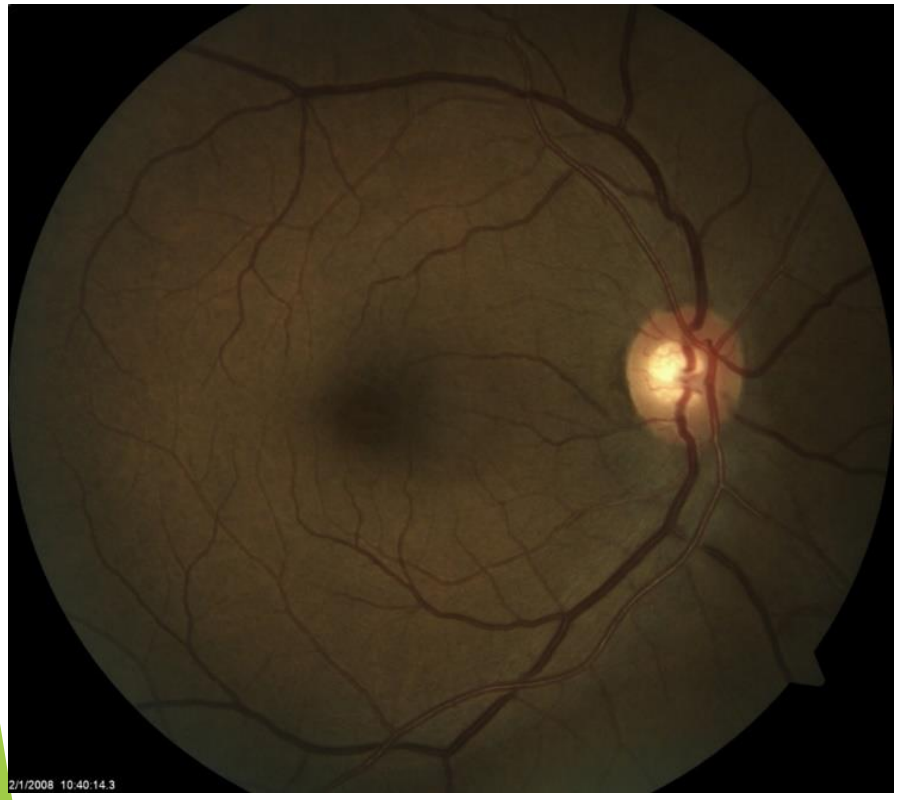
- No ocular treatment; manage by controlling systemic disease

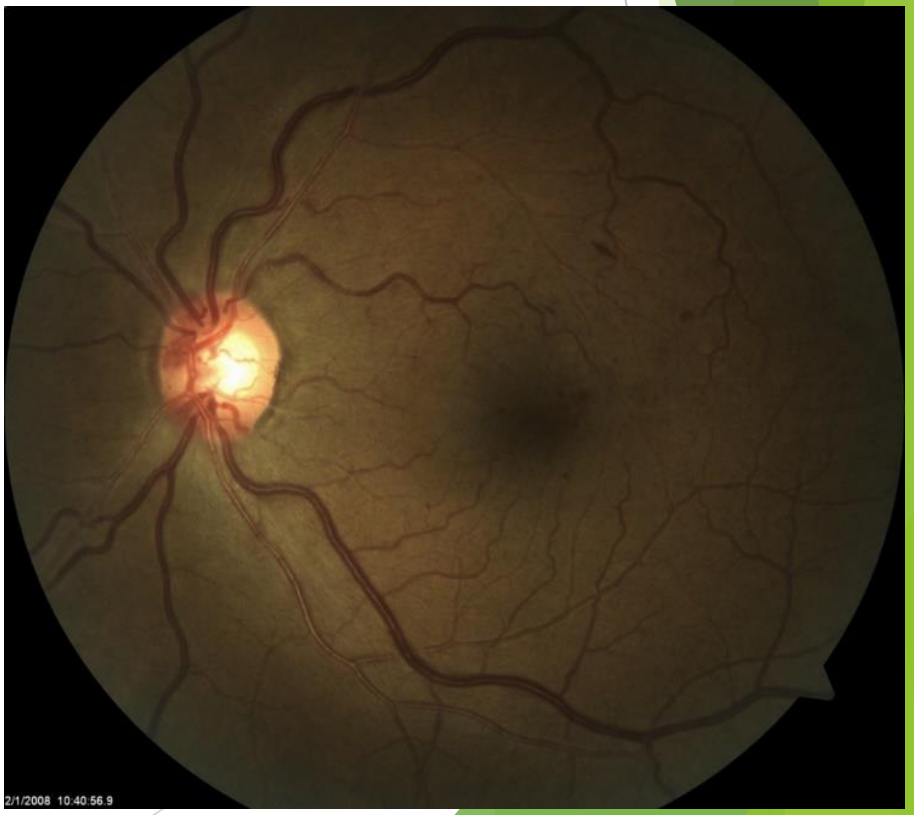
HTN vasculopathy



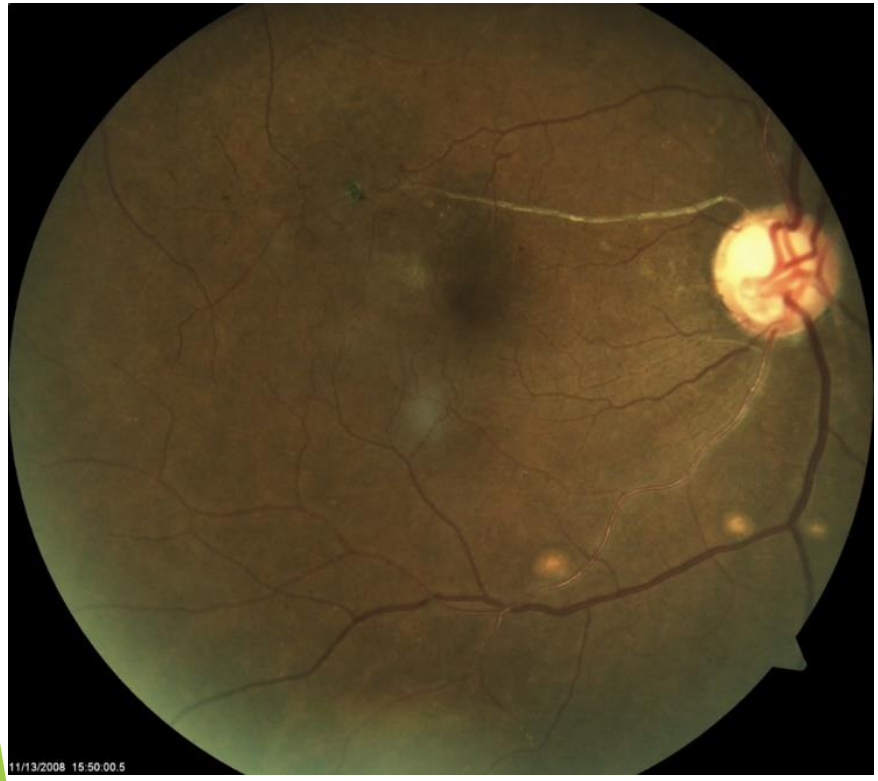


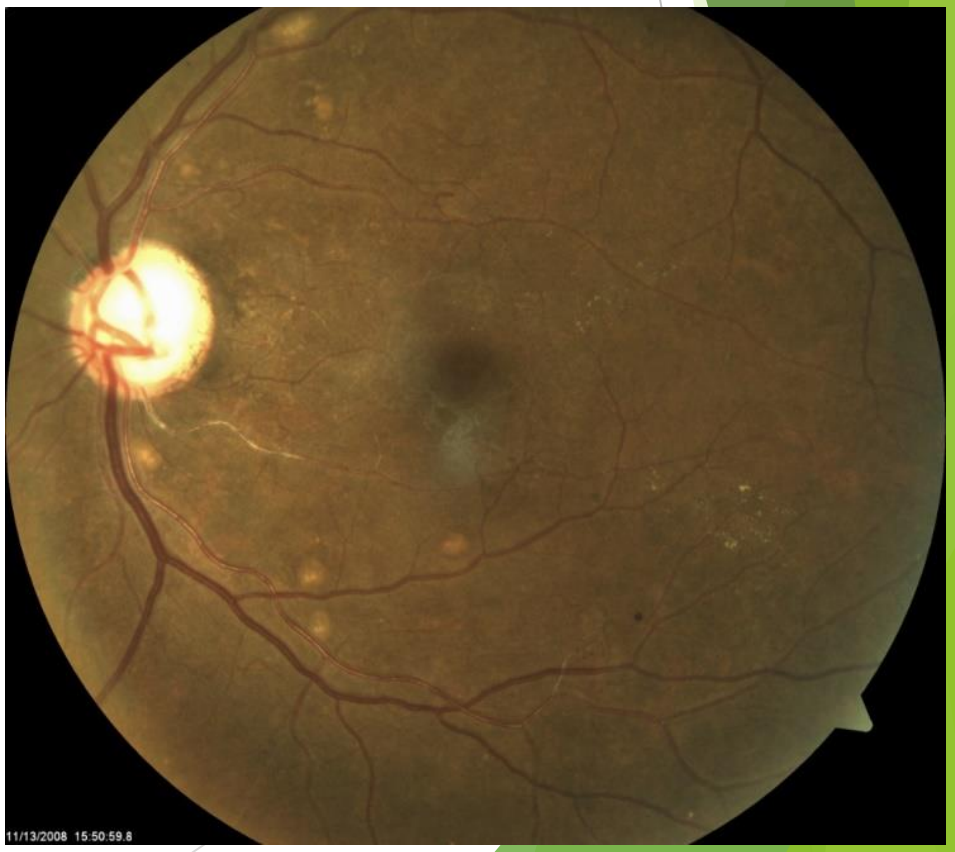
HTN vasculopathy and BRVO





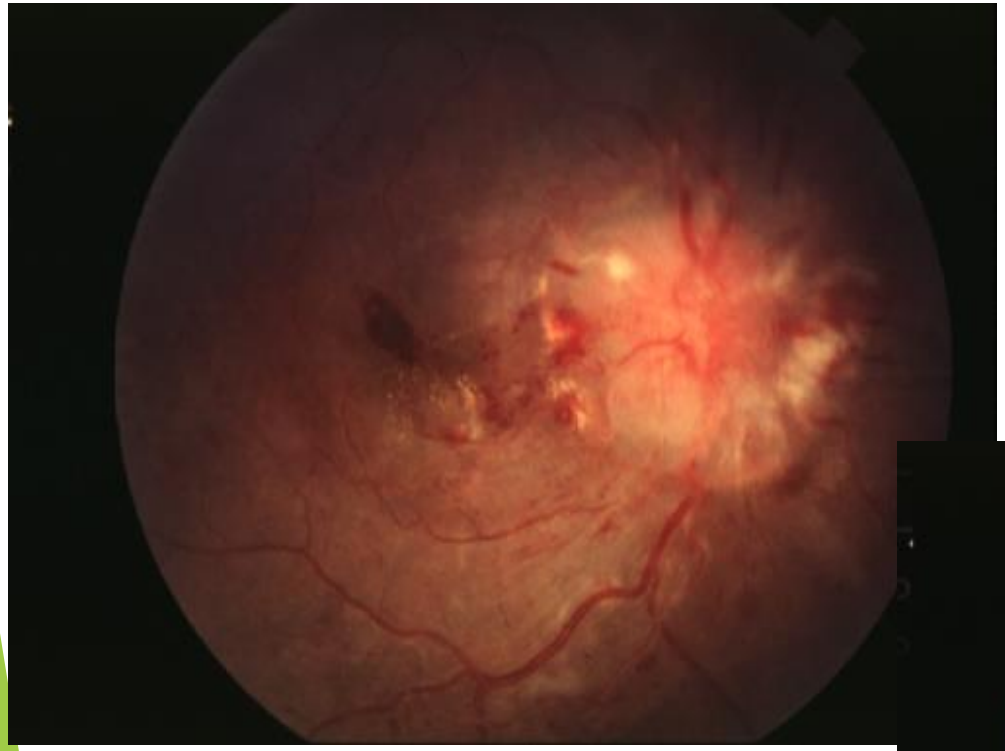
HTN vasculopathy

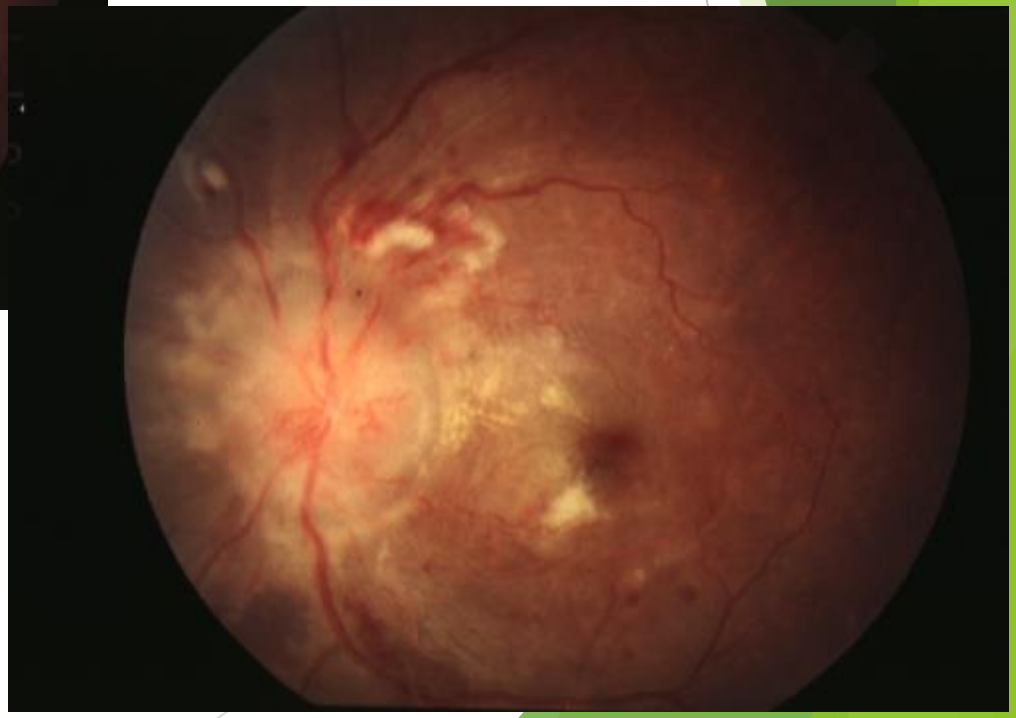




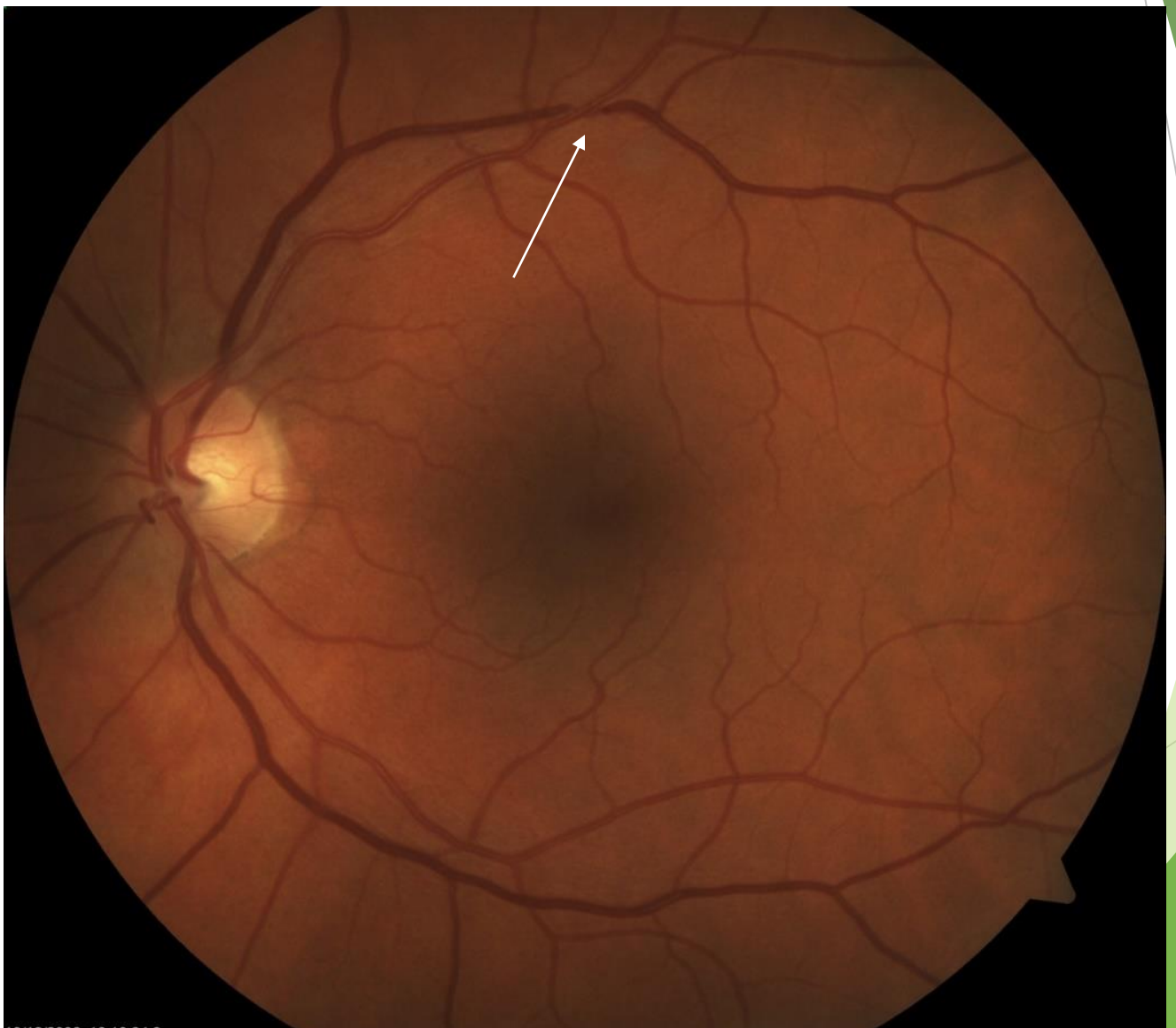
Elschnig spots OU

Malignant HTN papilledema

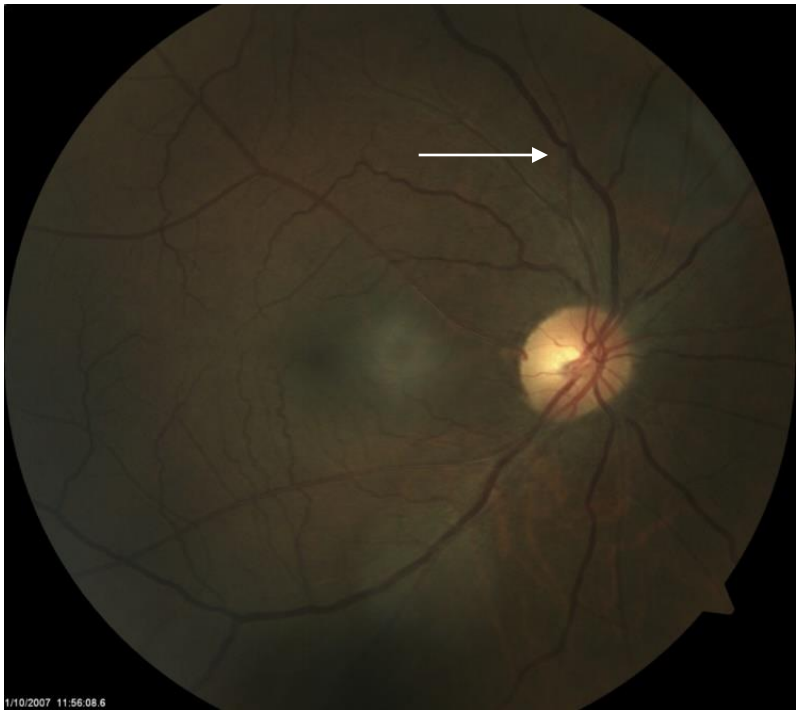


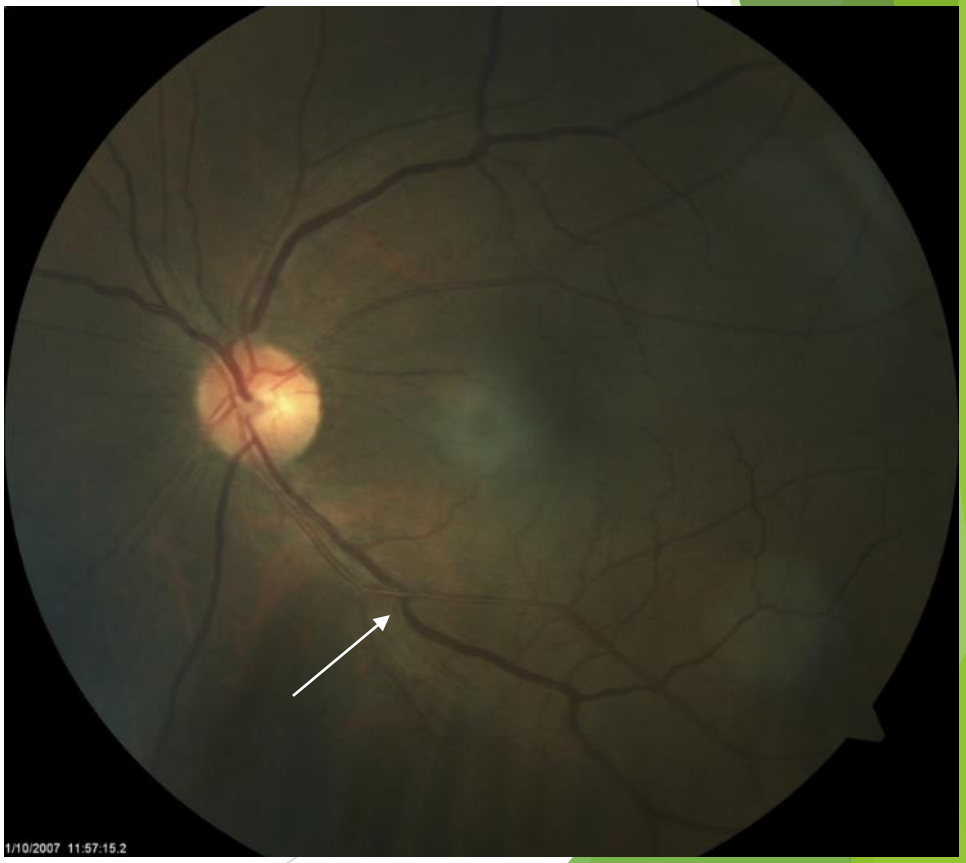


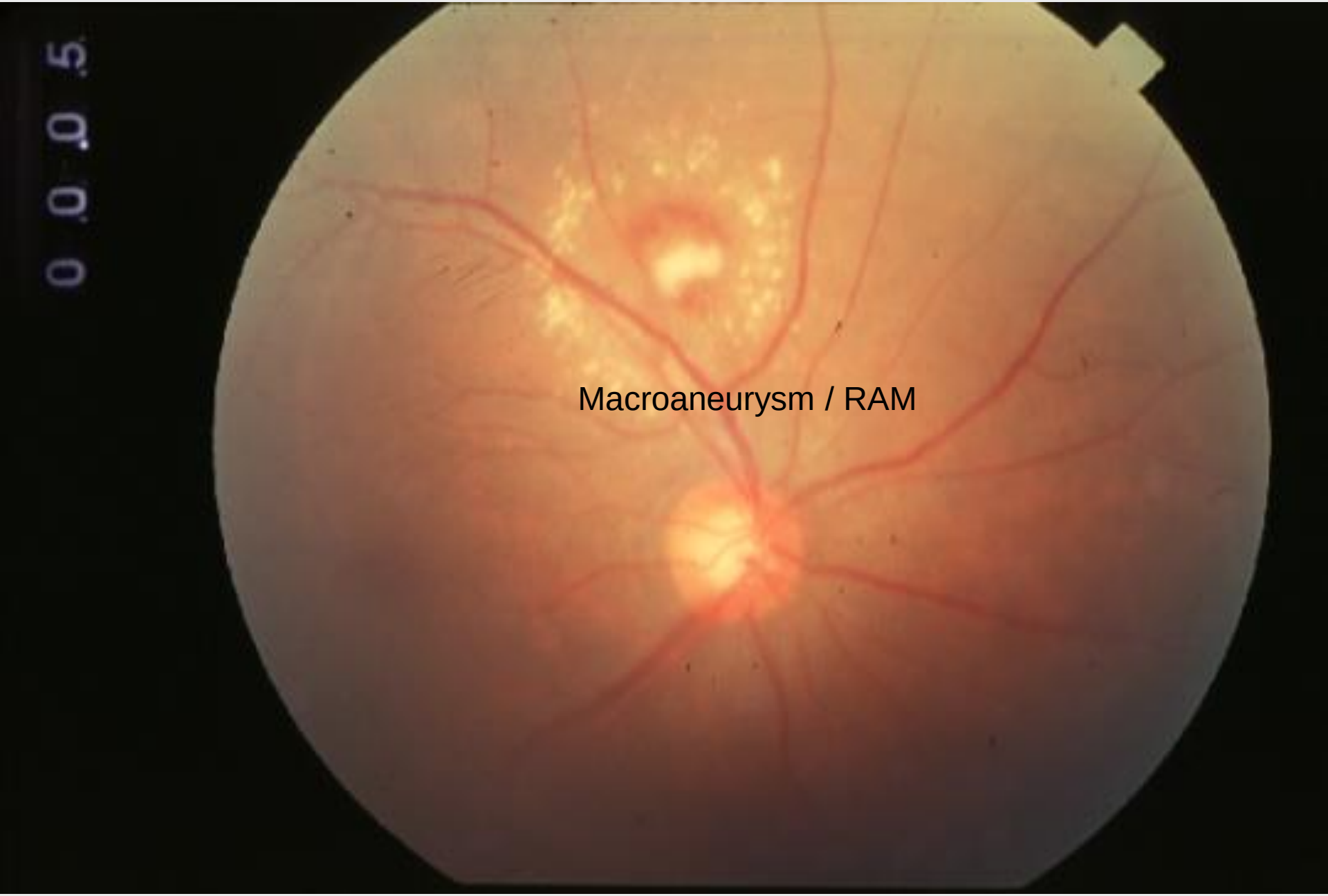
Crossing Change (Gunn's sign)



Crossing changes



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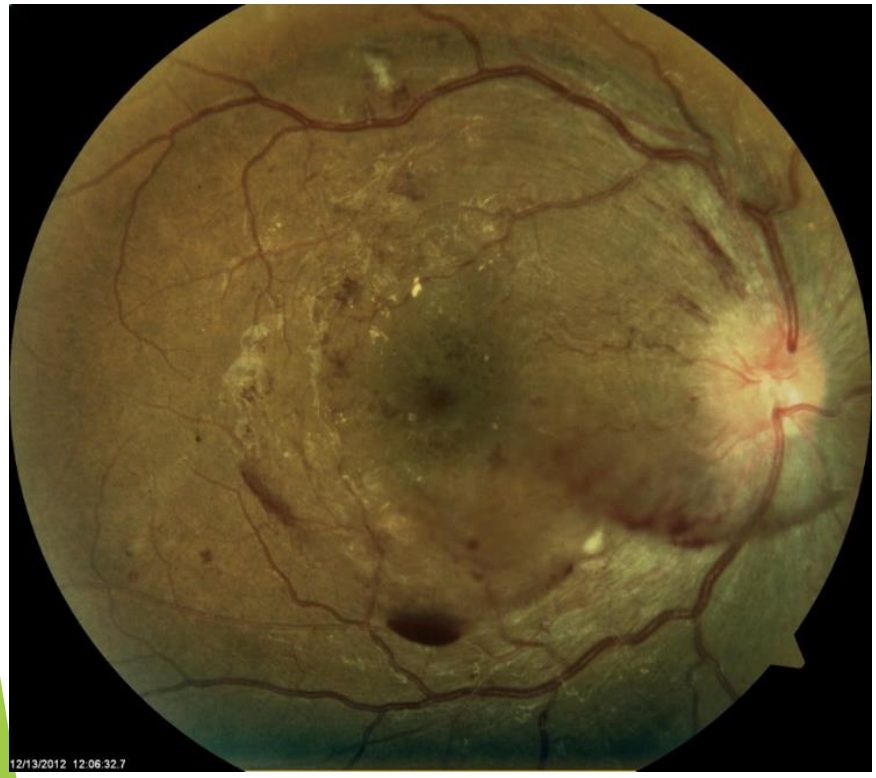
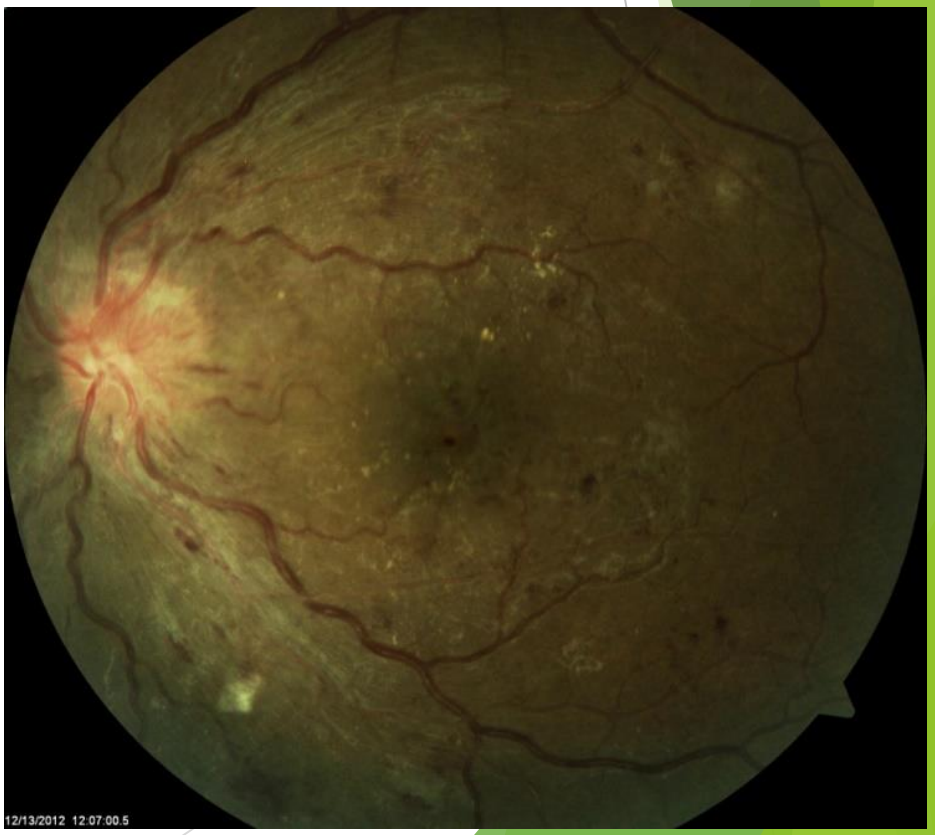
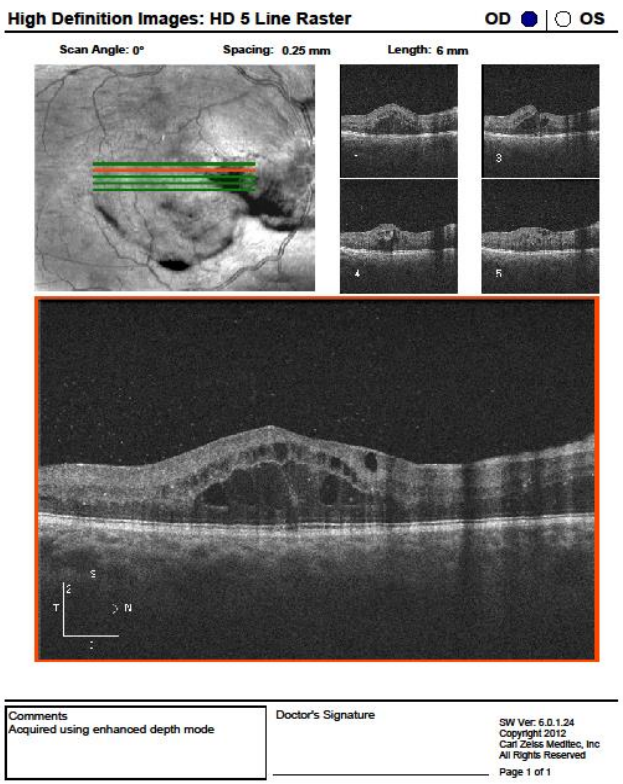
Macroaneurysm / RAM

HTN Retinopathy BP 240 / 135





BP 240/135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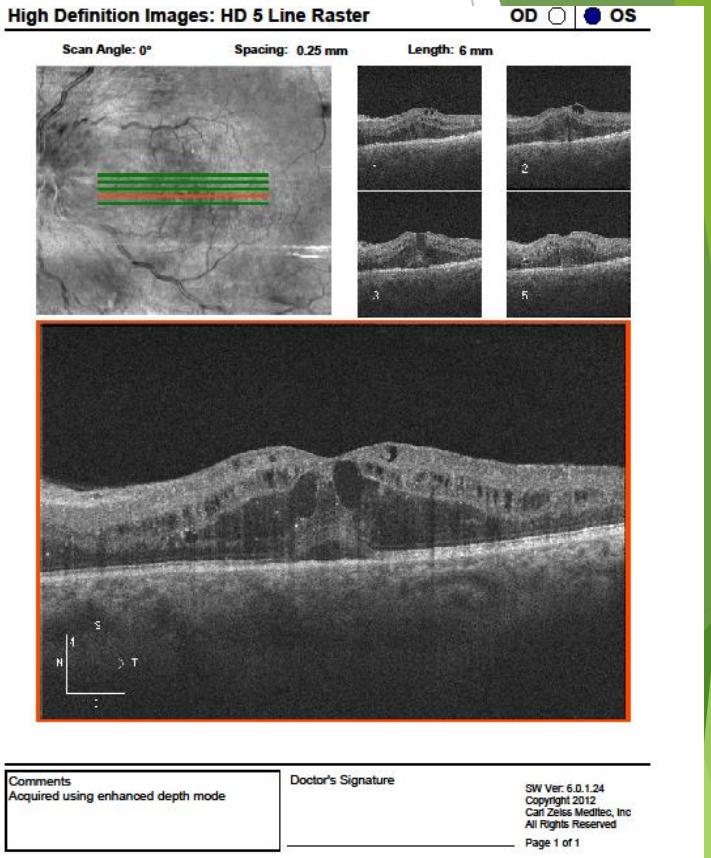
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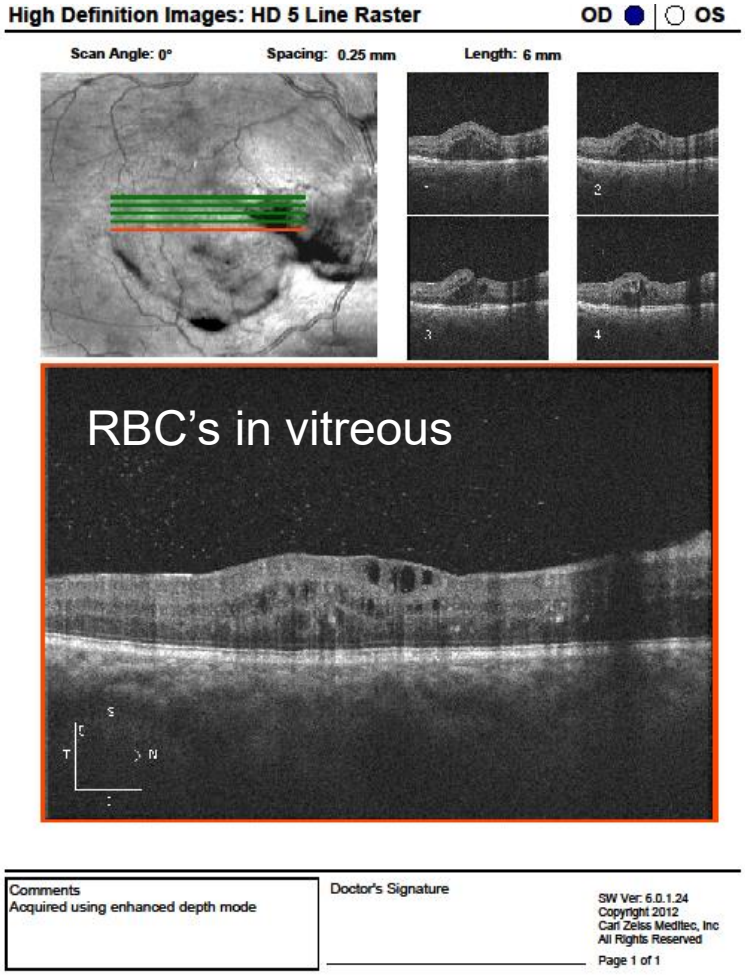
Bp 240 / 135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RBC's in vitreous
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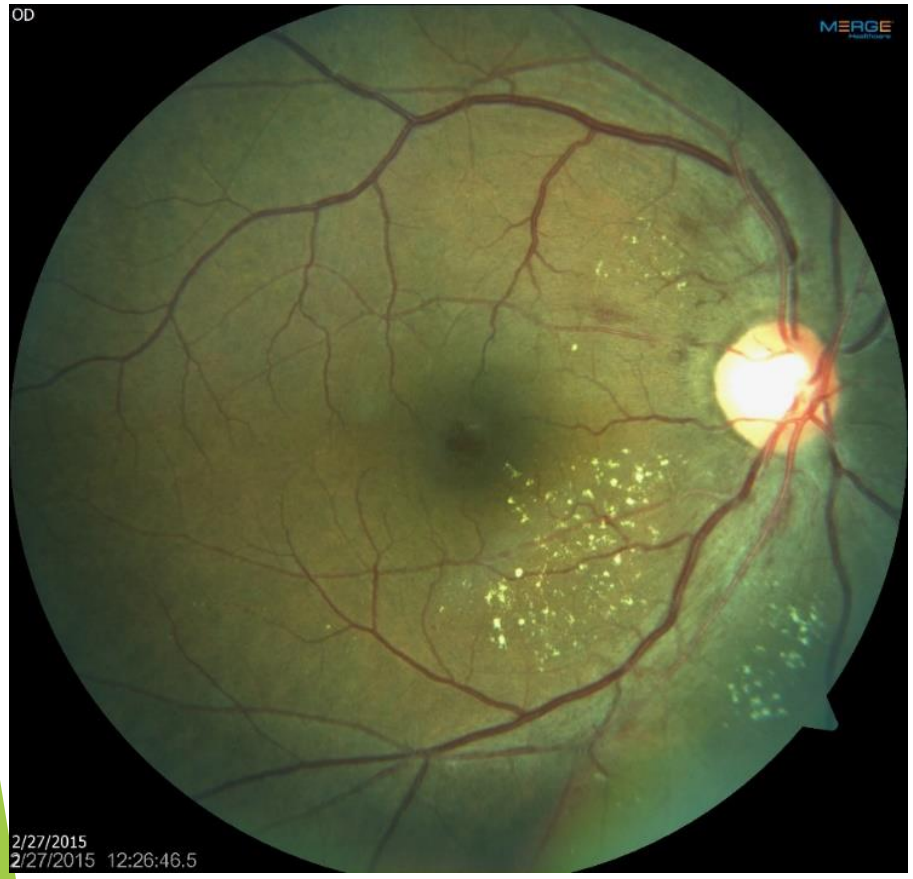
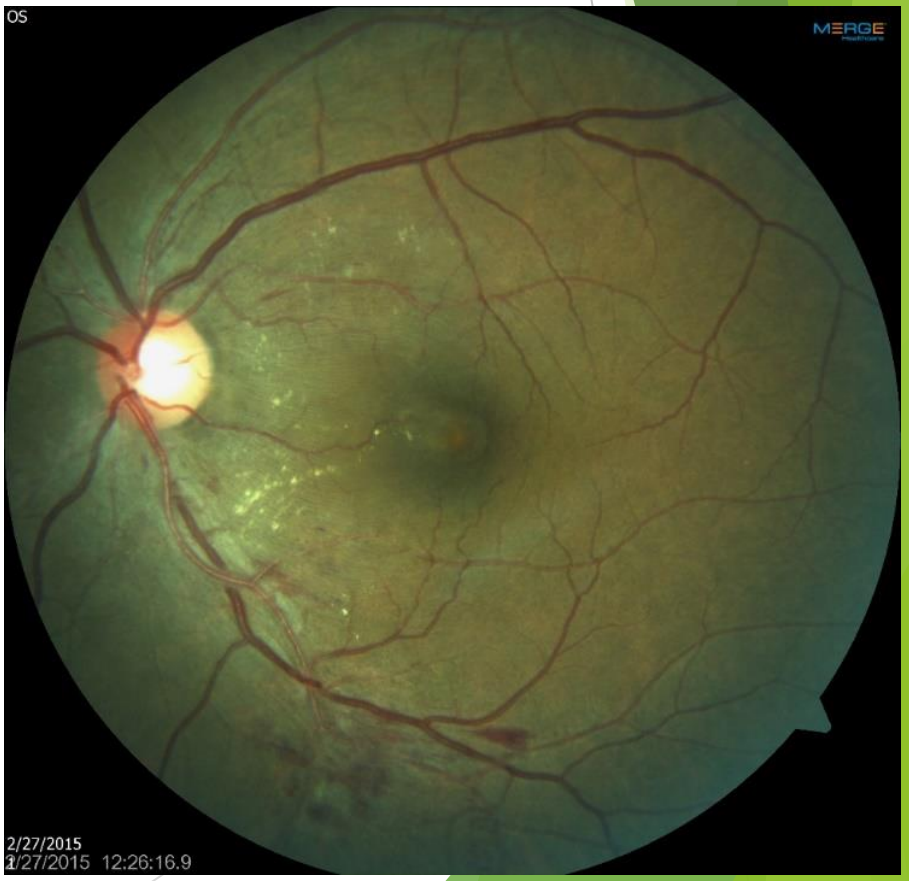
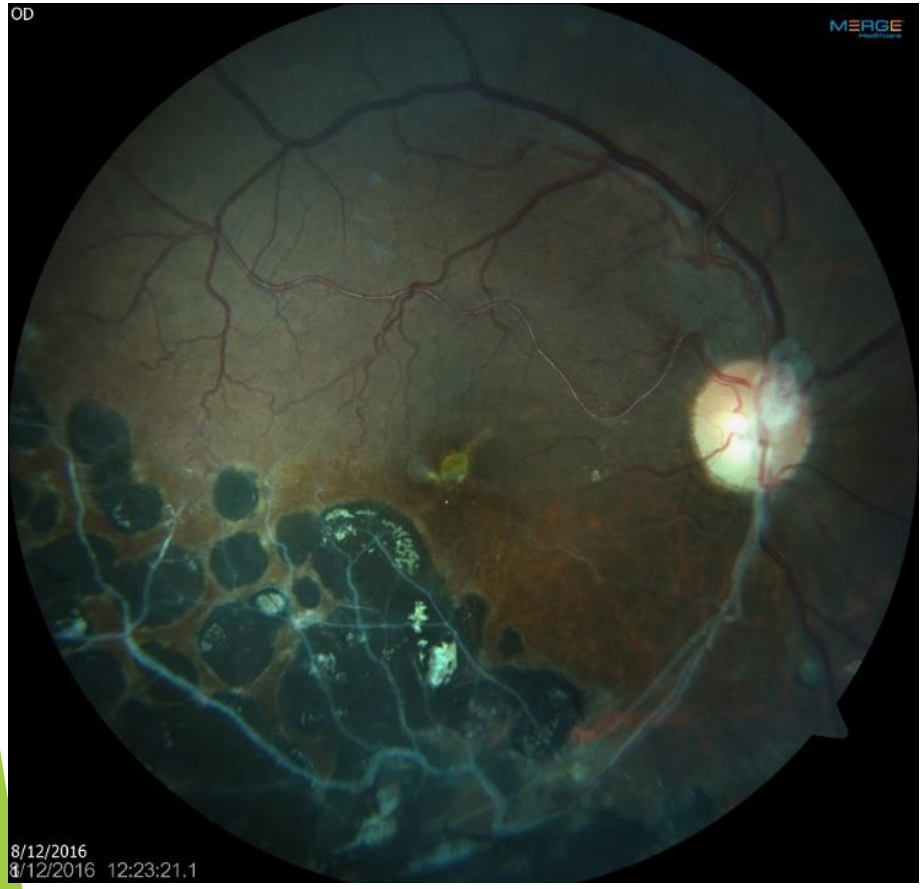
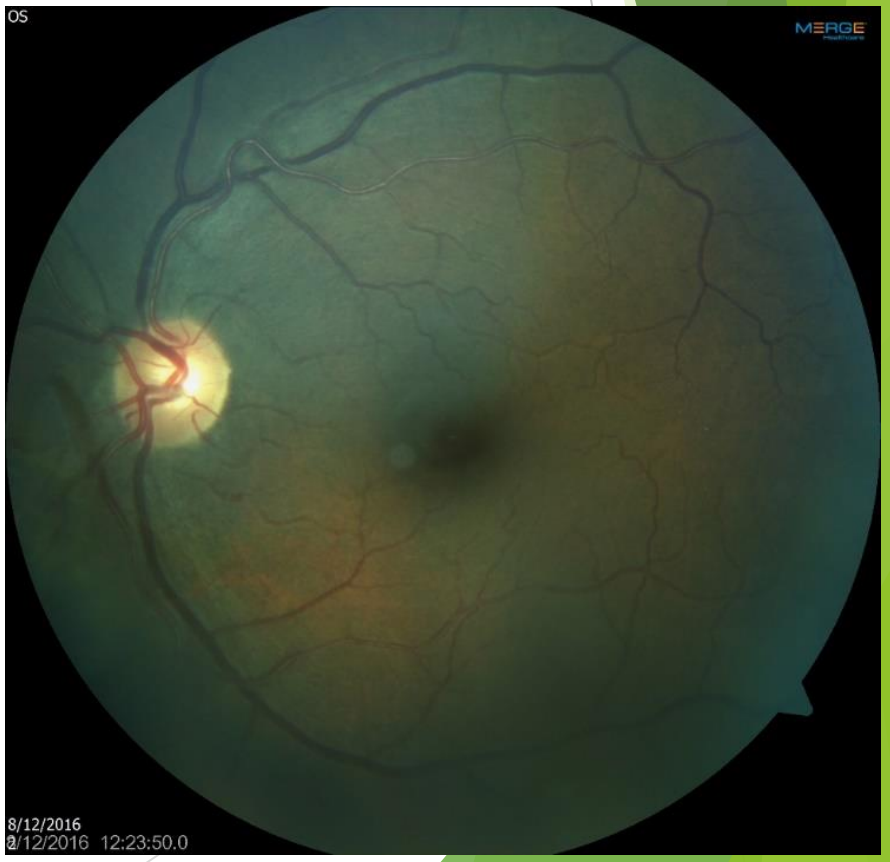
HTN retinopathy 20/20 OU



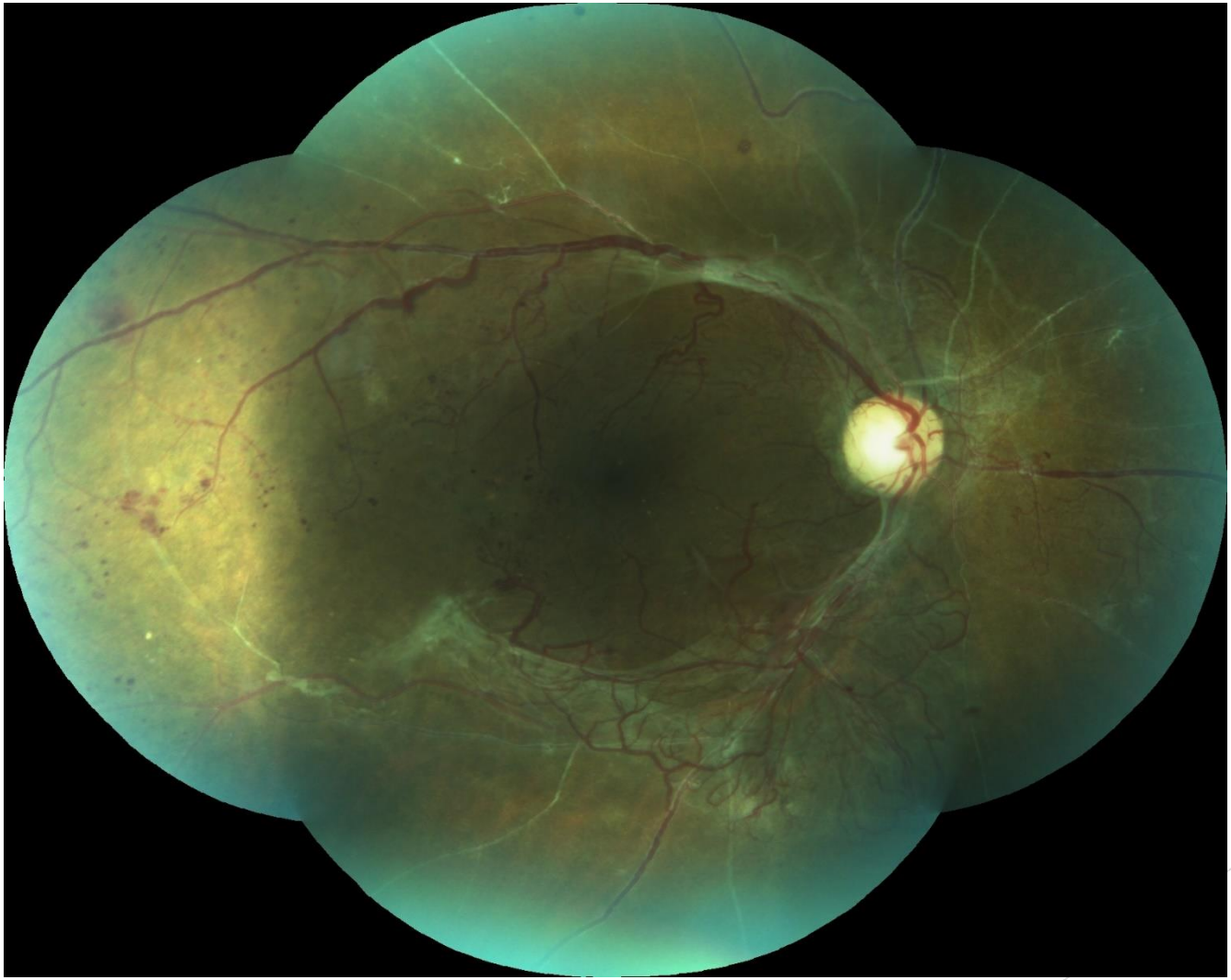


HTN vasculopathy

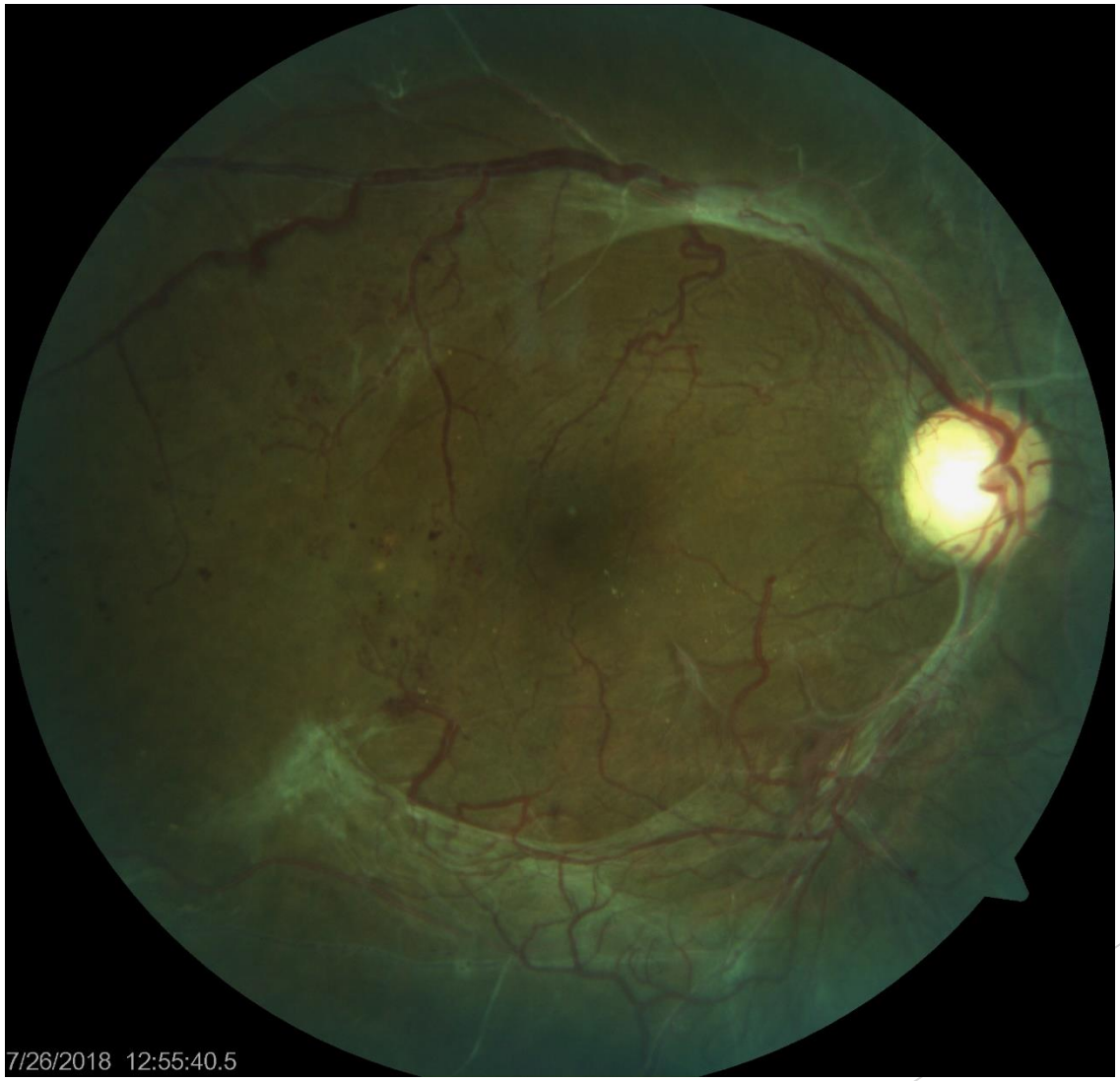




HTN vasculopathy and PDR



HTN vasculopathy and PDR



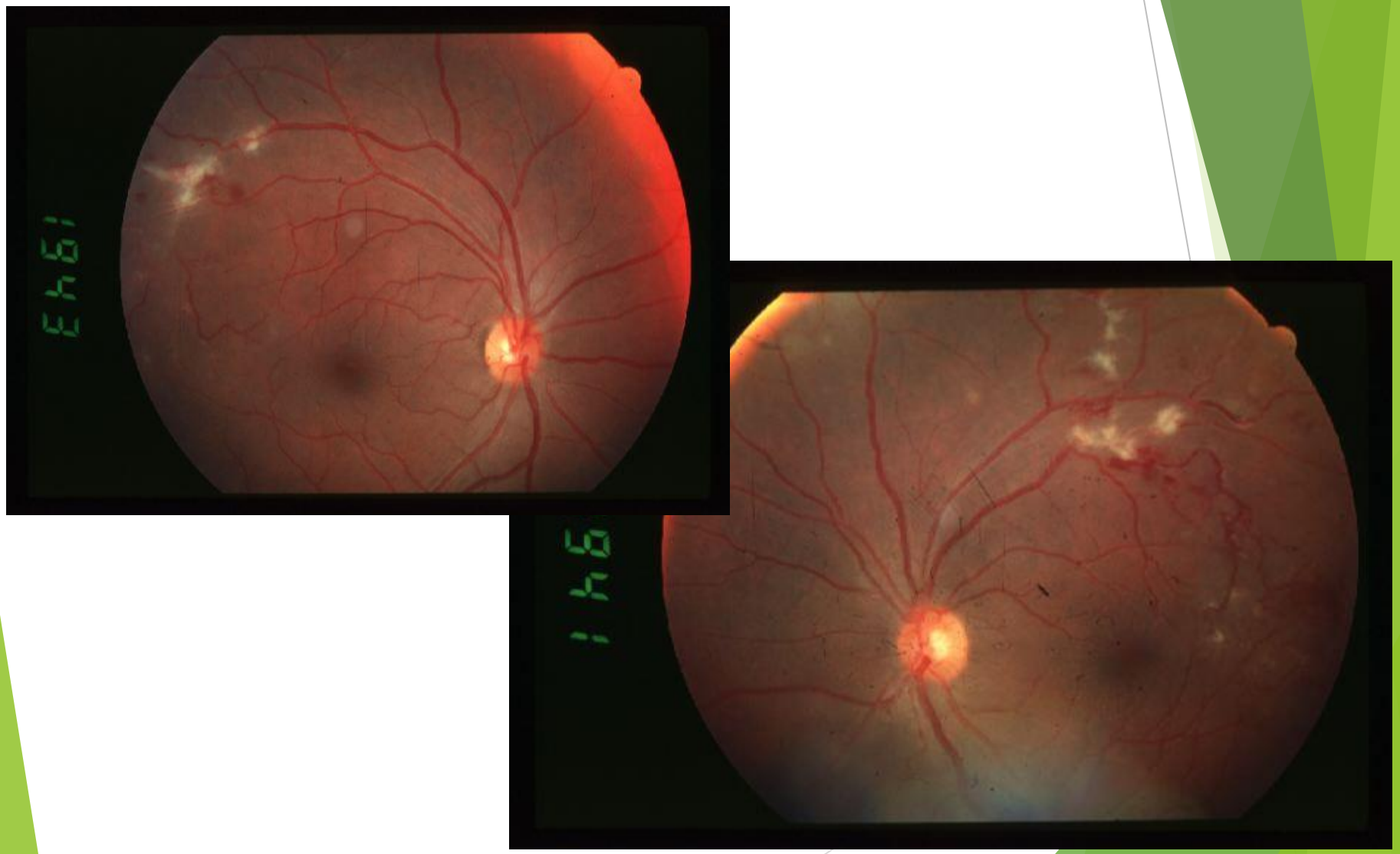
Eale's Disease

- Idiopathic vasculopathy affecting healthy, young adults. India, Pakistan, and Afghanistan / Middle East most common
- Usually strikes patients in their twenties or thirties. M>F
- Historical association with TB

Eale's Disease

- Retinal vascular sheathing and exudative sheathing in the periphery
- CME, vitreal cells, anterior chamber cells/flare, and peripheral retinal non-perfusion
- Neovascularization of the retina, disc, and iris are possible
- Treatment consists of PRP / cryo / anti VEGF

Eale's disease



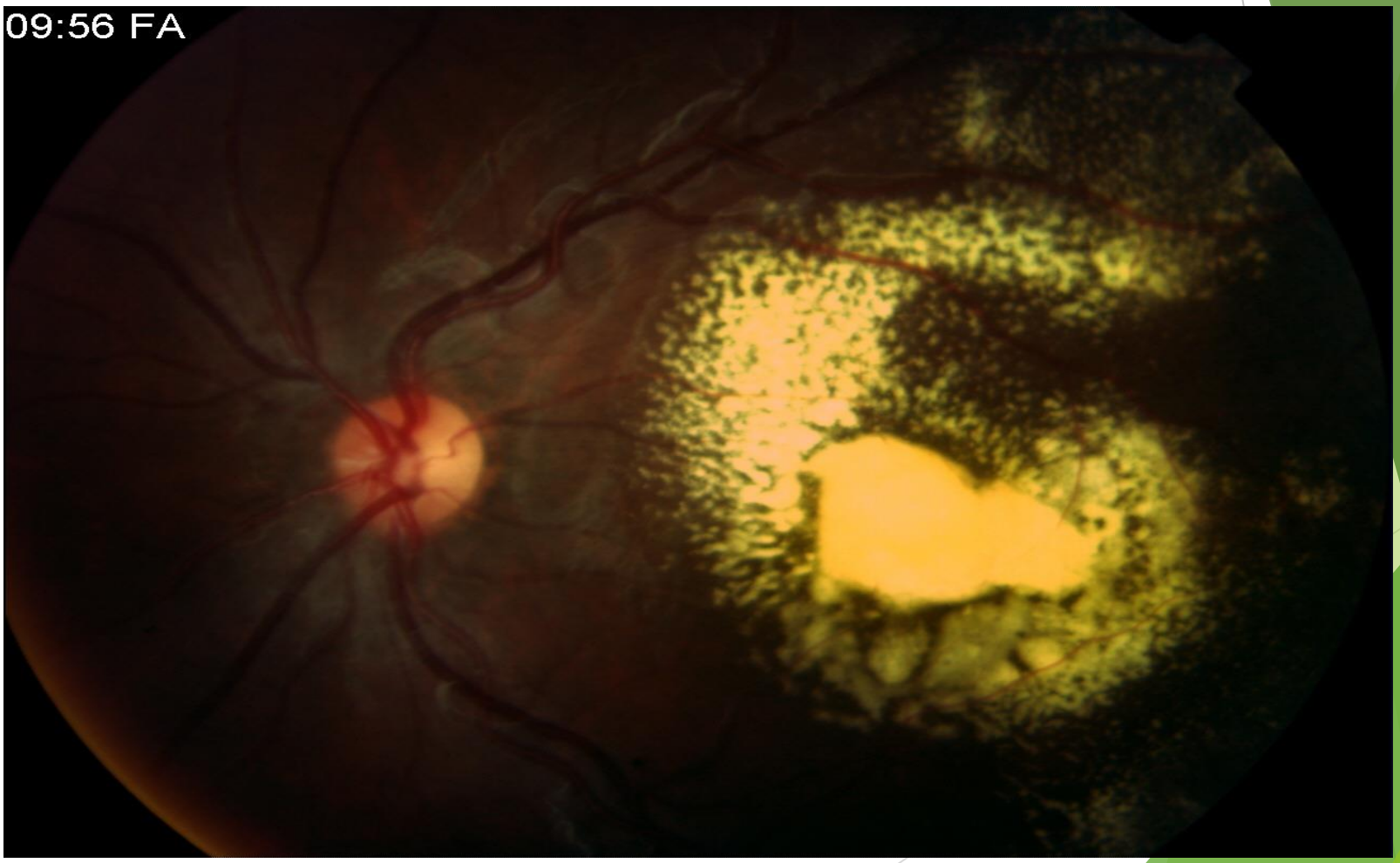
Coat's Disease

- Idiopathic retinal condition consisting of telangiectatic and aneurysmal vessels with significant exudation
- Adolescents typically
- Males 3x females ; 80-95% unilateral
- Leukocoria, poor vision, strabismus
- * What are the other main causes of leukocoria in children?

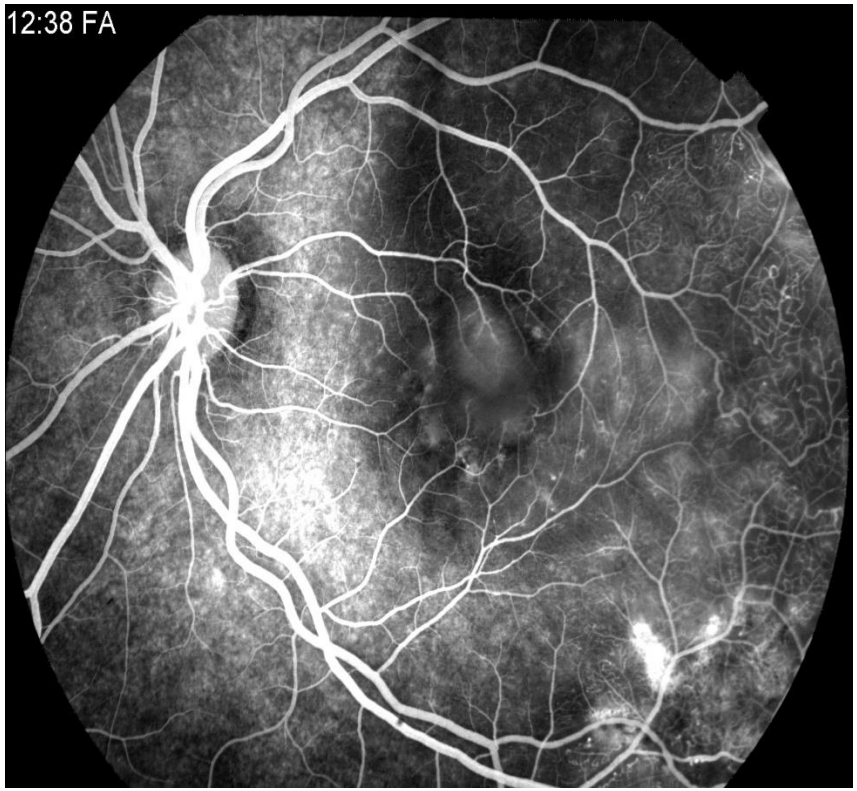
Coat's Disease

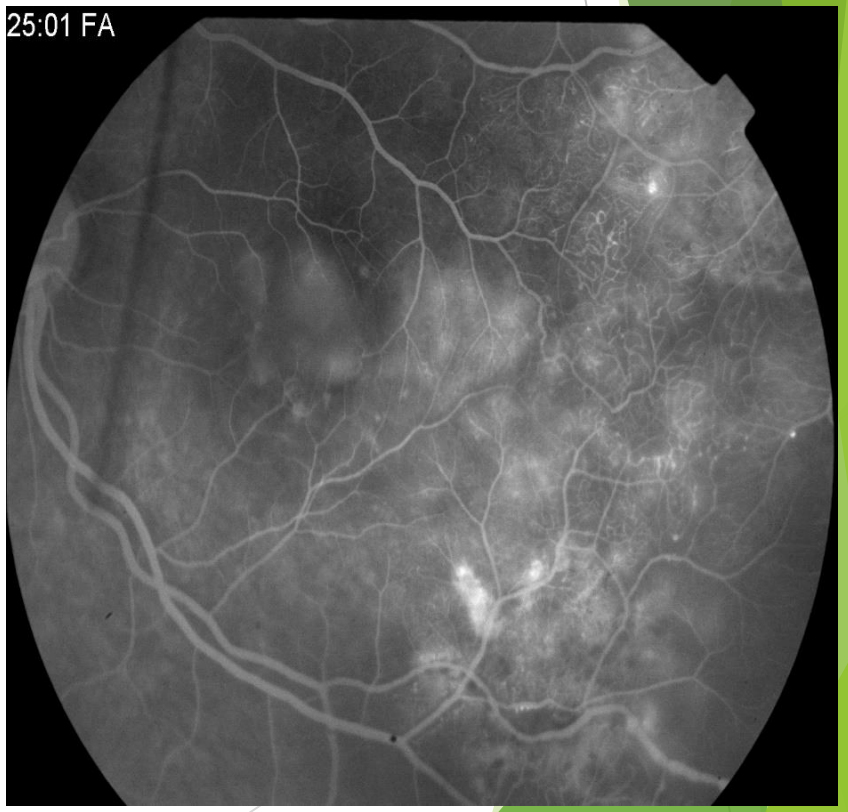
- Clinical picture variable
- IVFA / OCTA most helpful tool in making diagnosis
- Progressive with exacerbations and remissions
- Tx with photocoagulation, possibly combined with anti-VEGF and / or steroids

Coat’s disease (images courtesy Dr. Dan Neely)

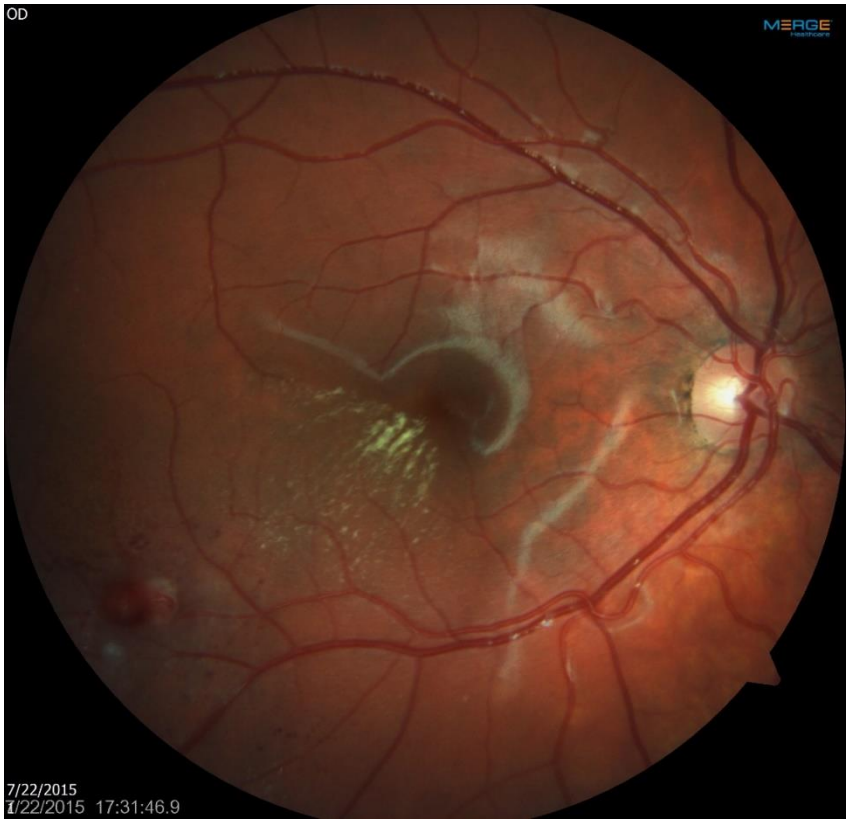


Coat's IVFA



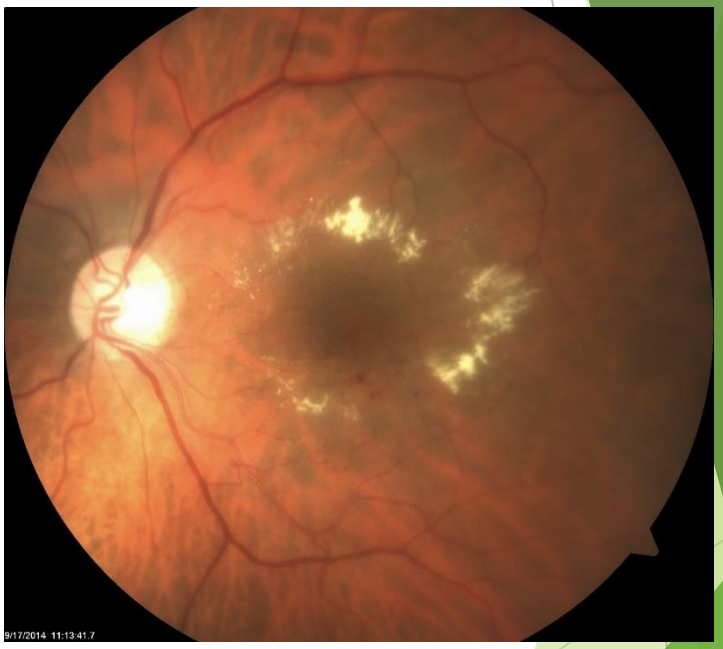


Coat’s disease

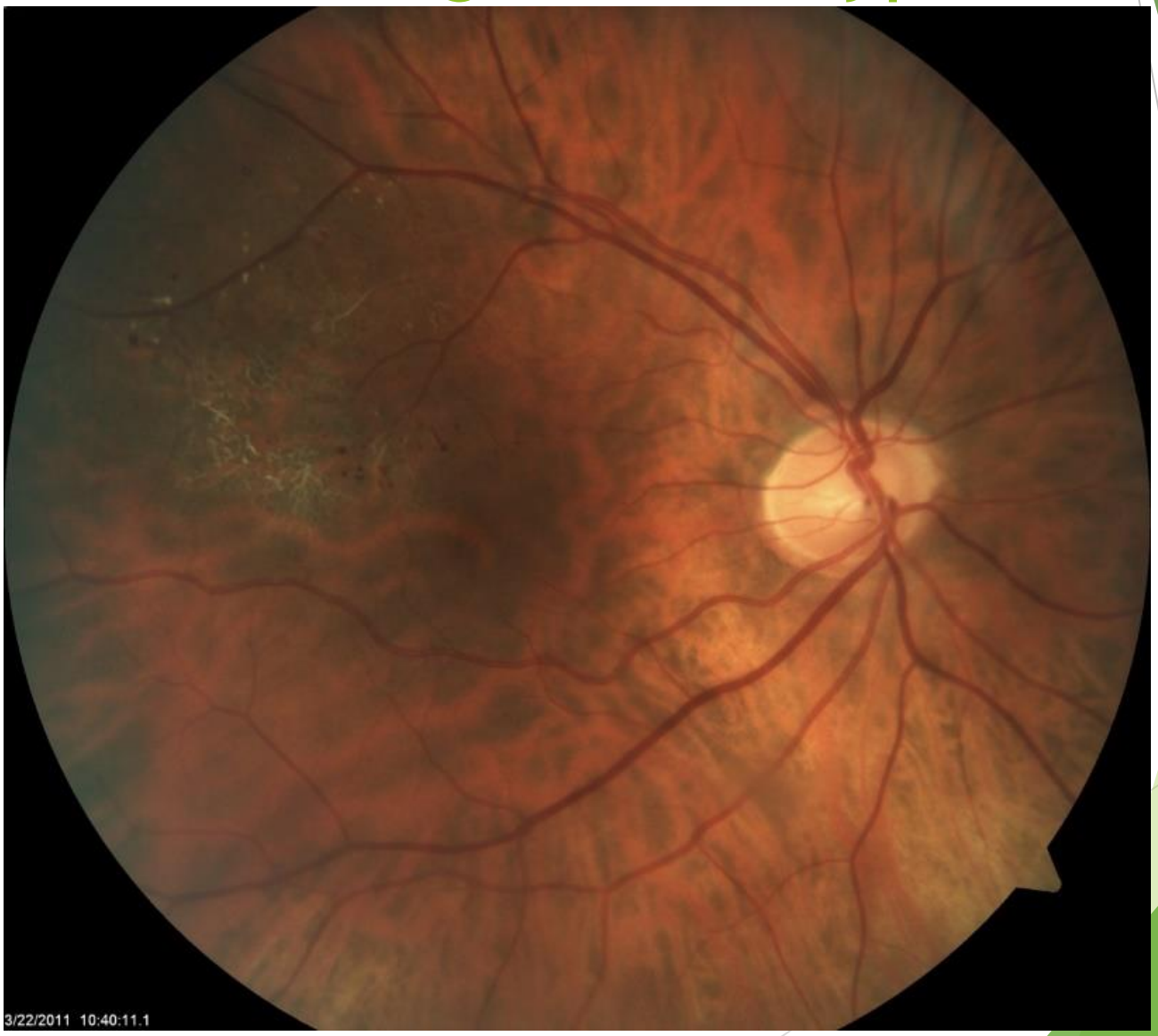


Macular Telangiectasia Type I

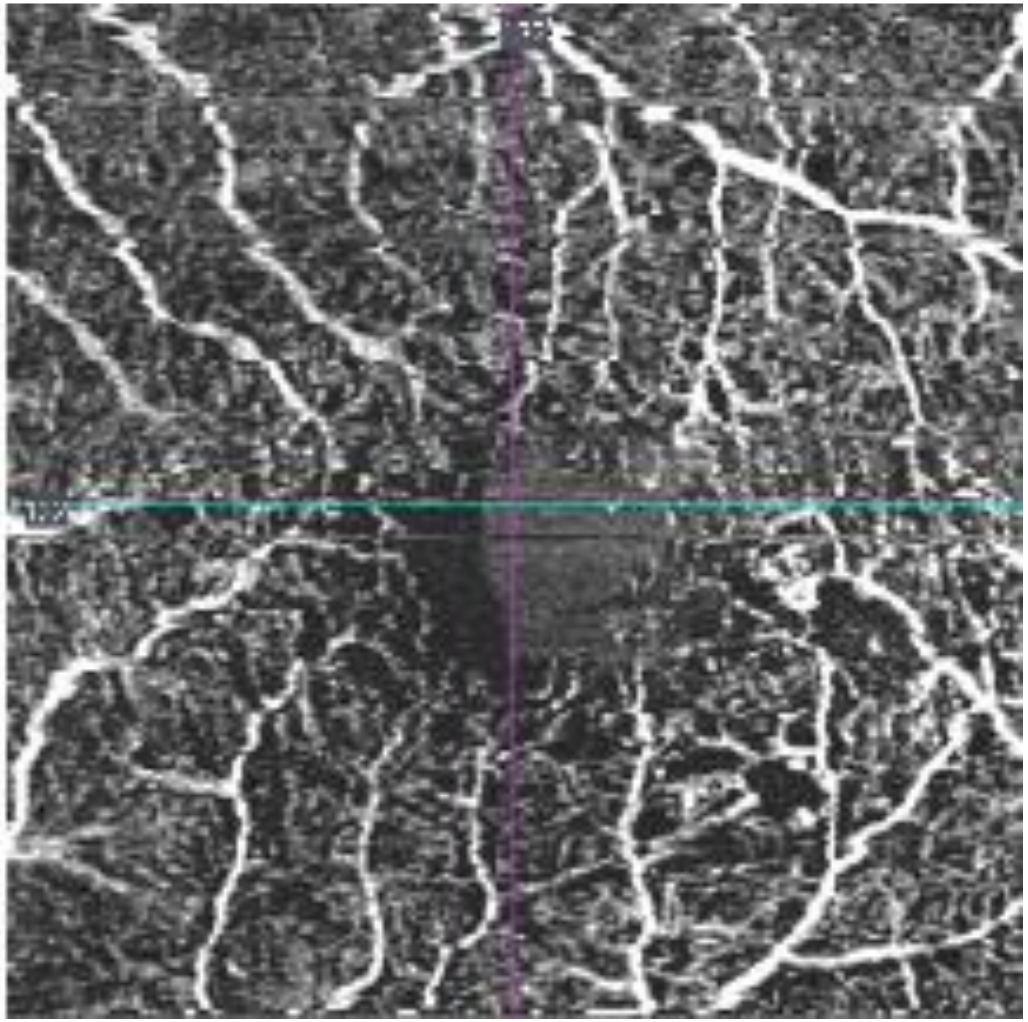
- Unilateral
- Mostly males, no racial predilection
- Mean onset 40 years old
- Prominent, visible telangiectatic capillaries
- Capillary drop out on OCTA
- Lipid exudation
- Macular edema
- Exacerbations and remissions
- IVFA, OCT / OCTA to confirm exudative edema
- Treat with laser photocoagulation, +/- anti VEGF or steroids

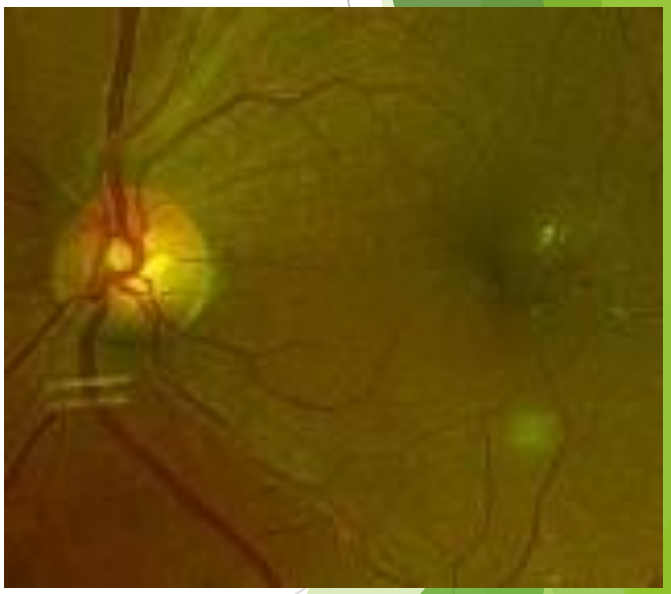


Macular Telangiectasia Type I



Mac Tel Type I

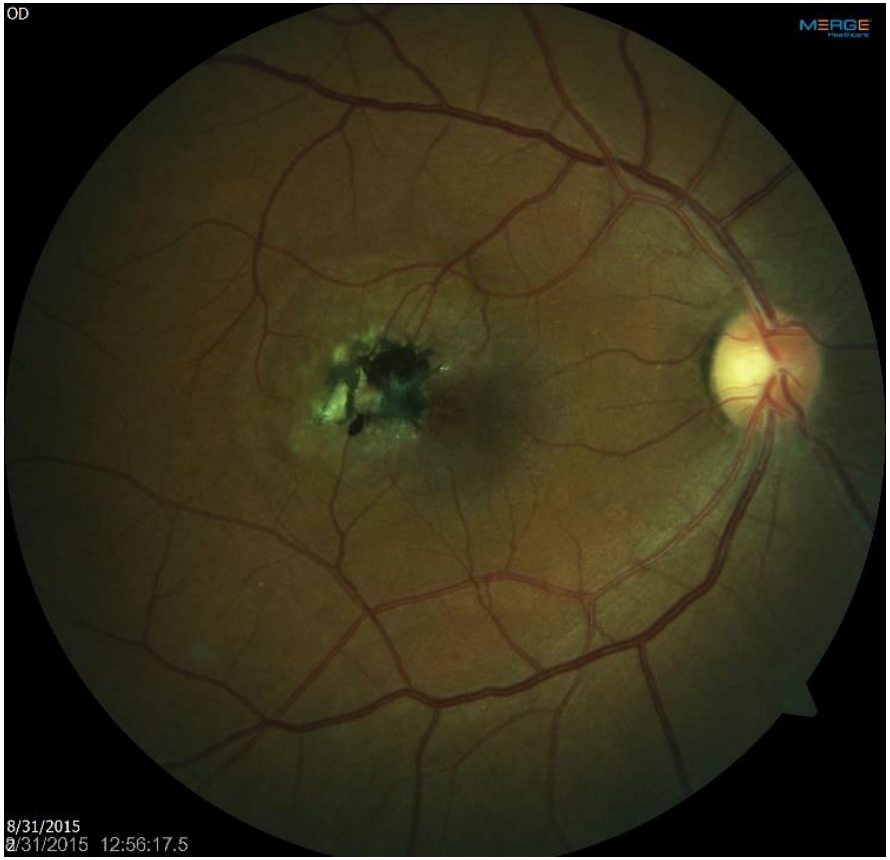


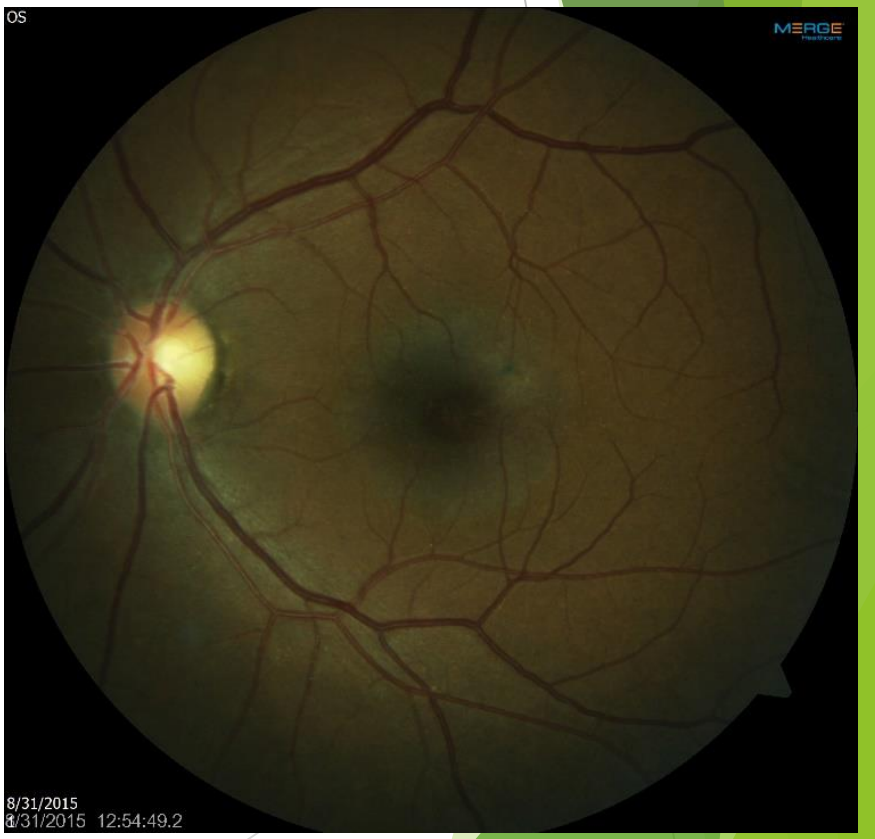


Macular Telangiectasia Type II

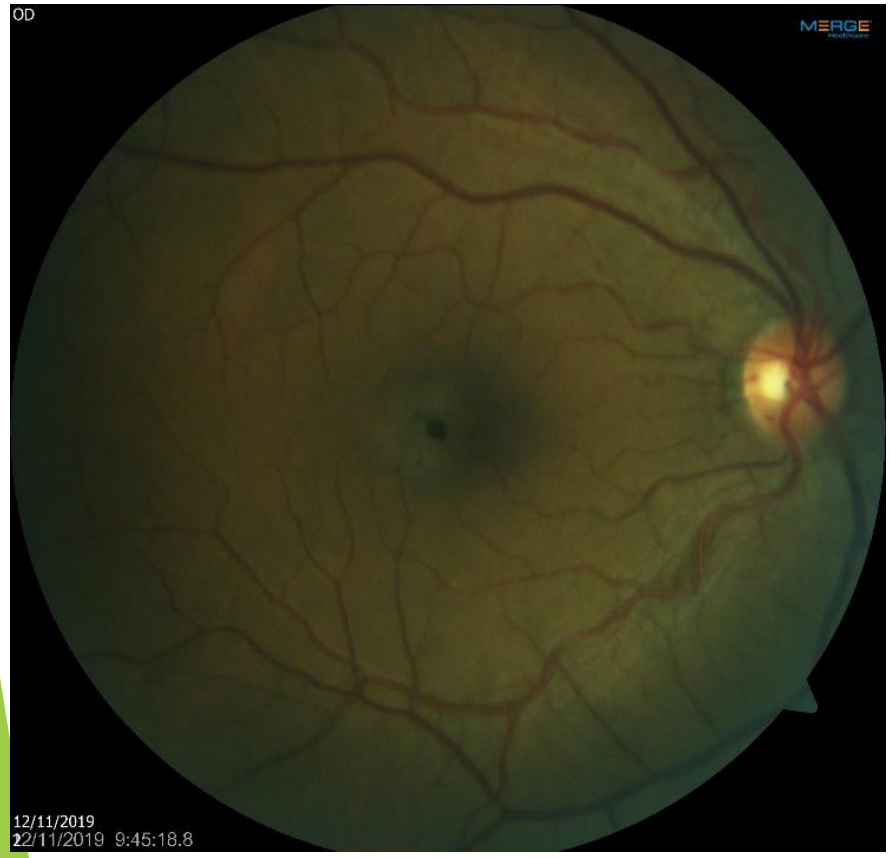
- No gender or racial predilection
- Mean age of onset 55 years old, bilateral
- Mueller cell depletion: may be primarily neurodegenerative
- Little to no exudation
- Parafoveal graying of the retina, parafoveal telangiectasias, crystalline deposits, macular edema
- ILM drape on OCT, right angle venules on OCTA
- Two subtypes: proliferative (1/3) and non-proliferative (2/3)
- Proliferative develop SRNVM and subsequent scarring
- Limited treatment options for non-proliferative
- Anti-VEGF for proliferative

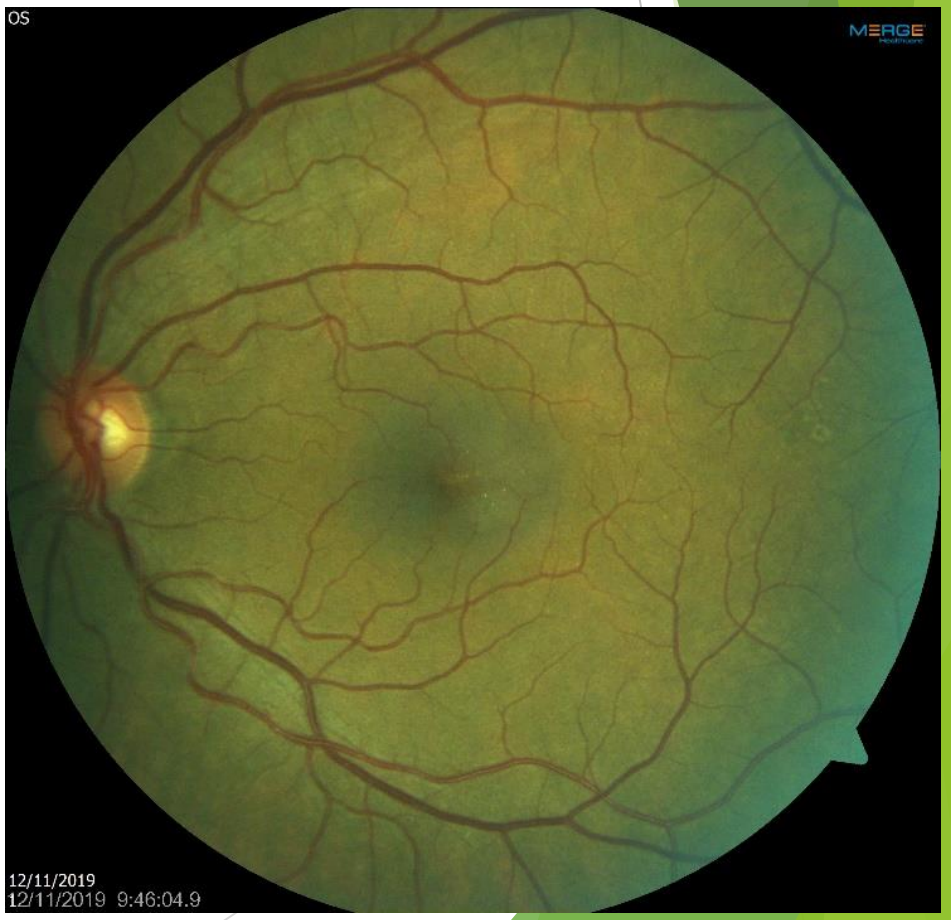
Macular telangiectasia Type II



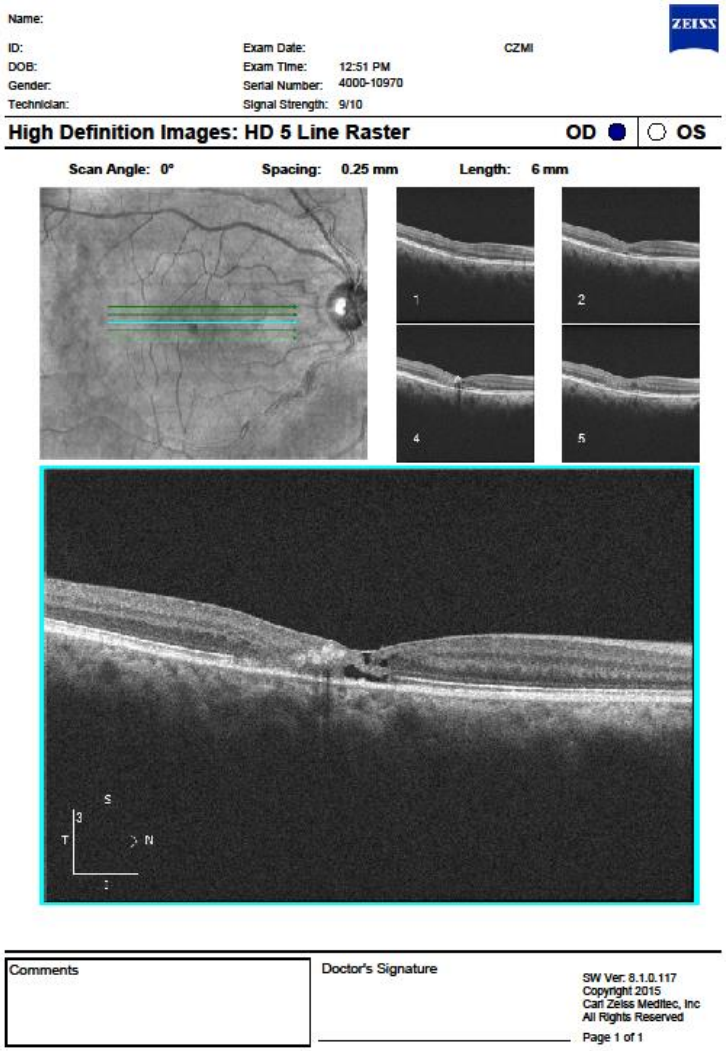


Macular Telangiectasia Type II



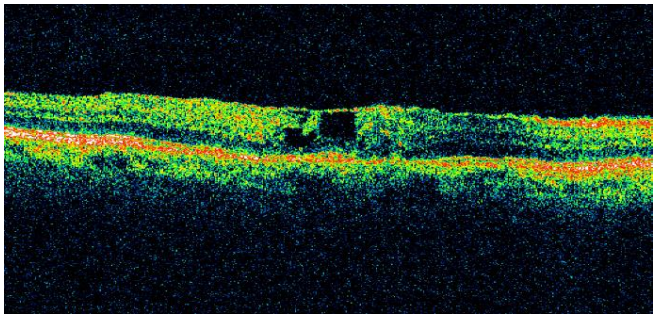


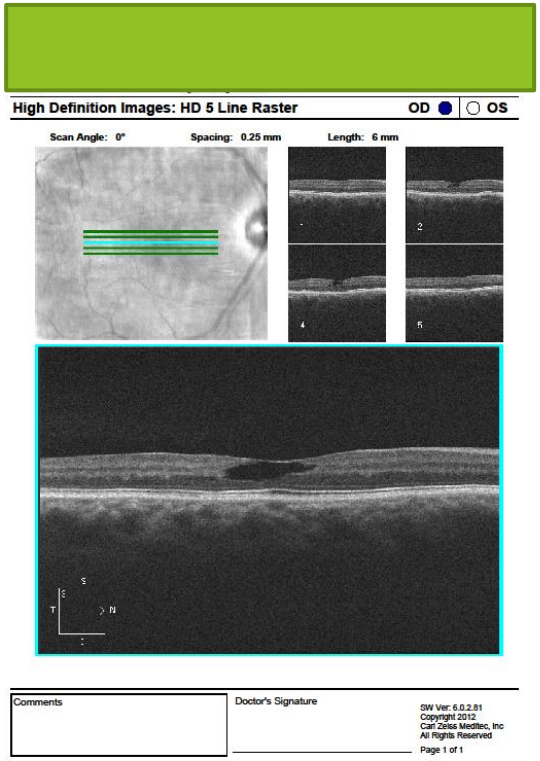
ILM Drape Mac Tel Type II



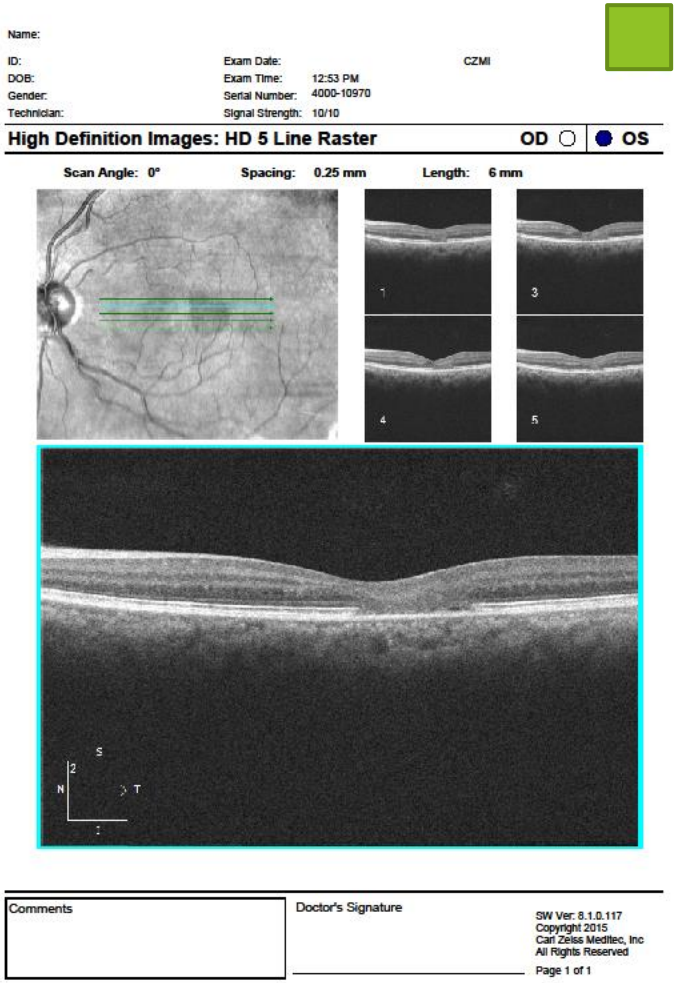
ILM Drape in Mac Tel II

Also get right angle venules on OCTA / IVFA





ONL / Photoreceptor loss in Mac Tel II



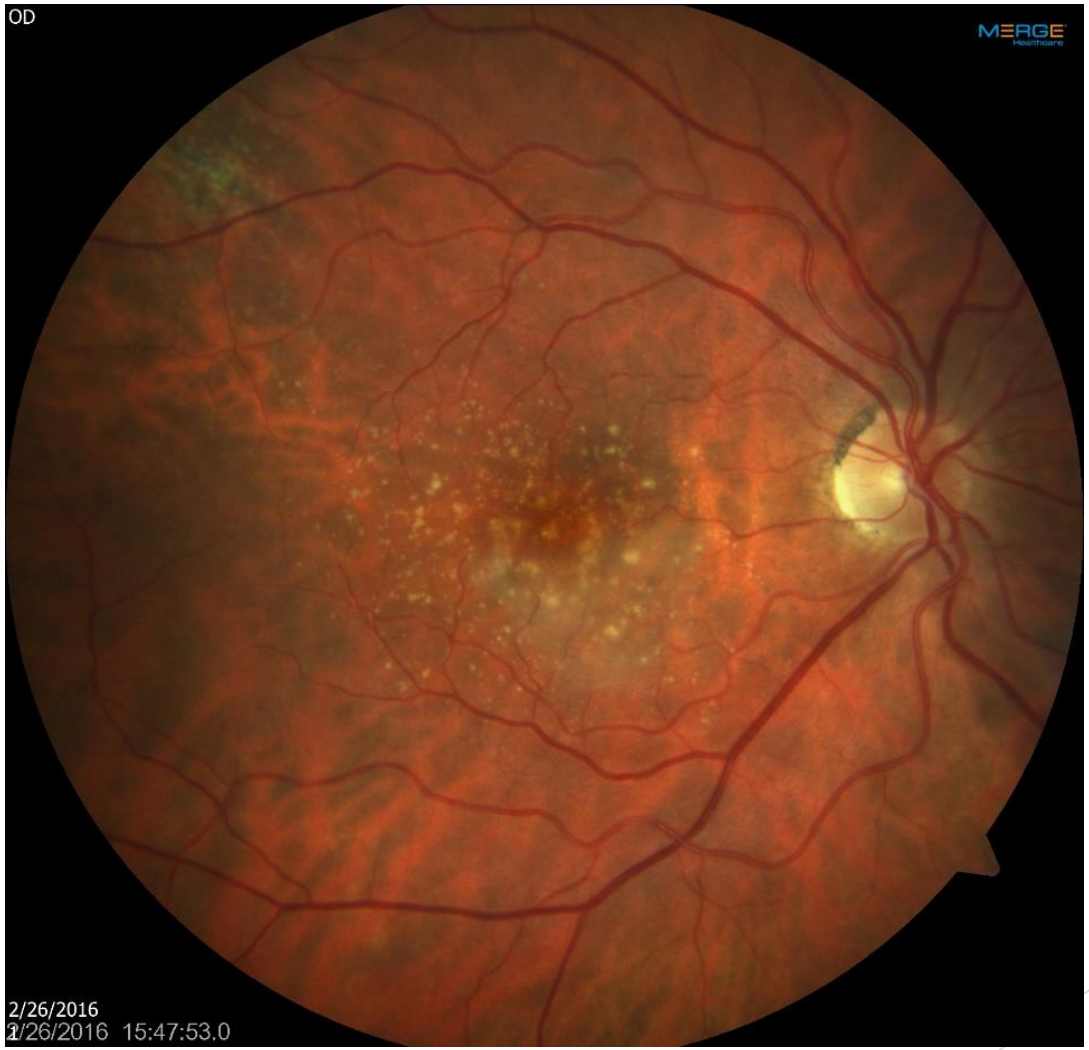
Macular Telangiectasia Type III

- Very rare
- Bilateral
- Perifoveal capillary obliteration and telangiectasias
- Minimal exudation
- Associated with systemic or cerebral disease.
- Poorly understood

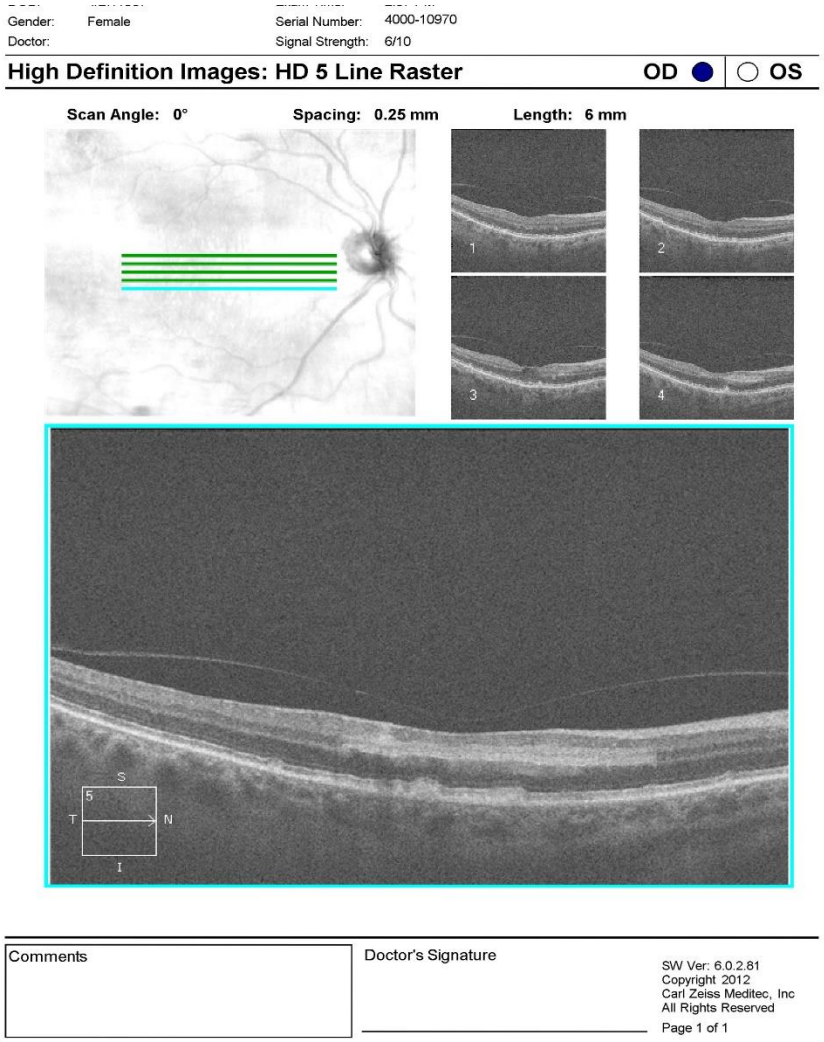
Middle maculopathy

- Paracentral acute middle maculopathy (PAMM)
- Acute macular neuroretinopathy (AMN)
- PAMM affects middle retina (INL)and is an infarct of the intermediate capillary plexus
- AMN affects the outer retina (OPL/ONL/PIL line area) and is an infarct of the deep capillary plexus
- Can be younger supposedly healthy patients
- Can have a flu like prodrome
- Unilateral or bilateral
- Sudden onset of central or paracentral scotoma
- Slow resolution of defect, but may be permanent
- Can be seen with diabetes and other vascular or retinal vascular conditions / vasculitis
- No treatment other than underlying disease management

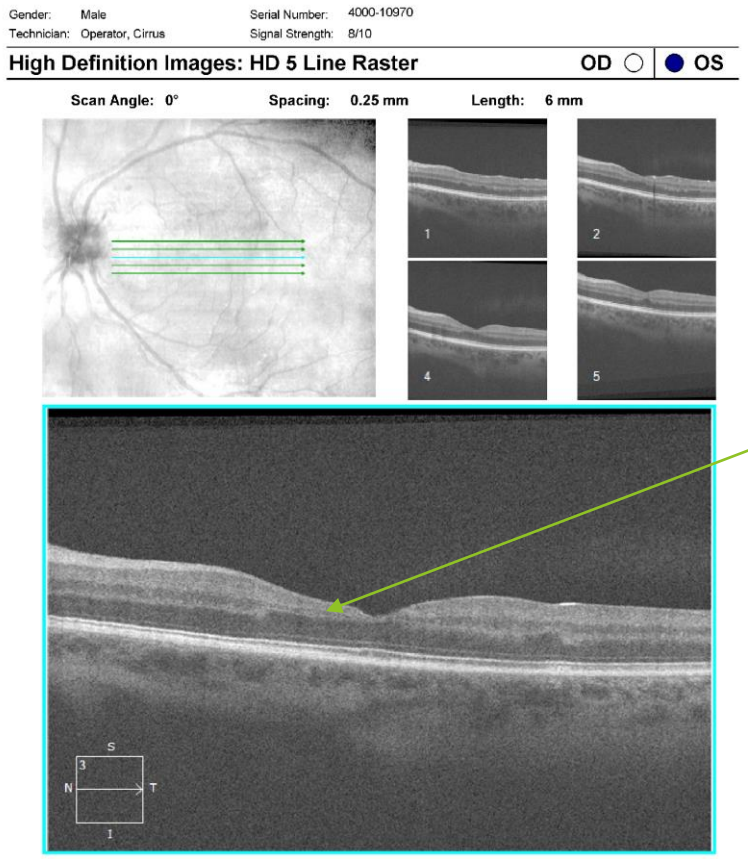
Middle Maculopathy (PAMM)



Middle Maculopathy (PAMM)

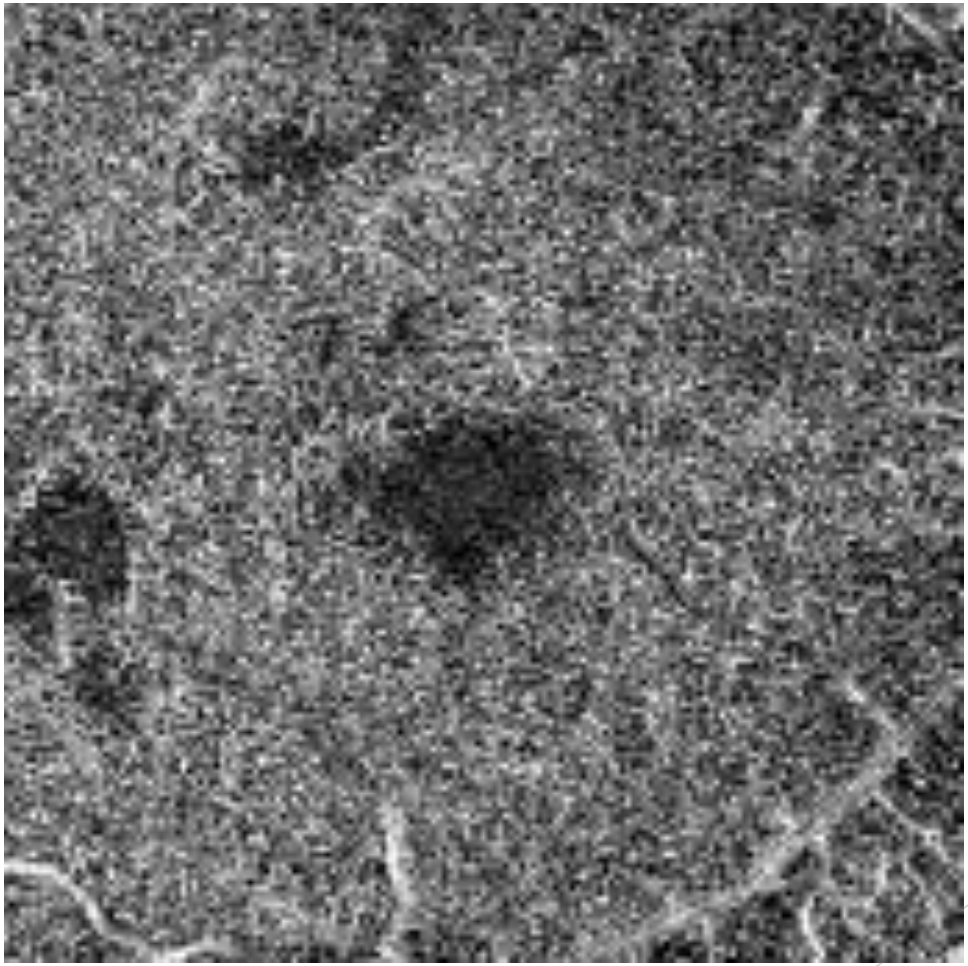


Old PAMM



Collapse

PAMM deep capillary plexus OCTA



PAMM

- 2019 study of 45 BRVO patients, 21 CRVO patients, and 57 matched normals showed resolved PAMM lesions in the fellow eye of 71% of CRVO patients, 71% of BRVO patients, and in at least one eye of 19% of the matched normals (all of which had systemic HTN)
- *Acta Ophthalmologica 2019 Maltsev, DS et al*

- So the presence of resolved PAMM lesions may indicate a higher risk of RVO

Artery Occlusions

- Embolus (frequent) vs. local thrombosis (rare)
- Other factors include vasospasm, necrosis, GCA (10%), and hyperhomocystinemia
- Can affect the ophthalmic artery, cilioretinal arteries, and retinal arteries
- Risk after facial filler injections (particulate material forced backward through the vascular system into the ophthalmic artery / central retinal artery)
- 2.3 x risk of CRAO in patients with diabetes

Types of embolic plaques

- Hollenhorst (cholesterol) ; about 80% of retinal emboli
- Calcific; about 6 % of retinal emboli
- Fibrino-platelet; about 14% of retinal emboli

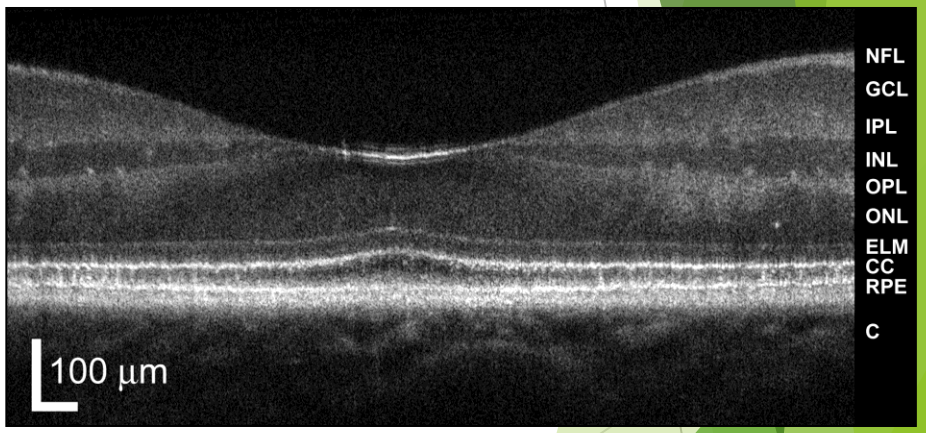
- H-H plaque mortality:
- 15 % @ 1 year
- 29 % @ 3 years
- 54% @ 7 years

CRAO

- CRAO characterized by sudden, painless, profound loss of vision. VA count fingers or worse in 75-90%
- Possible amaurosis fugax
- Retina can appear normal for first hour or so

CRAO

- Emboli visible in only 20% of cases (carotid, heart valves)
- Within hours the retina (posterior pole) becomes white and opaque due to ischemic NFL edema.
- Cherry red spot due to lack of ganglion cells in the foveola
- Box-car changes in retinal vessels due to lack of blood flow



CRAO

- Over the course of about one month, the retinal appearance returns to normal
- Residual optic atrophy / vessel attenuation long term
- 25-40% of cases have some sparing of the macular area due to cilioretinal artery perfusion

CRAO

- Sparing can result in reasonable visual recovery
- Without cilioretinal vessel perfusion or short-term blockage, VA does not make a meaningful recovery in most cases
- NVI,NVD,NVE are very rare complications
- Why is that? Because the retina dies so quickly, it can not send out a signal for VEGF. Neovascularization more likely in eyes with glaucoma and patients with chronic kidney disease, especially if on dialysis.

CRAO

- Life expectancy of 5.5 years compared to 15.4 years for age matched

- Treatment is notoriously ineffective
- Digital massage, IOP lowering (paracentesis), rebreathing in paper bag
- What are we really doing? Trying to dislodge the embolus (which is often already gone).
- Possibly TpA?
- ? ND-YAG lysis of emboli
- ? Hyperbaric Oxygen

Intravenous TpA

- Very controversial
- Some deaths from cerebral and hepatic hemorrhaging with Streptokinase in various trials
- Short window to be effective: likely must be within 4.5 hours of symptom onset. Practically, this is very unlikely to occur

- One meta-analysis of available trials and studies showed that patients starting with VA of 20/200 or worse, the chance of improving to 20 /100 or better was……..
- 17.7% without intervention
- 7.4% with traditional massage / paracentesis, etc.
- 50% with IV TpA within the first 4.5 hours
- Many questions remain, however
- Very controversial, risky

CRAO: Hyperbaric Oxygen therapy

- 2018 Meta Analysis of 7 RCT's, 251 patients
- Oxygen therapy delivered early = 5.61 X chance of improved vision
- 100% hyperbaric O2 delivered soon after event for 9 or more total hours best

Types

- Believed to be four sub types of CRAO……each with a unique natural history (SS Hayreh)

- I) NA-CRAO
- II) NA-CRAO with C-R artery sparing
- III) Transient NA-CRAO
- IV) Arteritic CRAO

CRAO

- Chance of any degree of visual recovery depends entirely upon sub-type and presence or absence of complete occlusion
- Transient NA-CRAO and NA-CRAO with CR artery sparing may improve
- NA-CRAO improvement is rare but can occur if occlusion was not complete
- Arteritic CRAO very, very rarely improves
- Improvement possible over first seven days or so

BRAO

- BRAO's reportedly occur less frequently than do CRAO's (?) 68% have visible embolus
- A smaller embolus , usually temporal to the macula (80%)
- Appearance is similar but in a localized area
- Susac syndrome (triad of encephalopathy, BRAO's, and hearing loss) most common in young women.

BRAO

- VA usually good: scotoma!
- Survival rates are actually lower than for CRAO
- Cilioretinal and ophthalmic artery obstructions are also possible: think GCA!

BRAO / CRAO

- Ophthalmic artery obstruction mimics CRAO but vision is usually NLP, there is no cherry red spot, and the findings are more pronounced
- Get carotids (cholesterol) and heart valves (calcium) checked as indicated. Check for GCA (ESR, CRP, CBC, platelets)
- Doppler of carotid checks neck only……not sections in thorax and skull
- Presence of plaques may be more important than level of stenosis (microemboli)
- Why would that be? Because there is enough blood flow past the blockage to dislodge an embolus
- 60% of CRAO patients do not have high grade ipsilateral carotid stenosis. 30% do not even have mild stenosis
- High rate of atrial fibrillation (AF) with CRAO. Equal to rate seen in ischemic cerebral stoke, and much higher rate than matched normal patients. Need to evaluate through cardiologist with continual halter monitoring

Hollenhorst plaque: Consider……..

- HHP with no visual symptoms and known cardiovascular disease > No Doppler. Consider 81mg Aspirin
- HHP with visual symptoms > Doppler indicated
- HHP with no known cardiovascular disease > full cardiovascular work-up

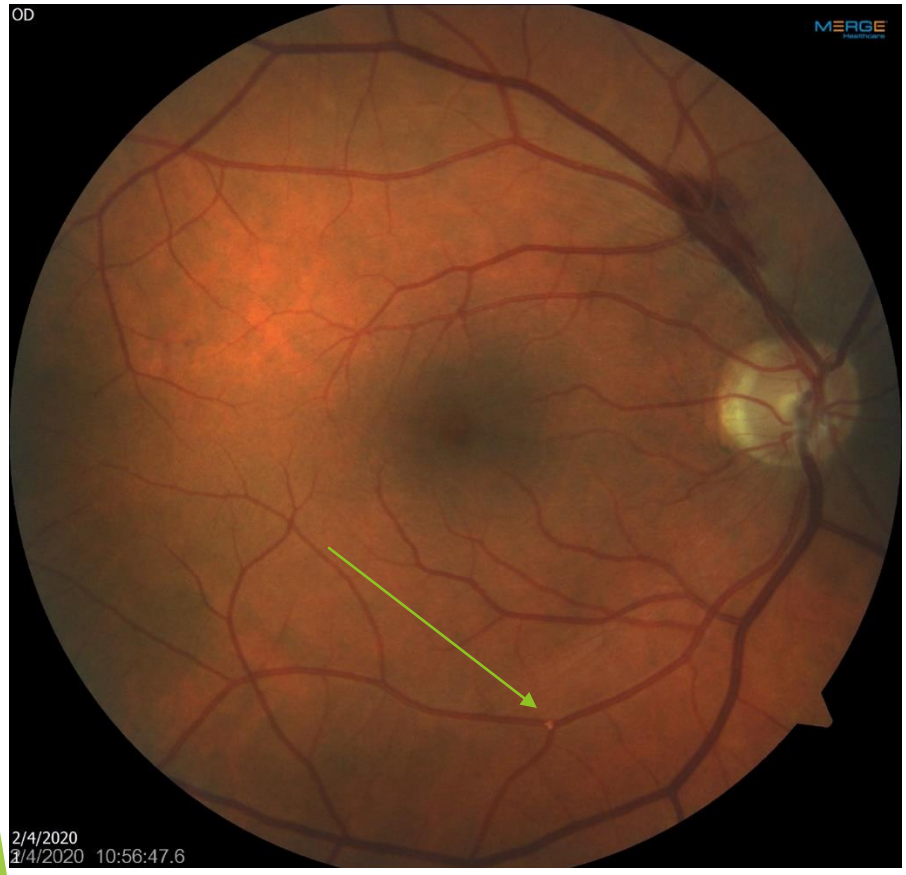
What about acute (often "silent") strokes with CRAO / BRAO

- One study found that 24% of patients with ocular TIA's suffered an acute cerebral stroke at the same time. Often symptom free ("silent")
- A second study looked at 33 patients with new onset CRAO or BRAO. 24% of them had concurrent acute (<14 days) cerebral strokes. Again, often "silent"
- Even more, newer reports as well finding the same thing

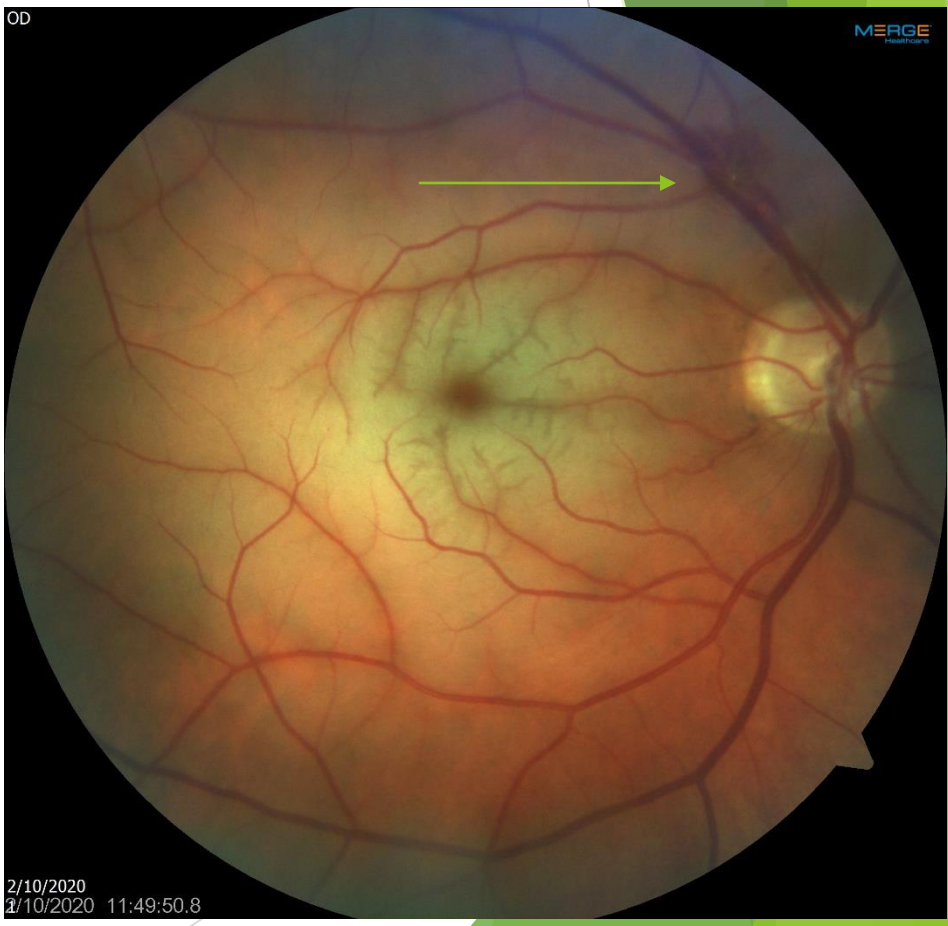
Acute strokes with CRAO / BRAO

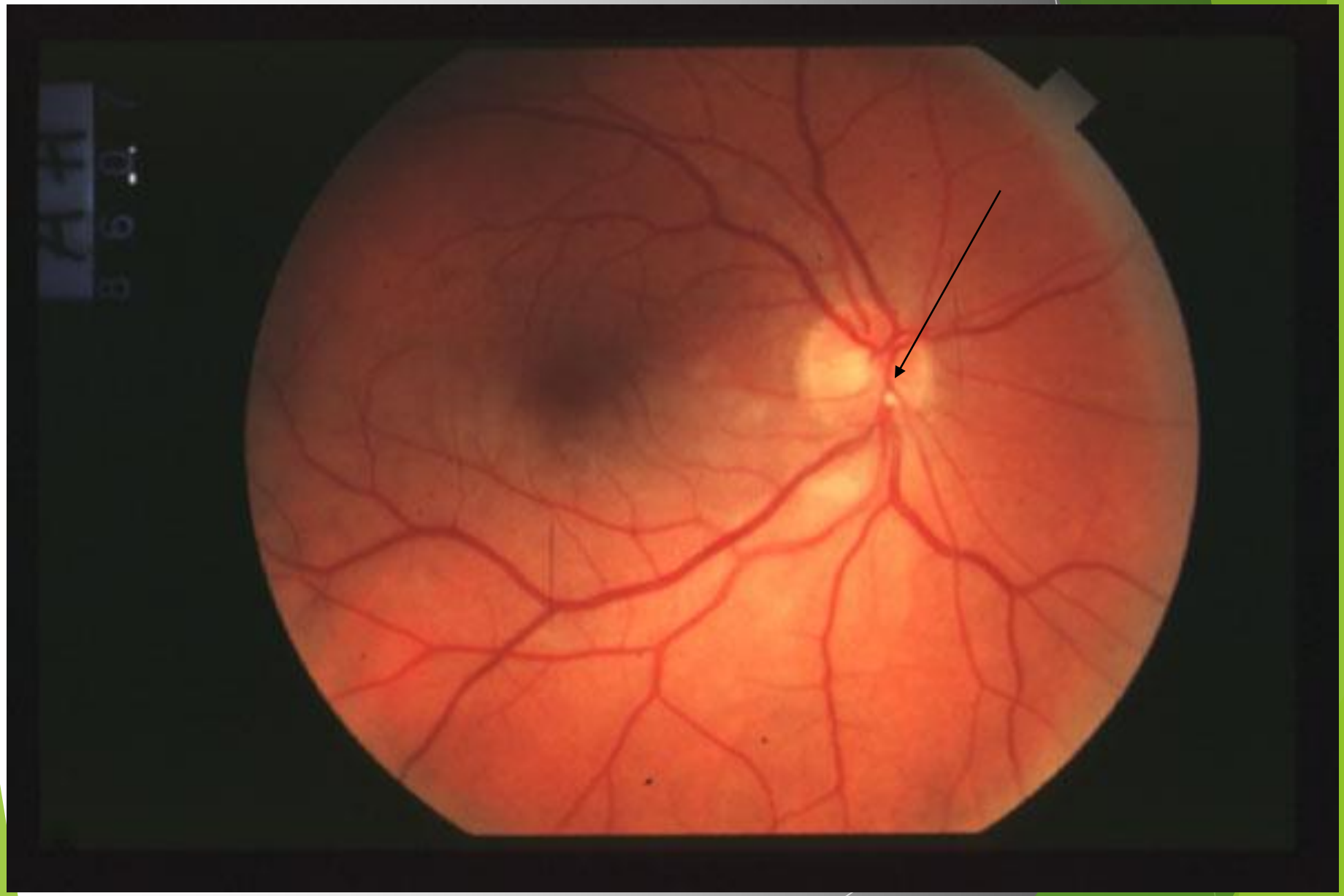
- Must run diffusion-weighted MRI, not standard T2-weighted MRI
- It better distinguishes acute infarcts from old lesions
- Recommendation from these studies, editorials, the American Heart Association and the National Stroke Association: Obtain emergent diffusion weighted brain MRI, preferably in a stroke center, on ALL patients with ocular TIA, BRAO, or CRAO
- Some controversy still, for several reasons (cost, what is then done, etc.)

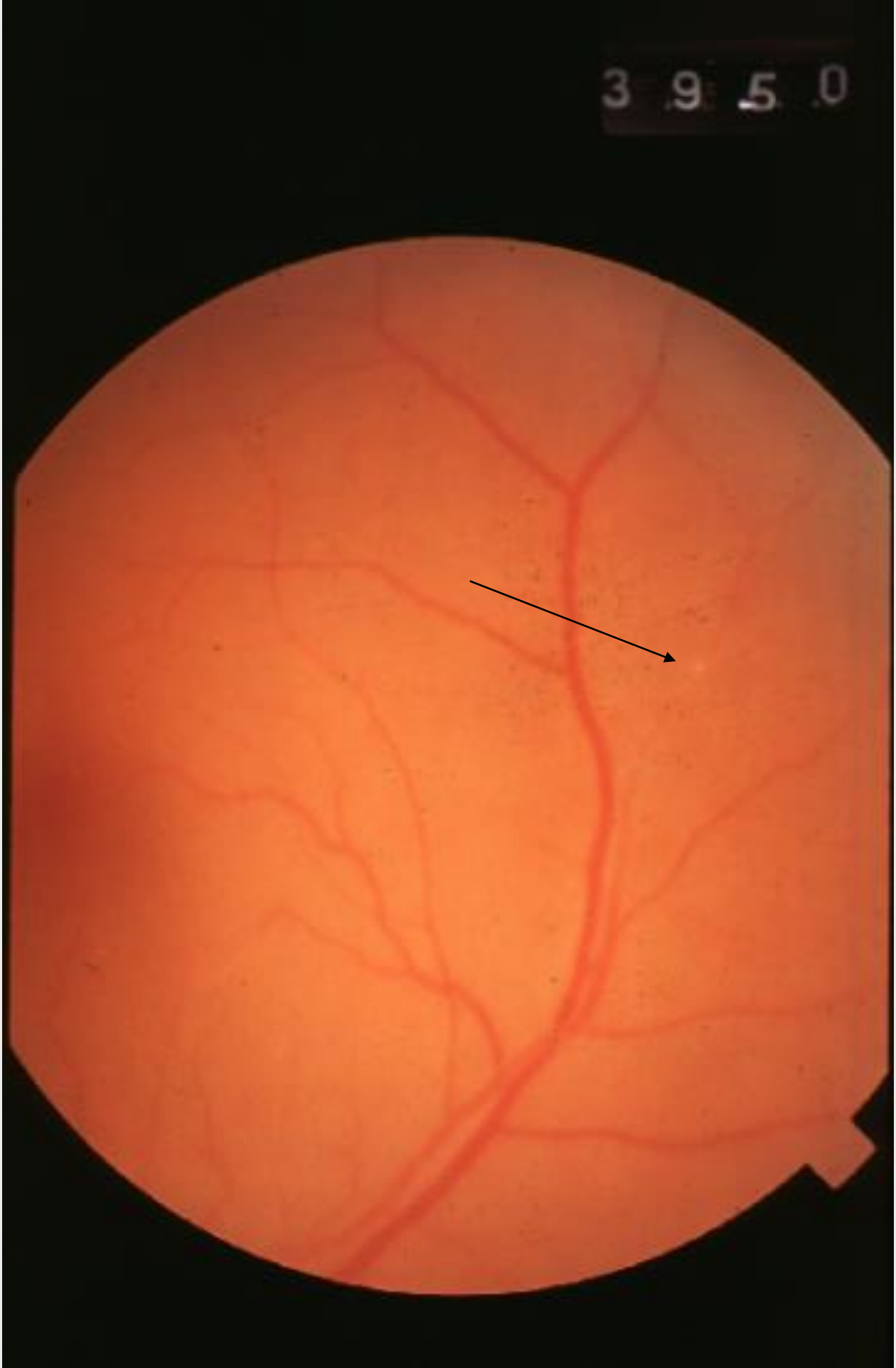
Hollenhorst Plaque-then CRAO



Six days later



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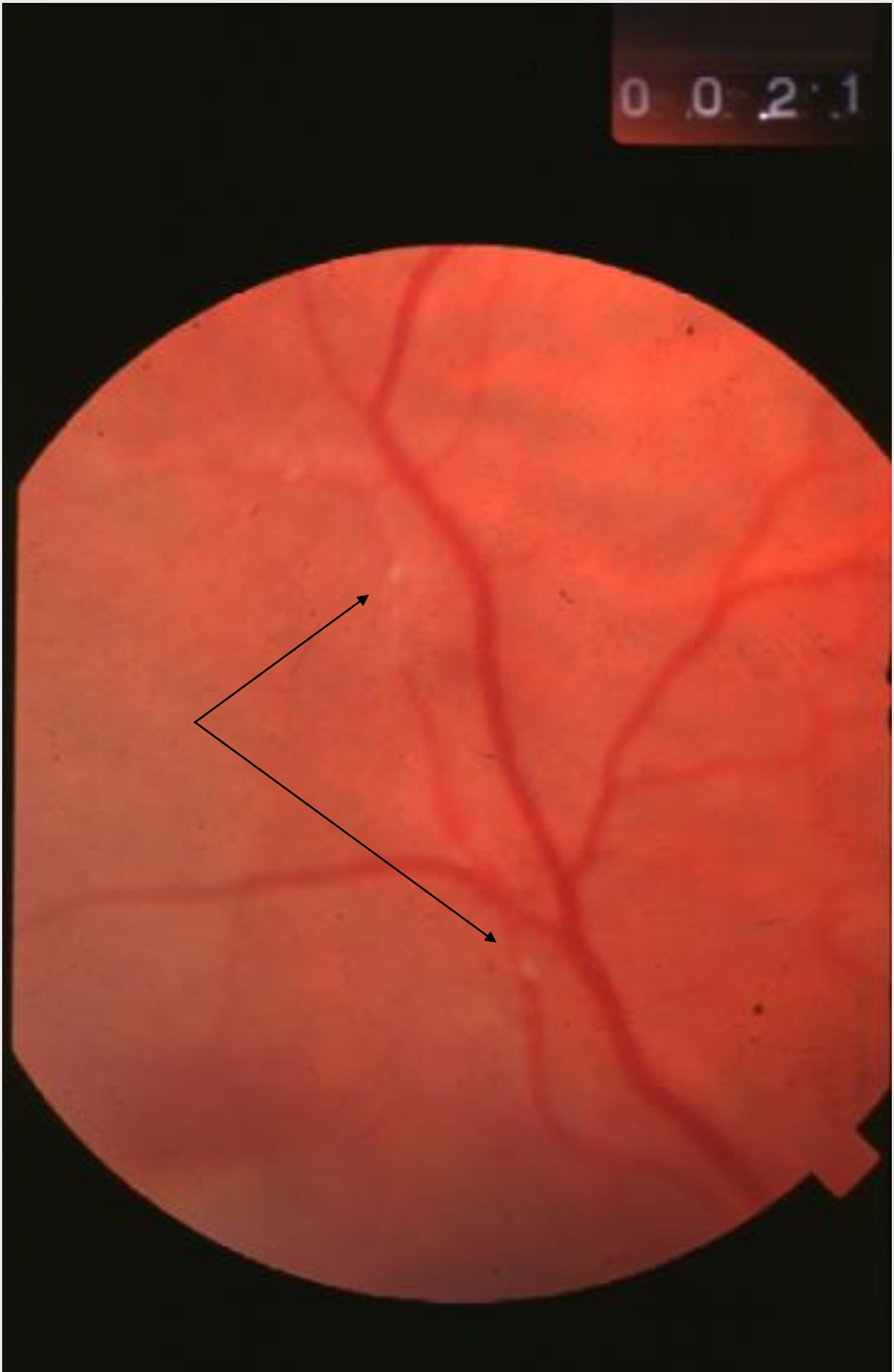
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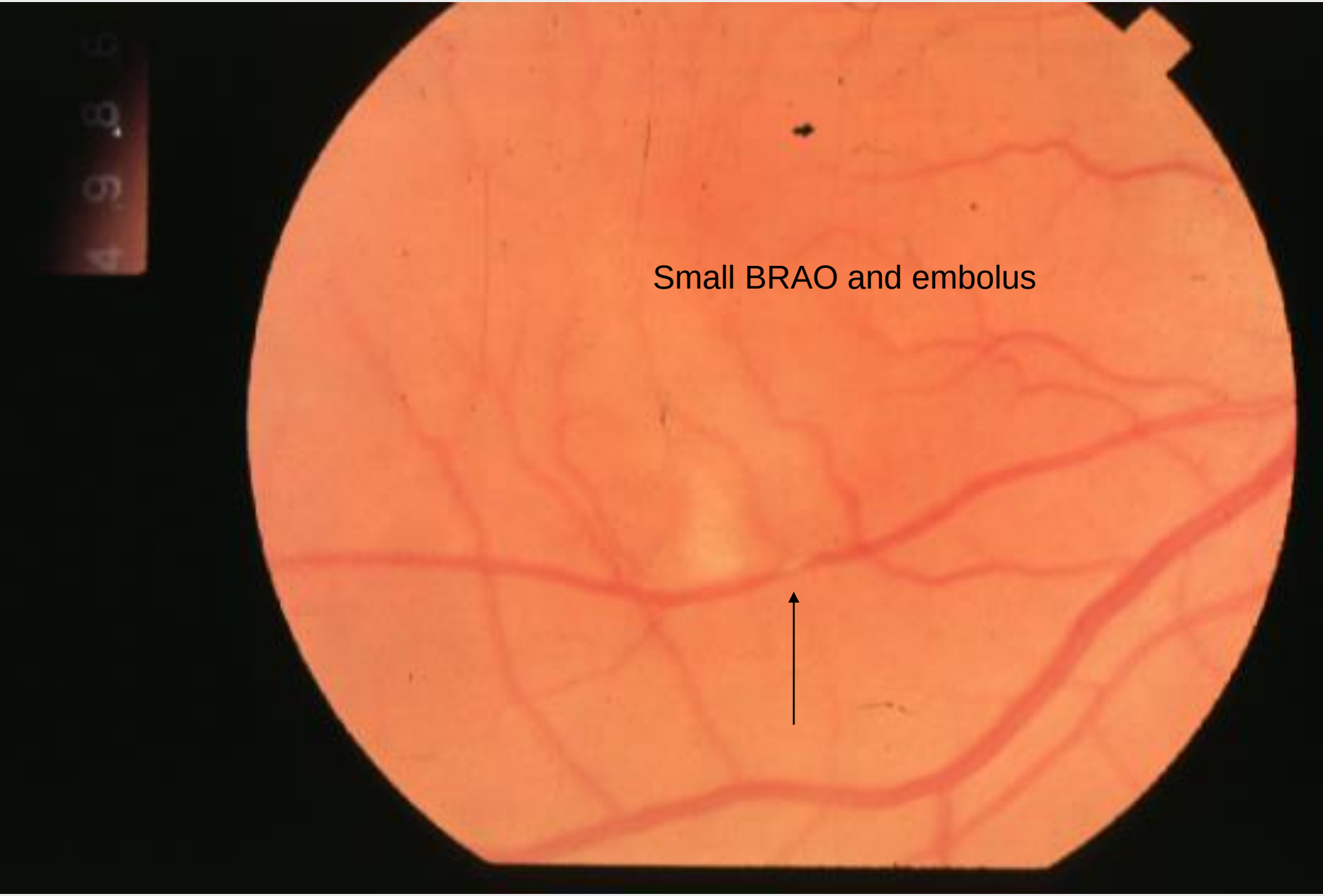
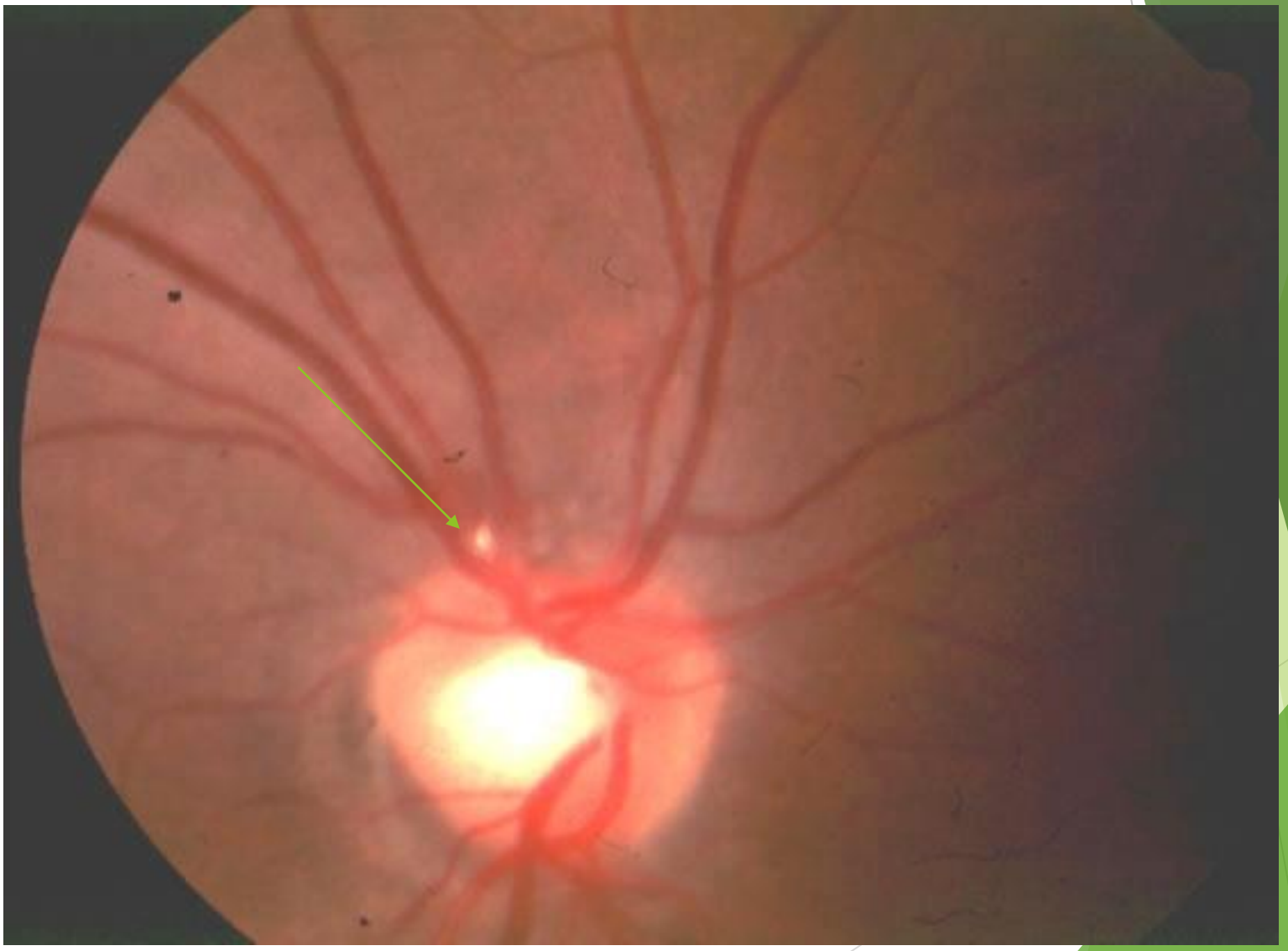
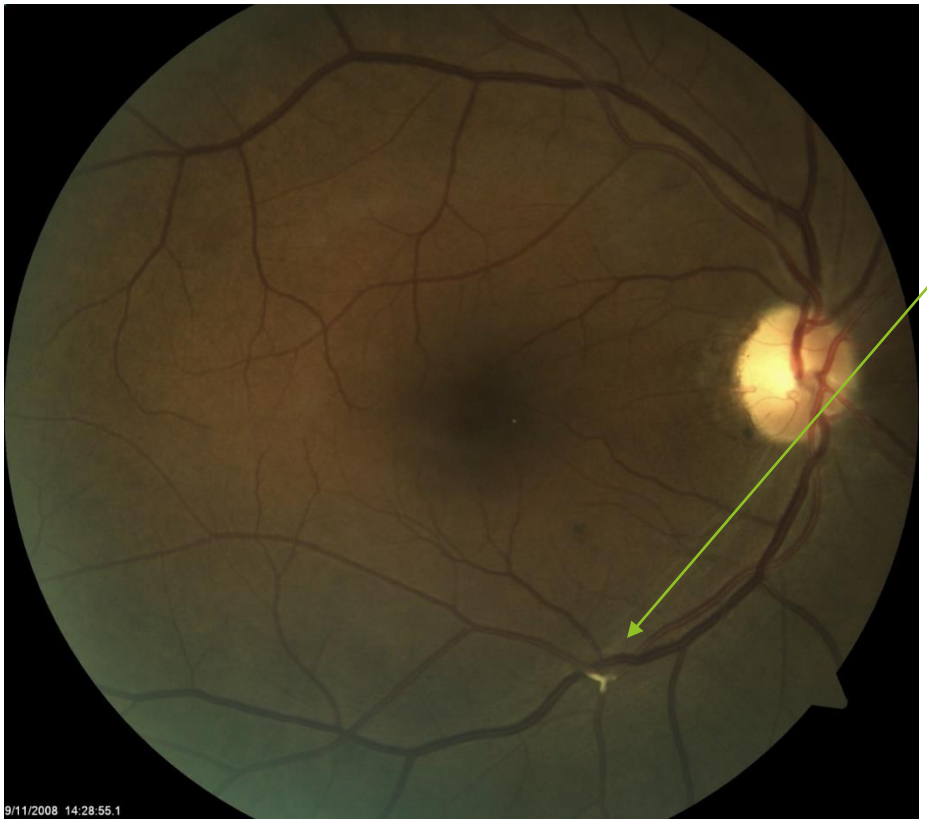

Small BRAO and embolus

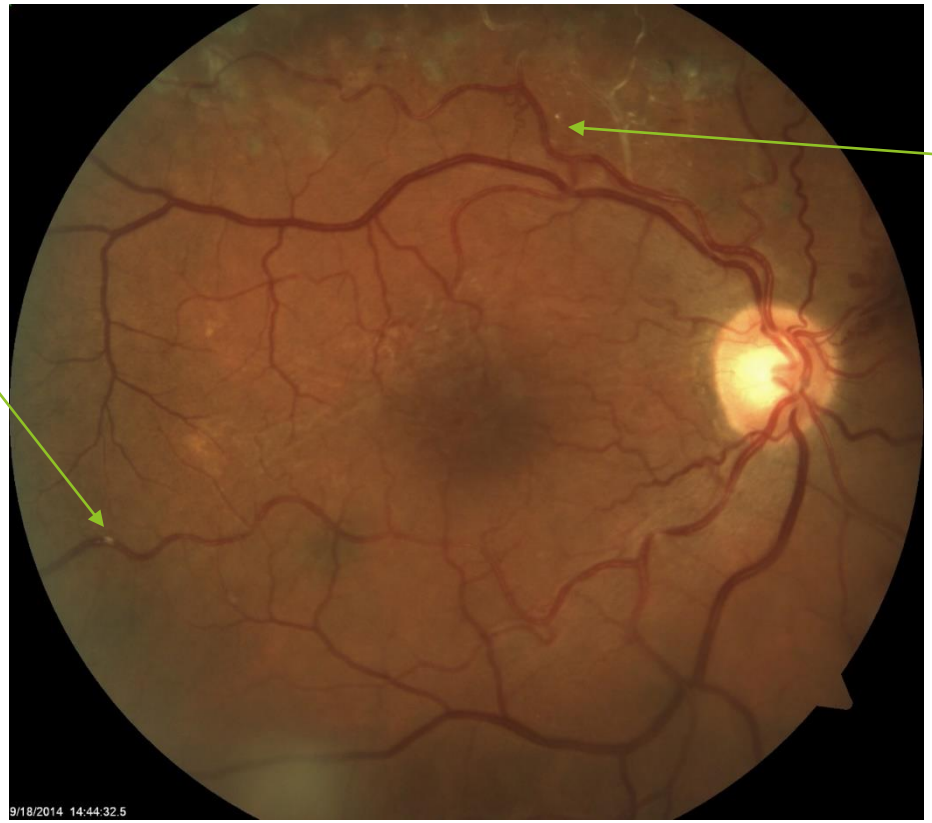
Calcium Embolus



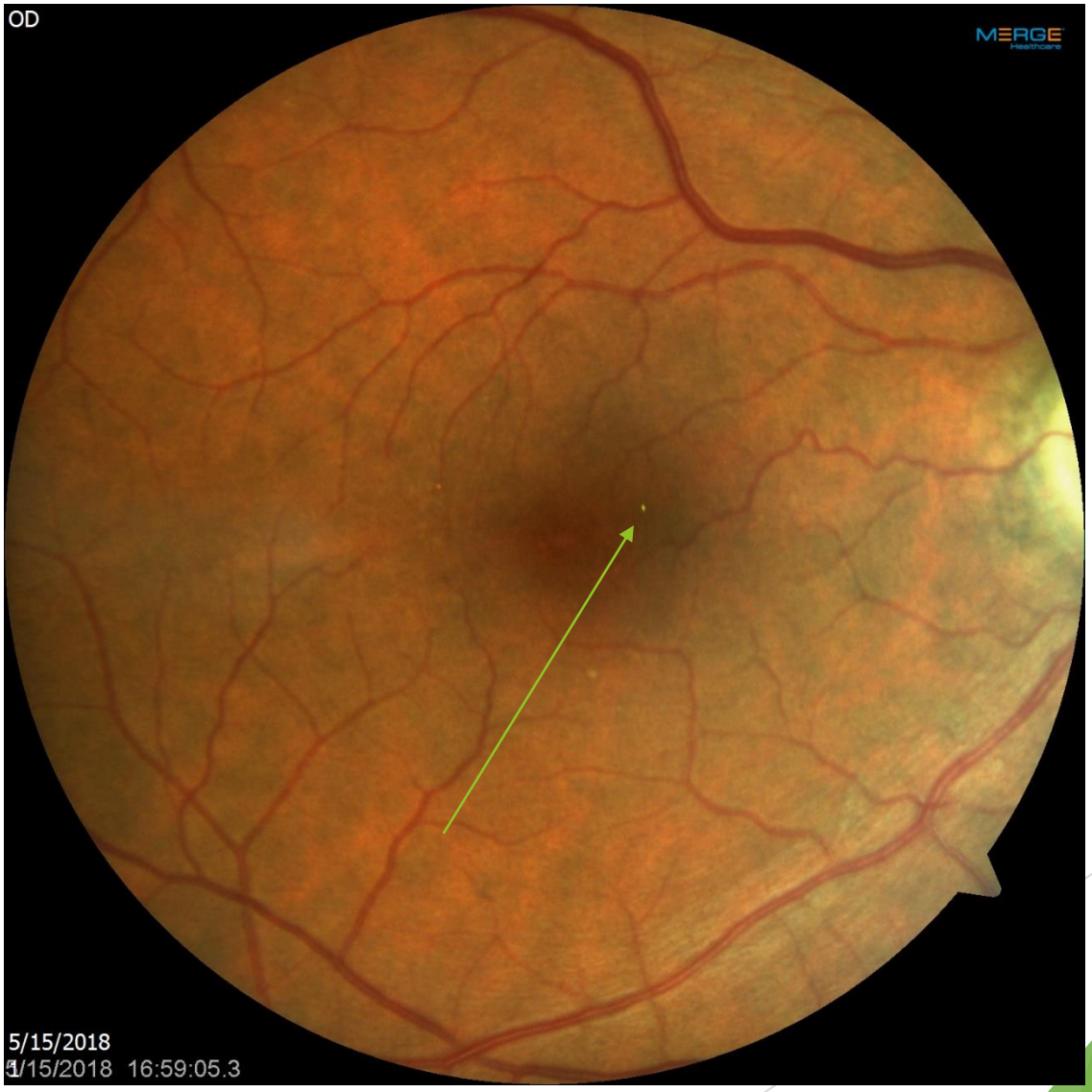
Bifurcation fibrinoplatelet plaque



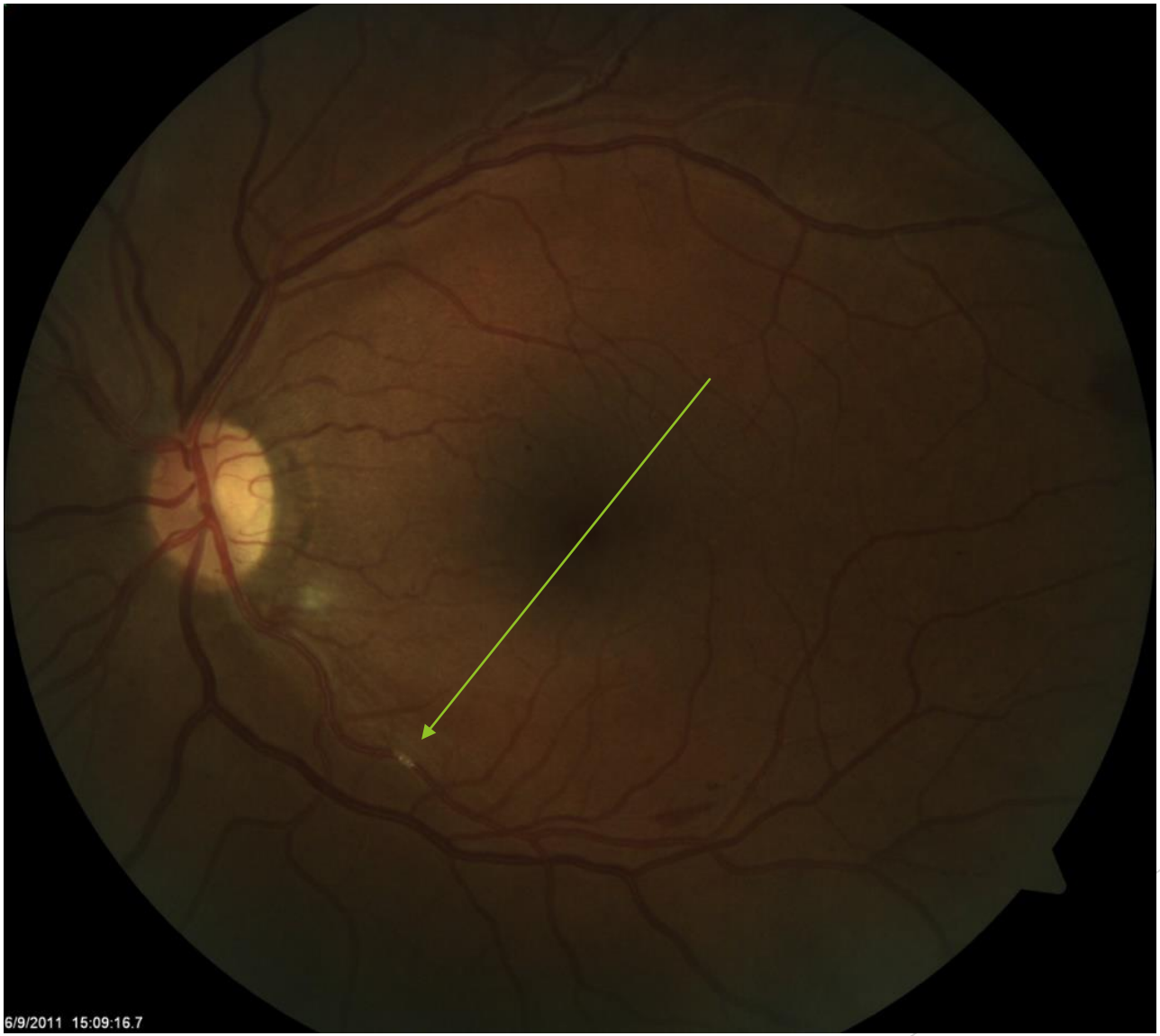
HH Plaque



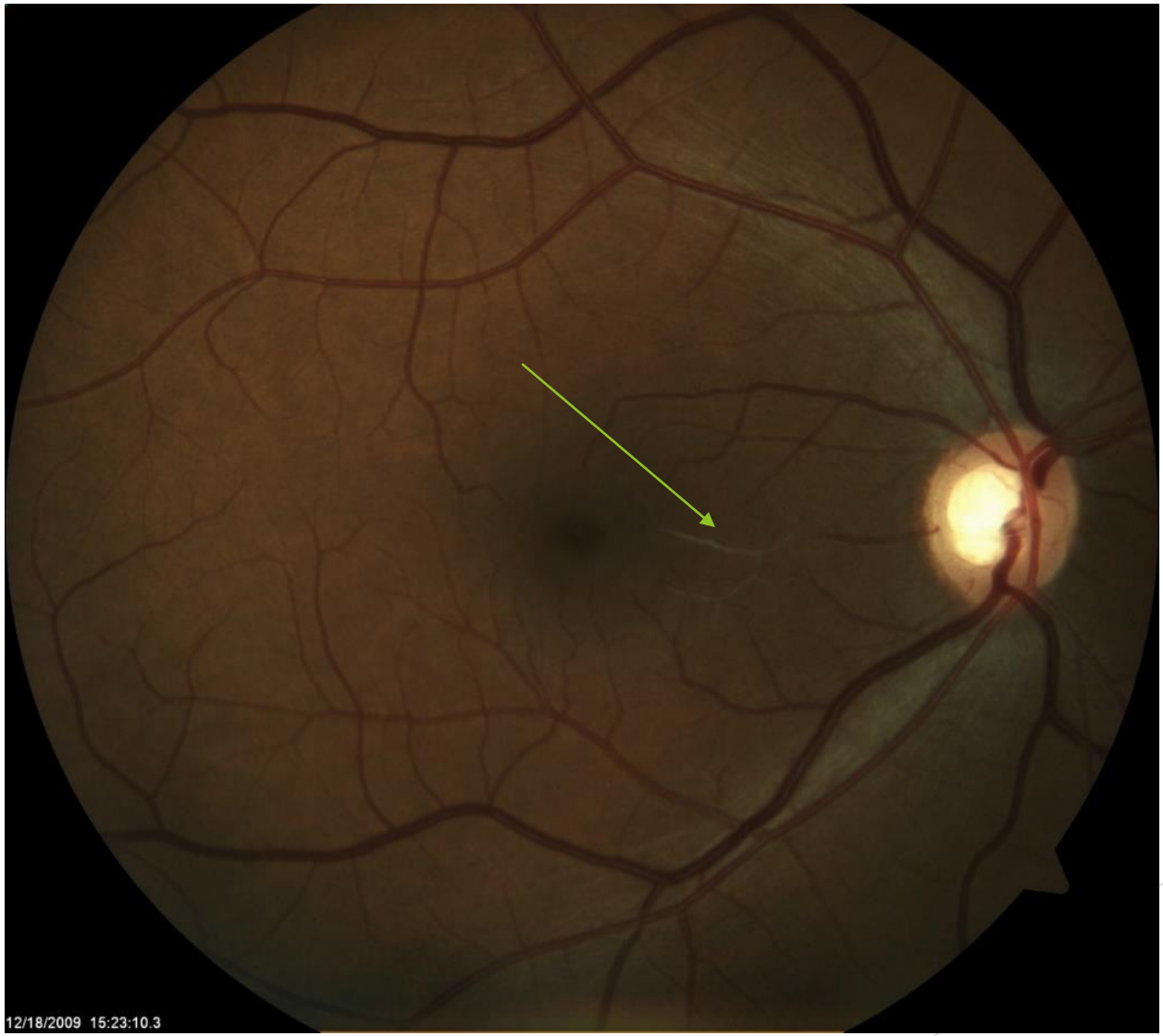
HH Plaque



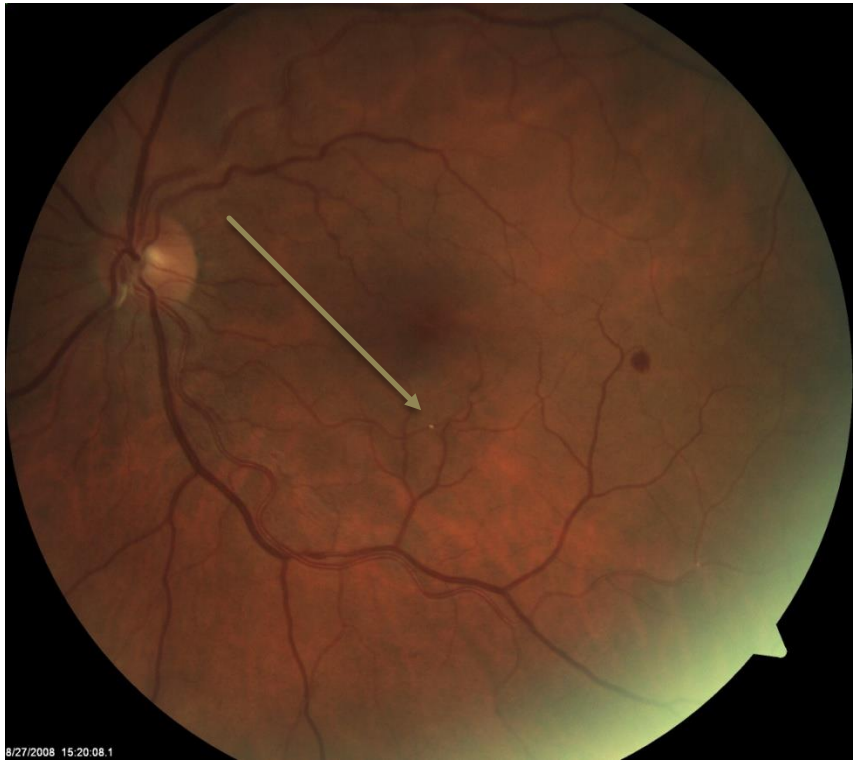
fibrinoplatelet plaque

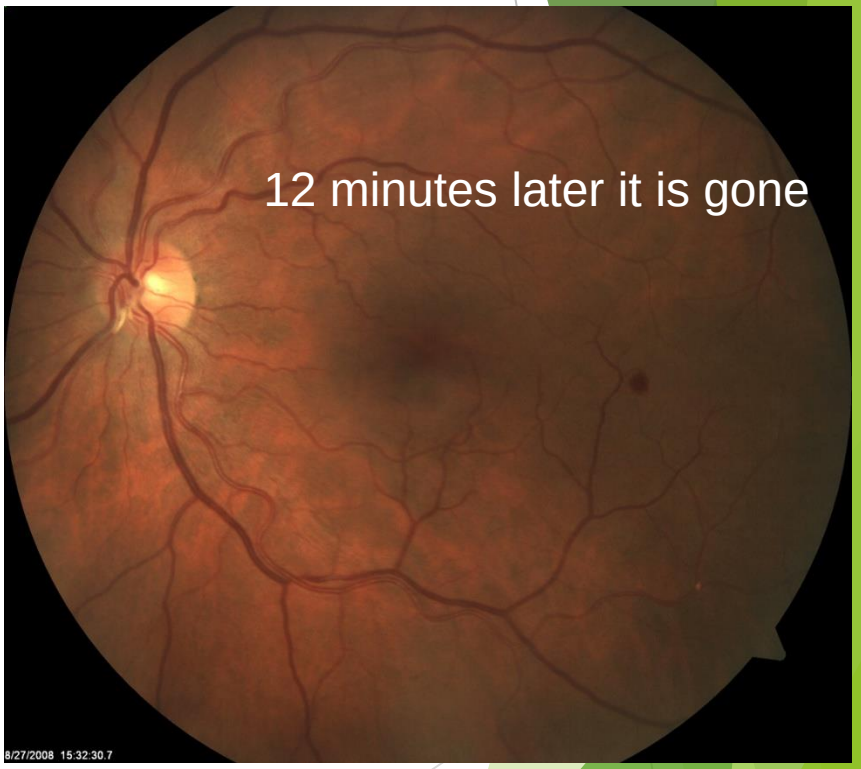


Cilioretinal artery sclerosis

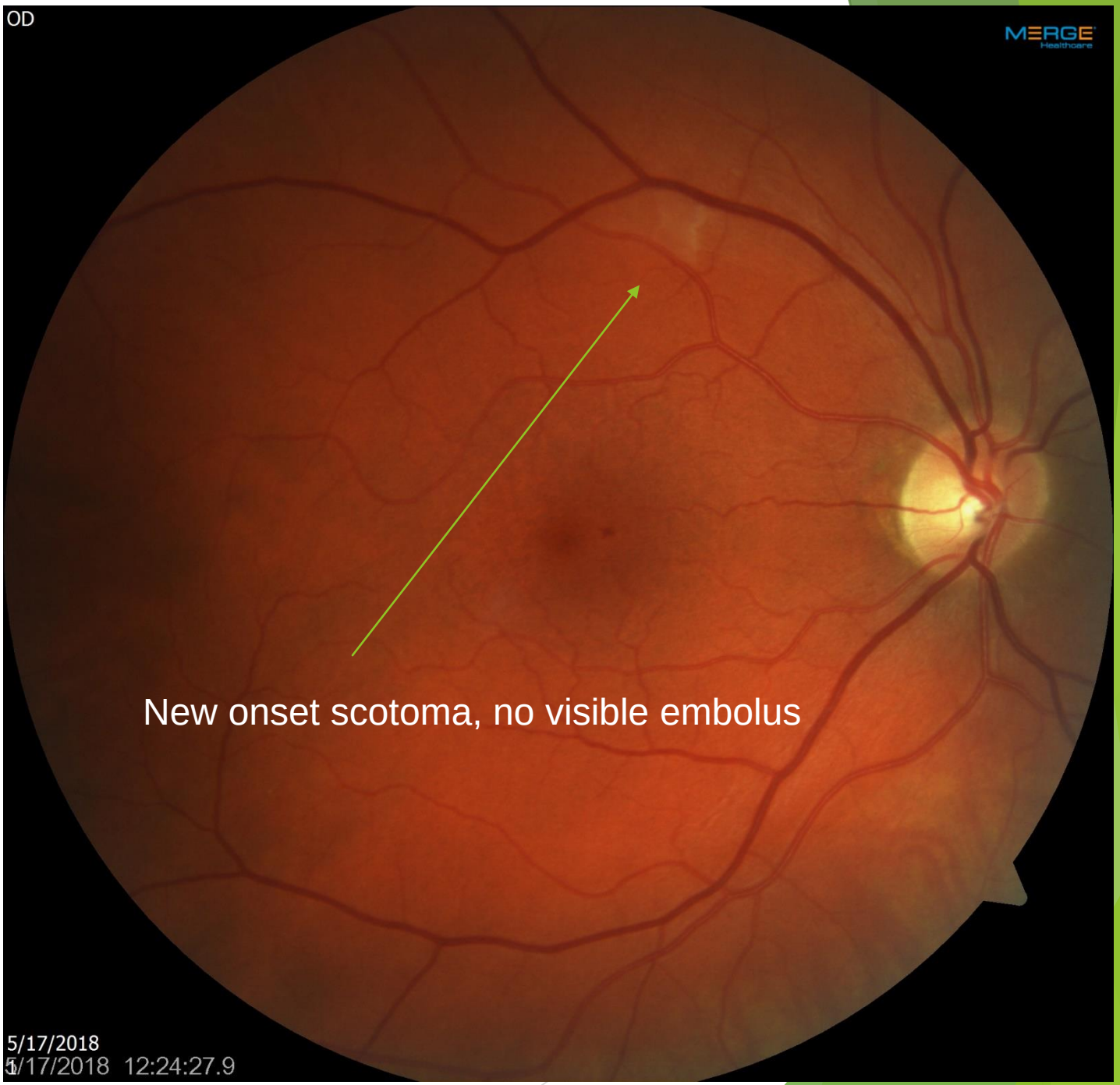


Now you see it…….





BRAO


OD
MERGE
Healthcare
New onset scotoma, no visible embolus
5/17/2018
5/17/2018 12:24:27.9

Retinal Vein Occlusions

- CRVO or BRVO
- Leading cause is hypertension (branch>central) but others include hypercoaguable states and atherosclerotic events. 20% associated with POAG. Link with OSA
- Second only to DBR in frequency
- Occlusion of vein leads to dilated and ruptured capillaries, often with edema

Virchow's triad for thrombosis in general:

- 1) Abnormalities of the vessel wall
- 2) Abnormalities in blood viscosity / coagulation
- 3) Abnormalities in blood flow / velocity

Retinal vein occlusion: systemic associations

- Large study published in September 2019 AJO looked at over 117,000 Korean patients with vein occlusions and found that, compared to matched patients without vein occlusions, they had……
- Increased BMI and waist circumference
- Increased systolic and diastolic BP
- Higher fasting blood glucose
- Higher LDL, triglycerides, and total cholesterol
- Lower HDL

CRVO

- Most patients 50+
- 50+% have systemic association
- Greater risk at higher IOP
- Often increased Homocysteine levels, Antiphospholipid Antibodies
- Presenting symptom is a sudden, painless loss of vision
- Two types: ischemic and non-ischemic.

Non-Ischemic CRVO

- Non-ischemic CRVO (up to 80% of cases) has a less severe presentation and better prognosis
- VA moderately reduced; often 20/60 to 20/100

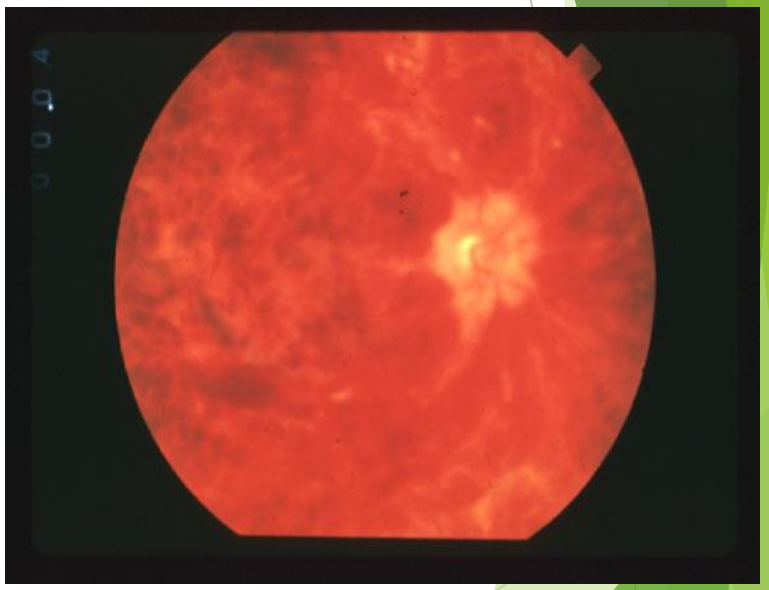
- No APD typically
- 2/3 have 20 / 40 + final VA
- Less capillary non-perfusion on IVFA
- No neovascular complications

Ischemic CRVO

- Ischemic CRVO much more severe
- VA markedly reduced, usually 20/200 or worse

Ischemic CRVO

- + APD
- Massive hemorrhaging in all four quadrants



Ischemic CRVO

- Severe macular edema, may also get disc edema
- More CWS than non-ischemic
- IVFA / OCTA shows significant capillary dropout and non-perfusion
- NVI very common, NVD and NVE reportedly less so??? Only 40-45% with NVI get NVG
- *How could NVD and NVE be less common than NVI? They are harder to see with all the blood in the fundus

Management

- Consider carotid Doppler with ischemic CRVO. Carotid occlusion highly associated with ischemic CRVO but not causative
- Blood pressure, cardiovascular work-up

Treatment

- Anti-VEGF injections for macular edema are the standard treatment (lucentis, avastin, eylea, etc.). Early loading doses then often treat and extend
- Intravitreal steroid implants / injections can be added in recalcitrant cases (would often switch anti-VEGF agents first)
- PRP still has a role if neovascularization develops, can combine with anti-VEGF

In general…..compared to anti-VEGF treatments

- PRP advantage with neo……..
- Can permanently “fix” the problem by decreasing oxygen demand below the needed threshold

- PRP disadvantage….
- ERM formation
- Night vision loss
- Peripheral vision loss
- Longer treatment session

In general……

- Anti-VEGF injection advantages…..
- Very low rate of side effects (endophthalmiits, elevated IOP with repeat injectiions)
- Work well, often very well

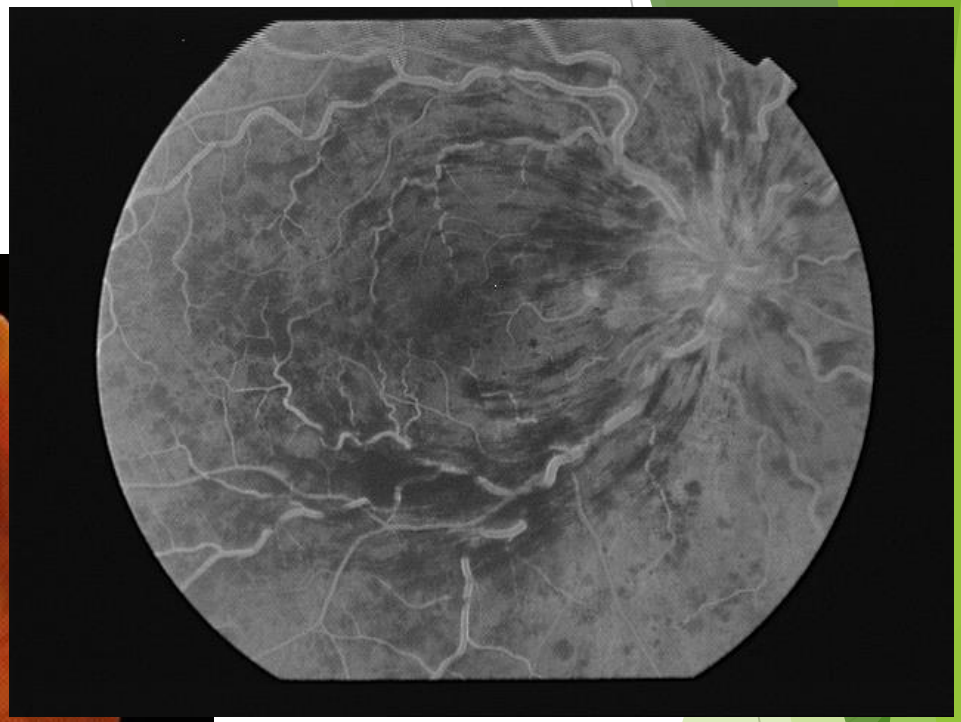
- Anti-VEGF injection disadvantages….
- Repeated injection after injection so issues with cost, transportation, “visit fatigue”, etc. Can need dozens of shots, less permanent

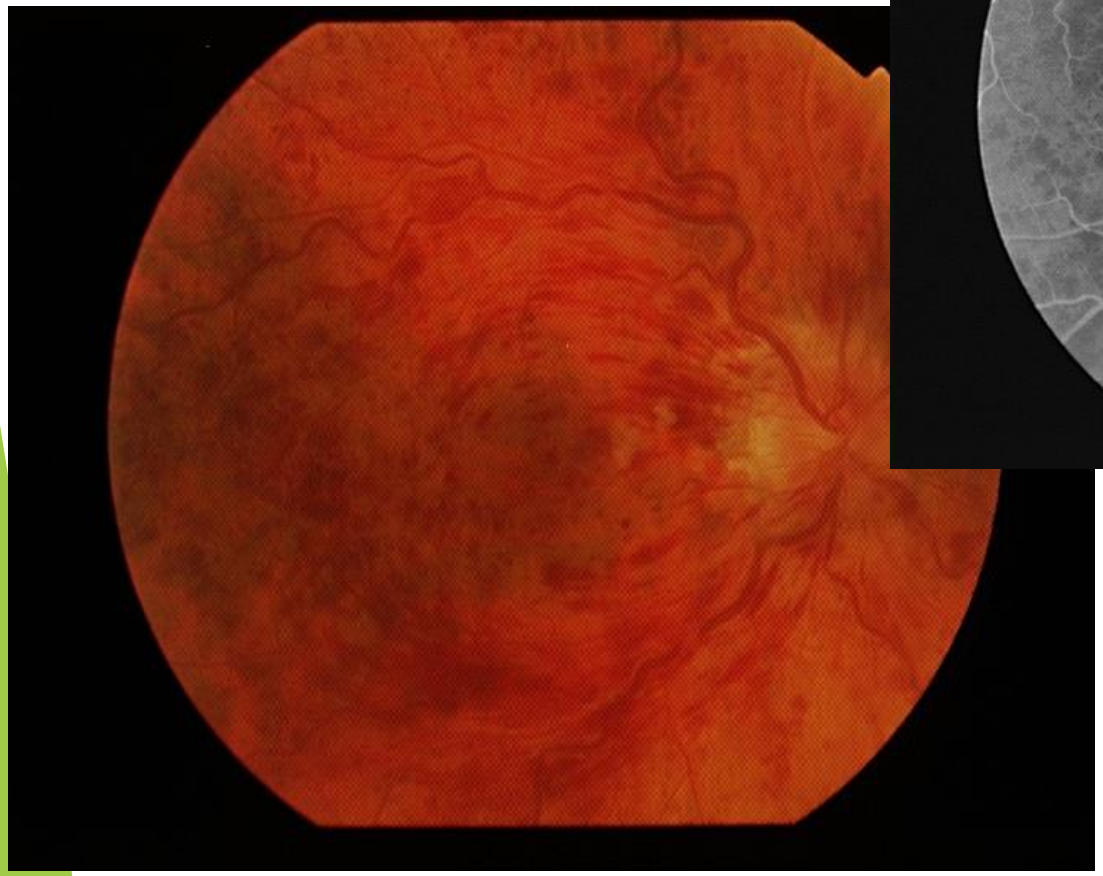
Treatment can be lengthy: RETAIN study for example

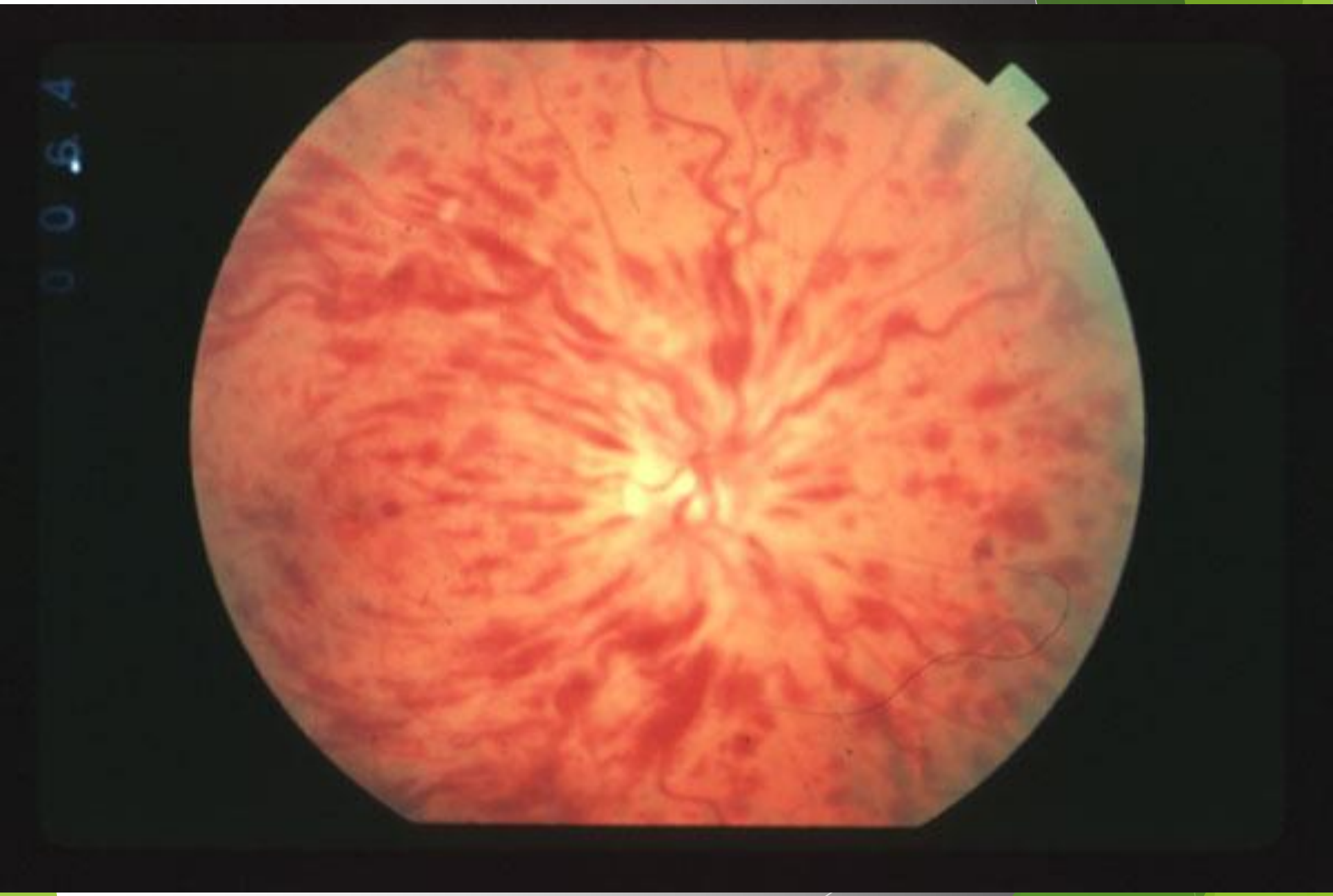
- 34 BRVO and 32 CRVO patients receiving Lucentis shots followed over four years.
- At the four year mark…….
- Average vision gain achieved at six months was still present at four years
- But…….. 50% of BRVO and 56% of CRVO patients were still getting shots four years out

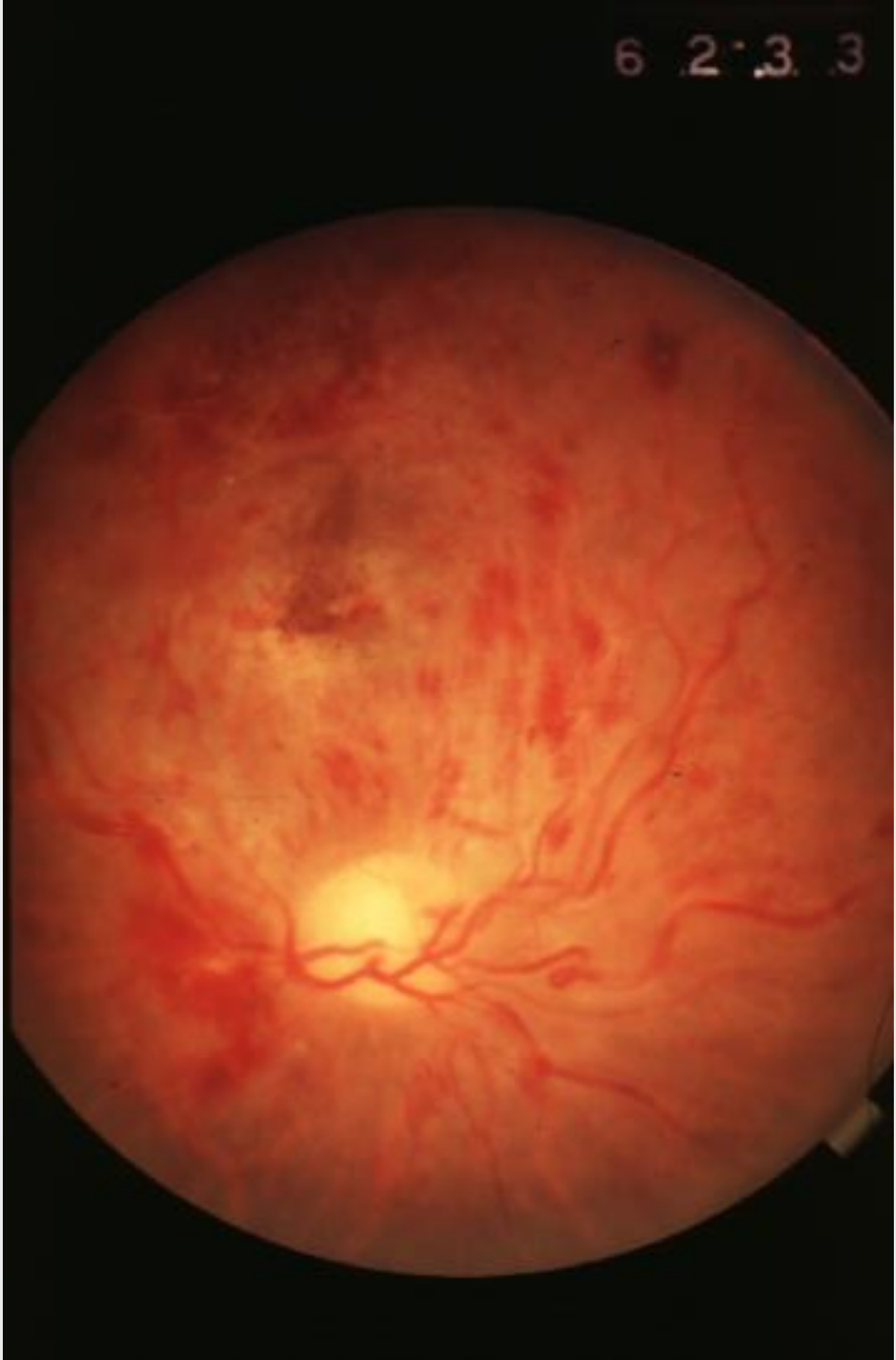
CRVO

- Non-ischemic





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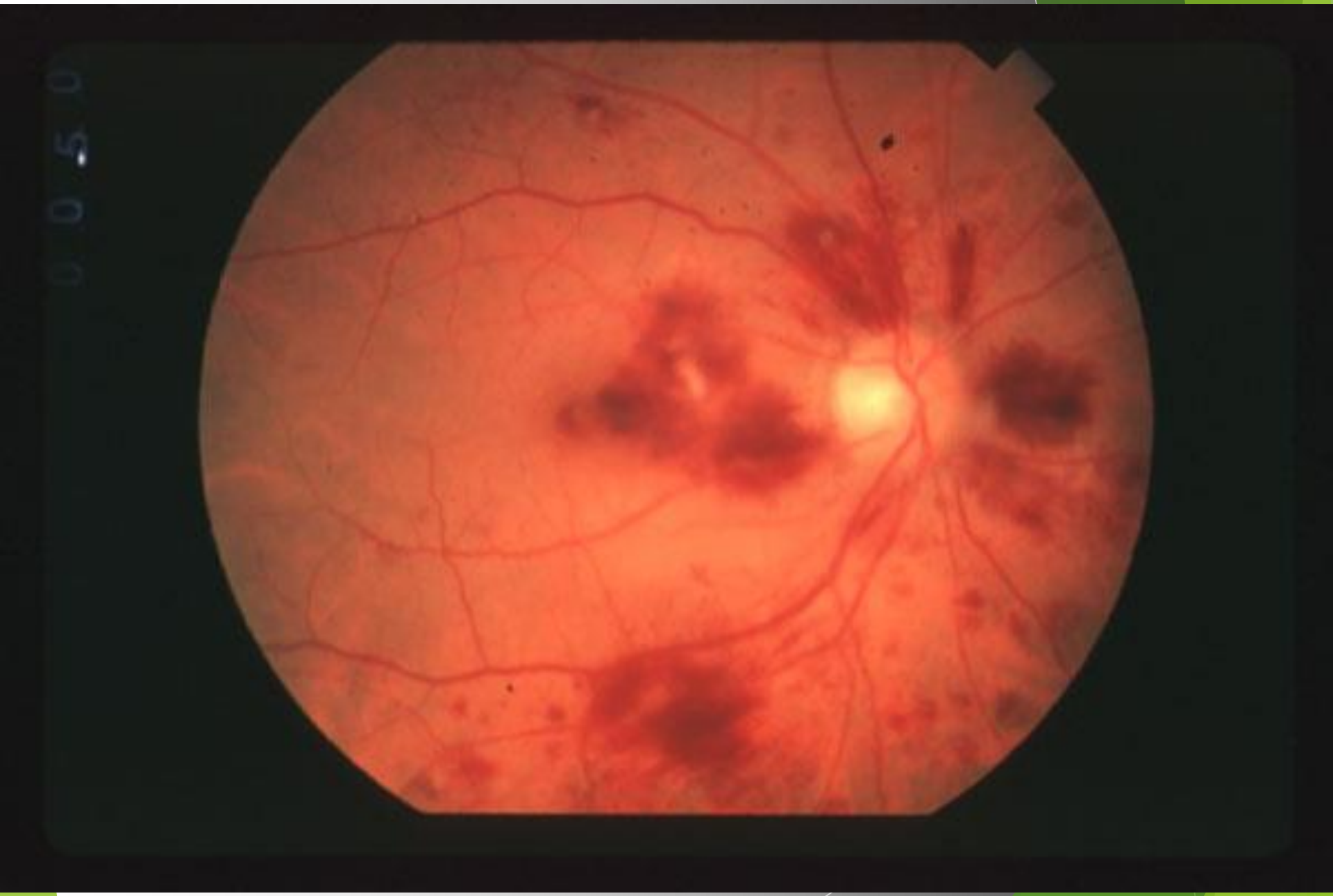
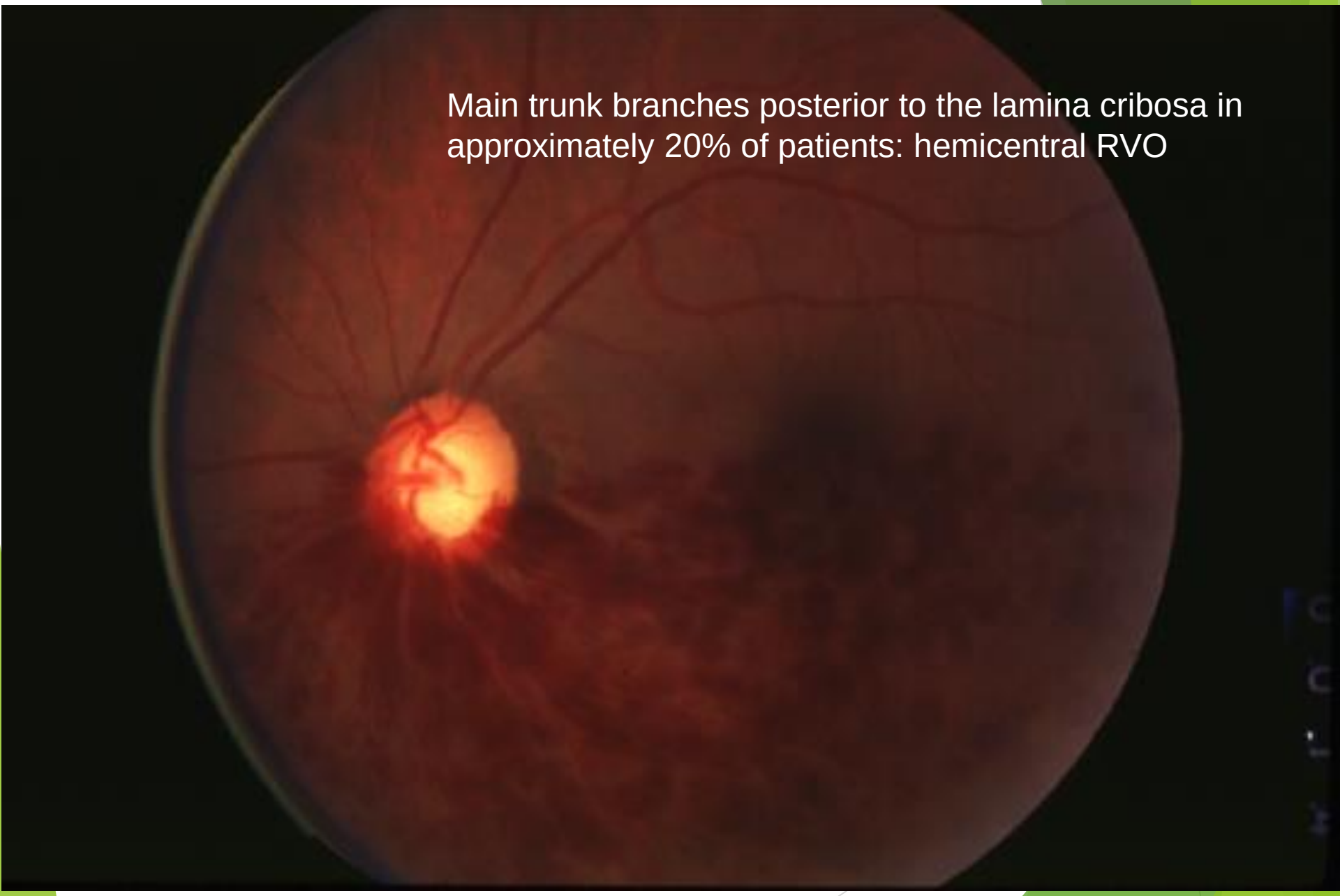
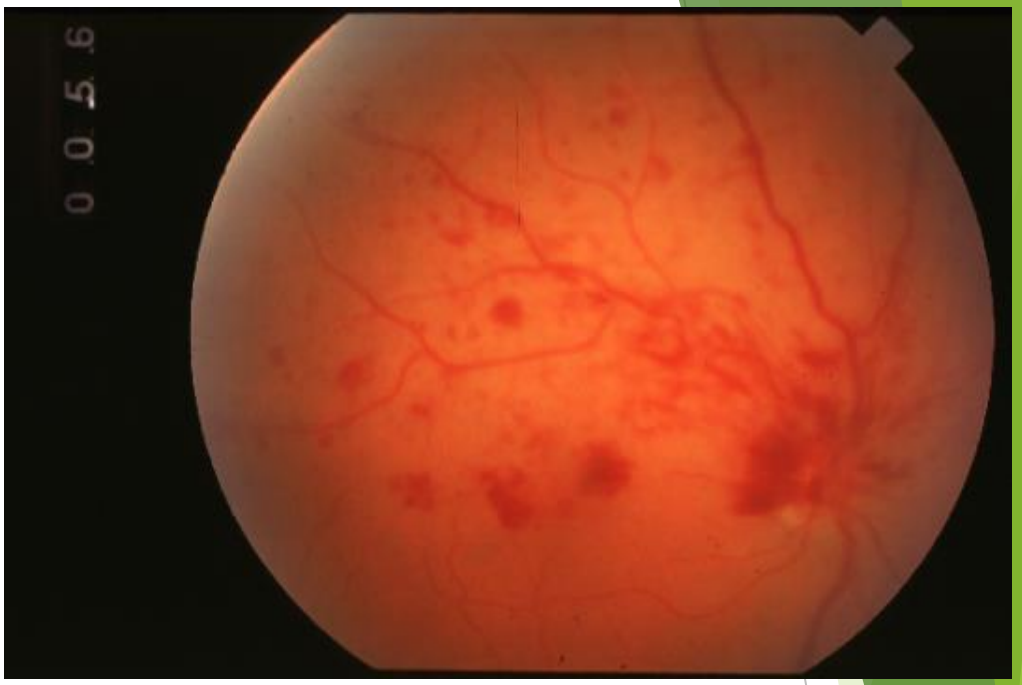
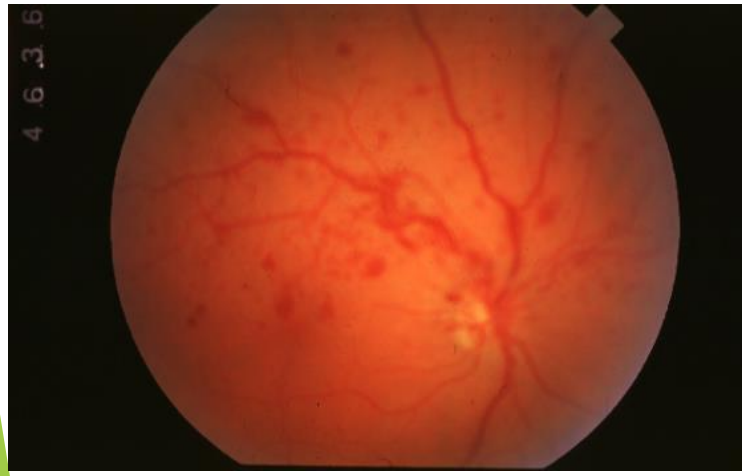
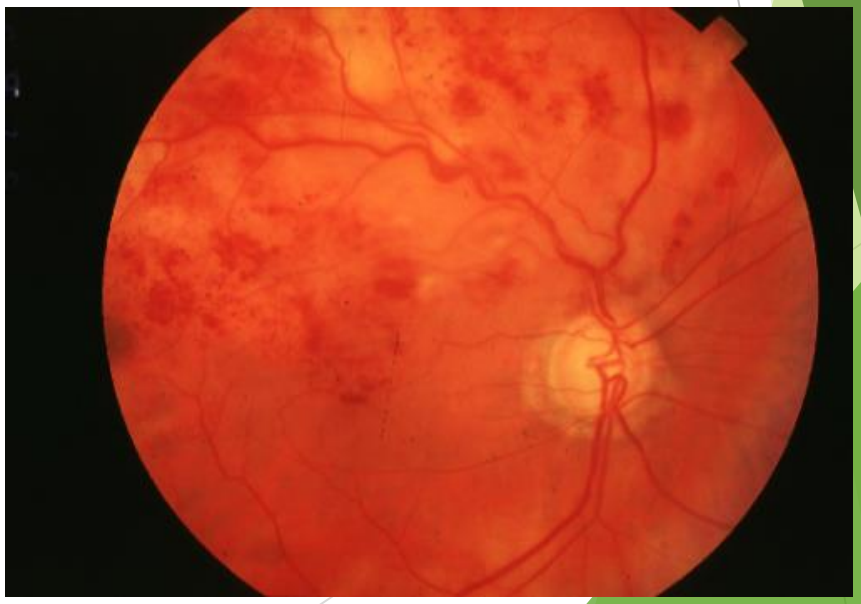
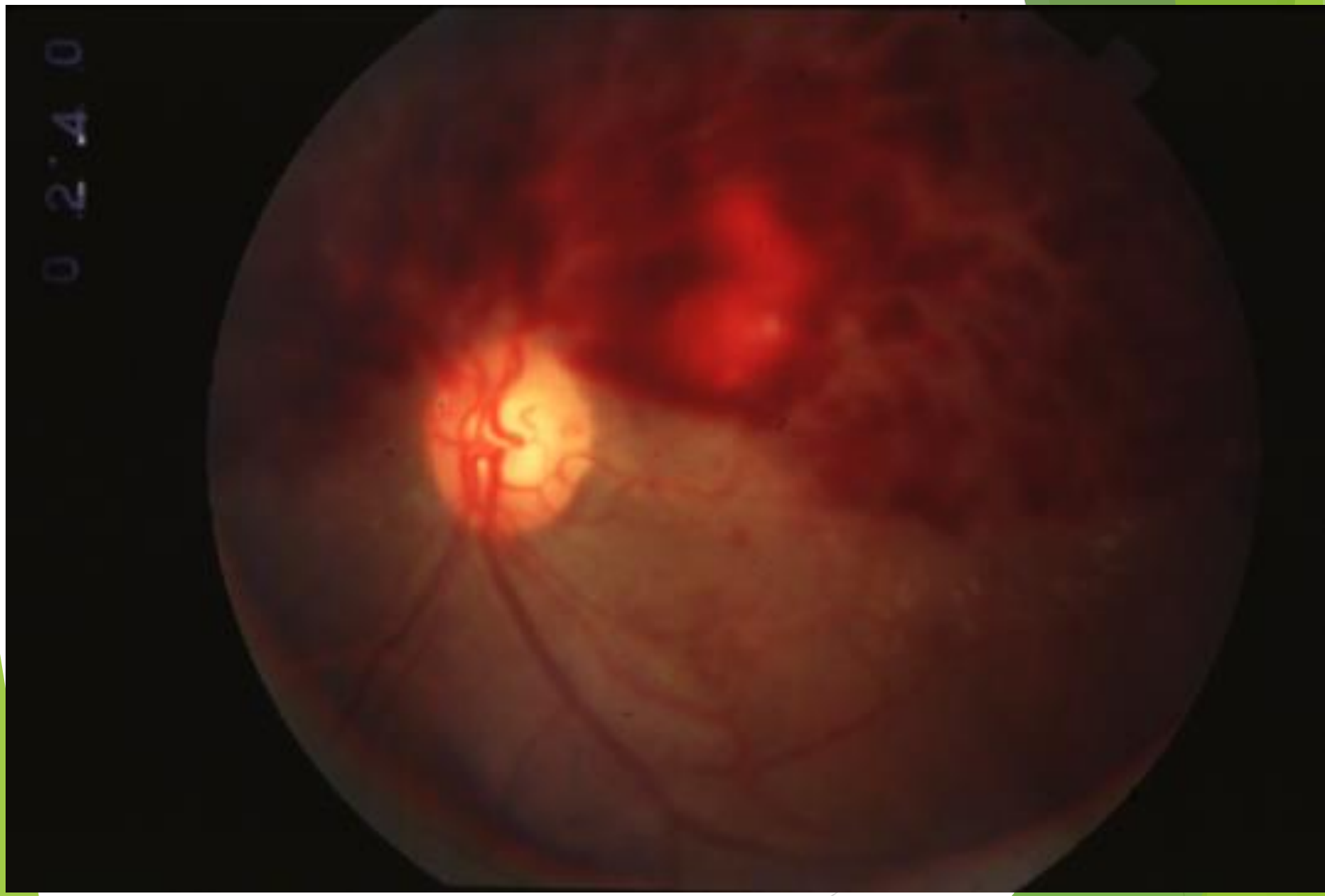



Main trunk branches posterior to the lamina cribosa in approximately 20% of patients: hemicentral RVO





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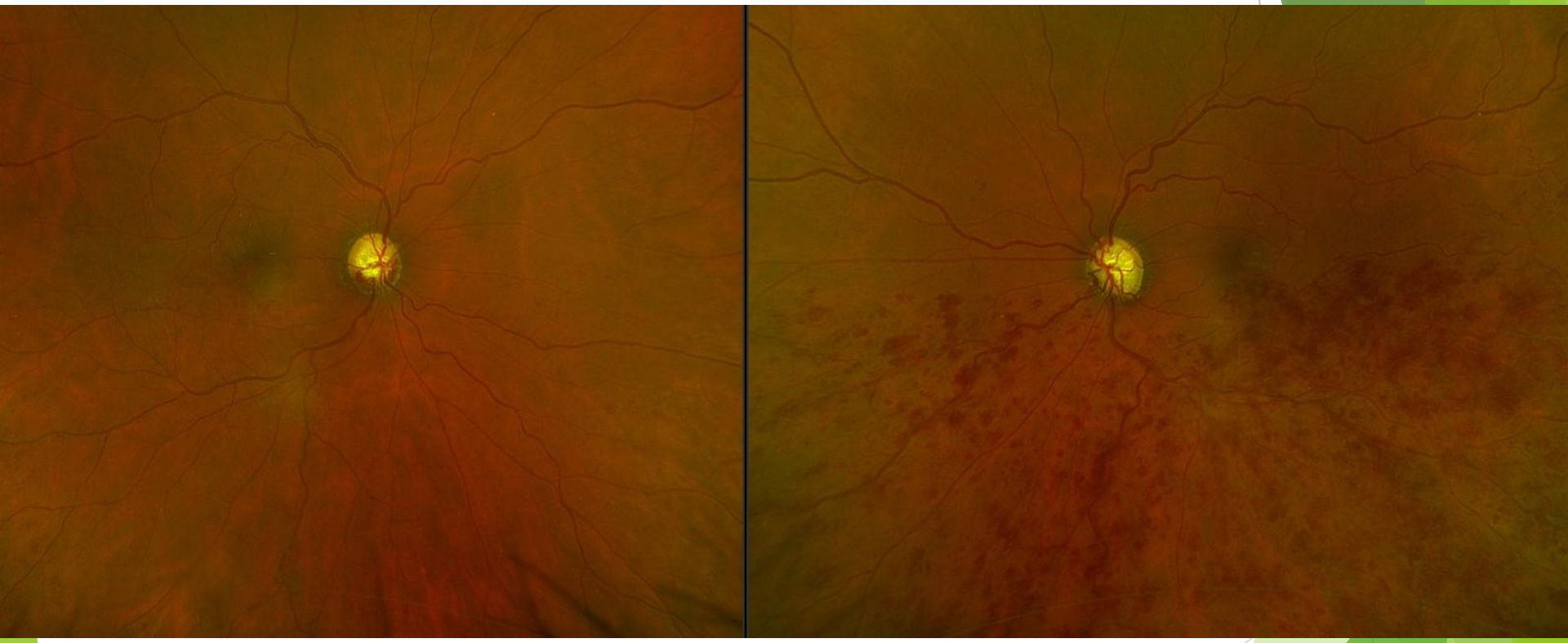
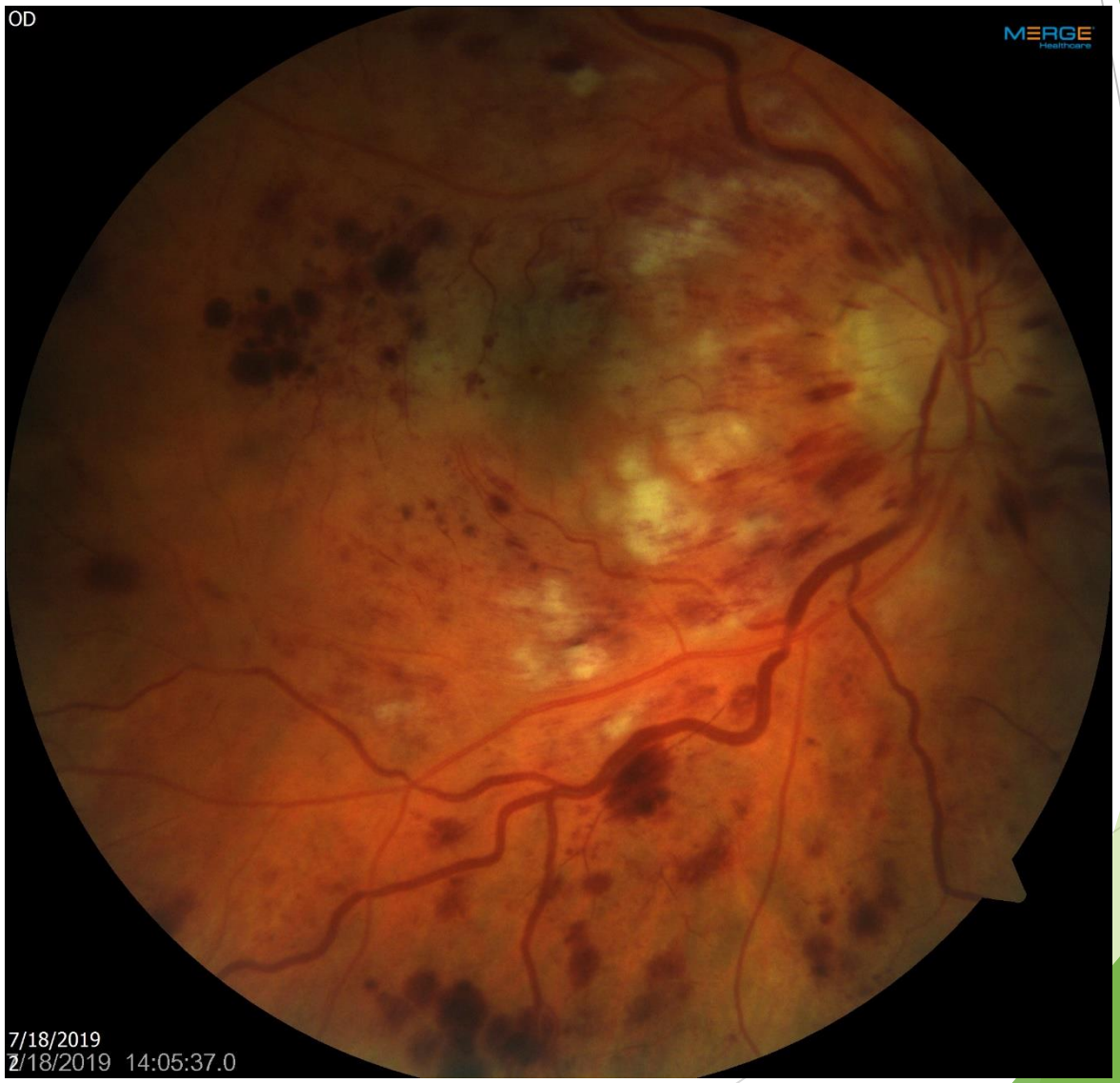
Past hemicentral RVO OD with collaterals, current hemicentral RVO OS



Combined CRAO /CRVO



BRVO

- Rarely ischemic, commonly non-ischemic.
- VA compromised if edema or blood reaches the macula or if there is long term macular ischemia
- Neo more rare, NVD/NVE >NVI
- 98% temporal. *Why? Because that is where most crossing changes are
- Prevalence in US of less than 1%

BRVO

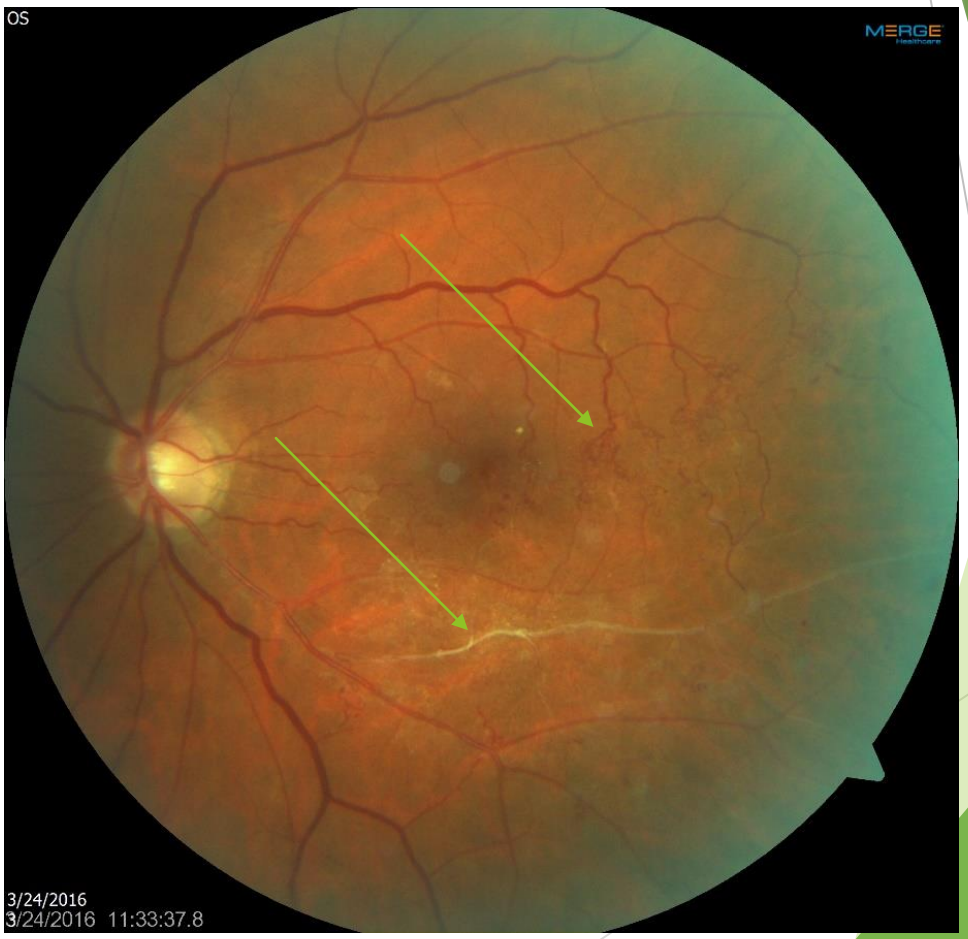
- Collateral vs. neovascularization
- Collaterals form over 6-24 months then often regress and close, except for the largest ones. Can also form after CRVO

BRVO treatment

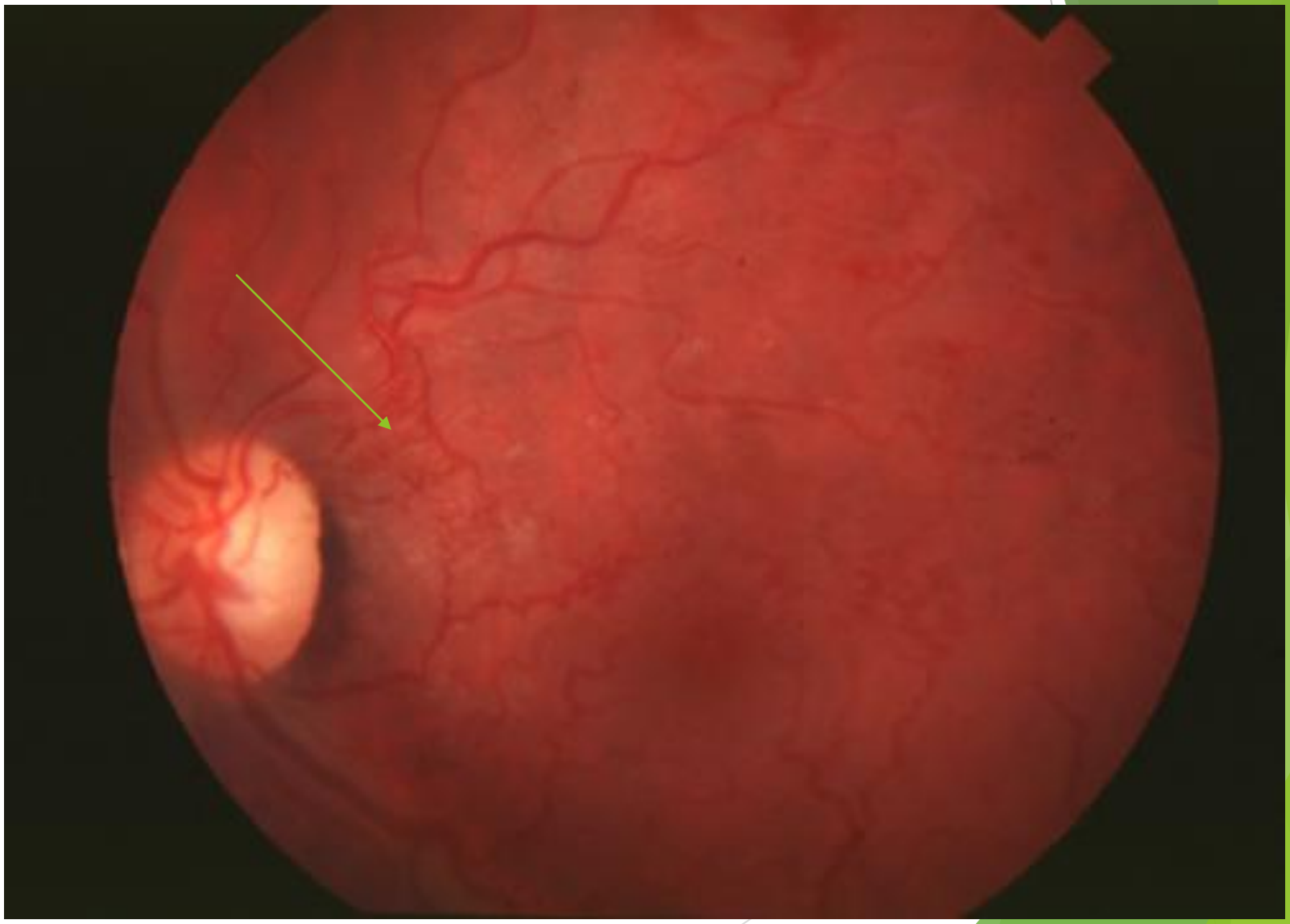
- Historically performed grid/ focal laser therapy if VA remained worse than 20/40 after 3 months. Now:
- Standard for BRVO induced macular edema is anti-VEGF injections with early loading doses then treat and extend. If no macular edema, monitor without treatment (unless neo develops, which is uncommon)
- Grid / focal laser therapy can be considered in special circumstances (can not travel for repeated injections, can not afford them, can not tolerate them, etc.)

- Evaluate blood pressure, cardiovascular work-up

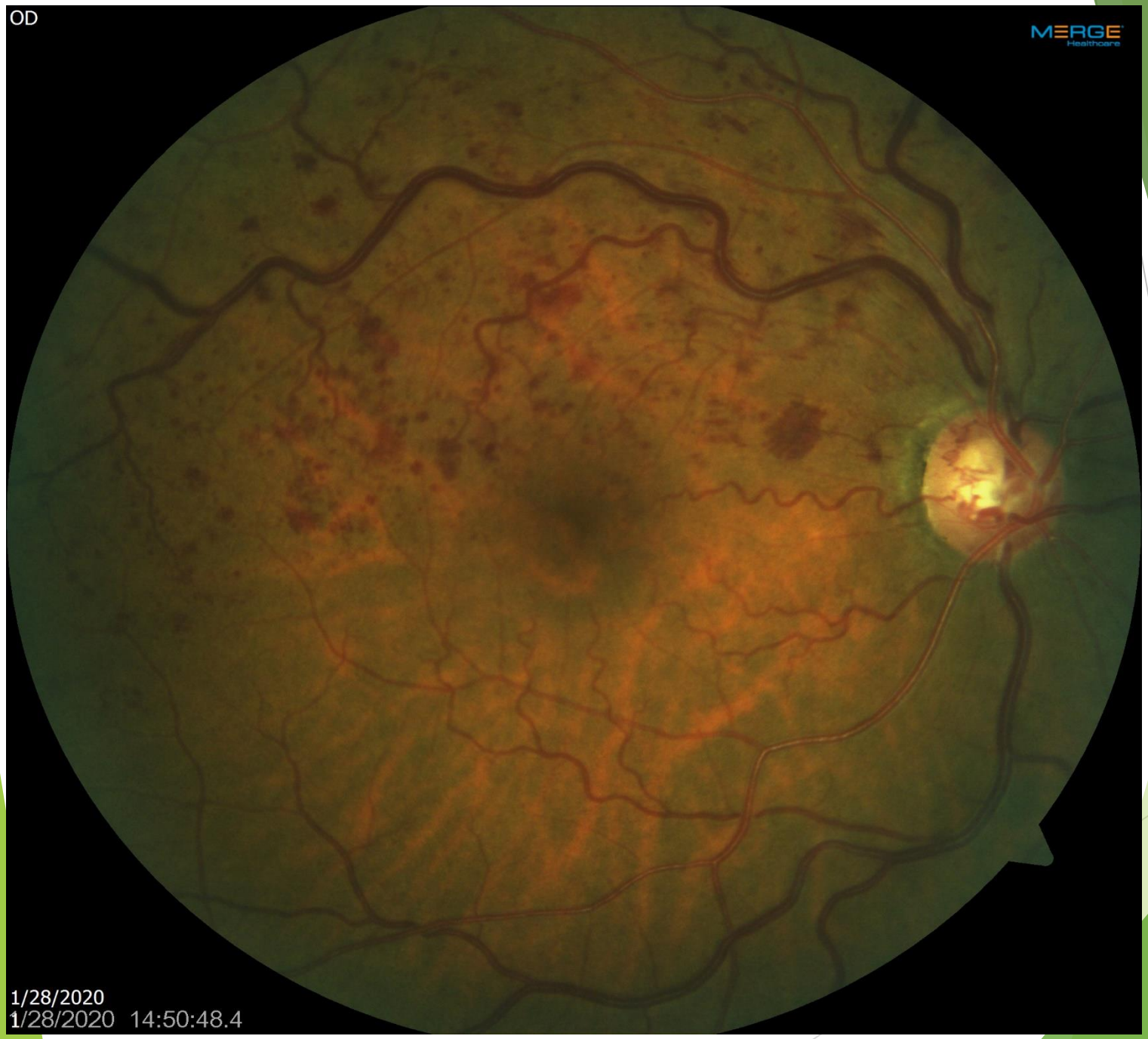
Collaterals & sclerosed vein post occlusion



Collaterals

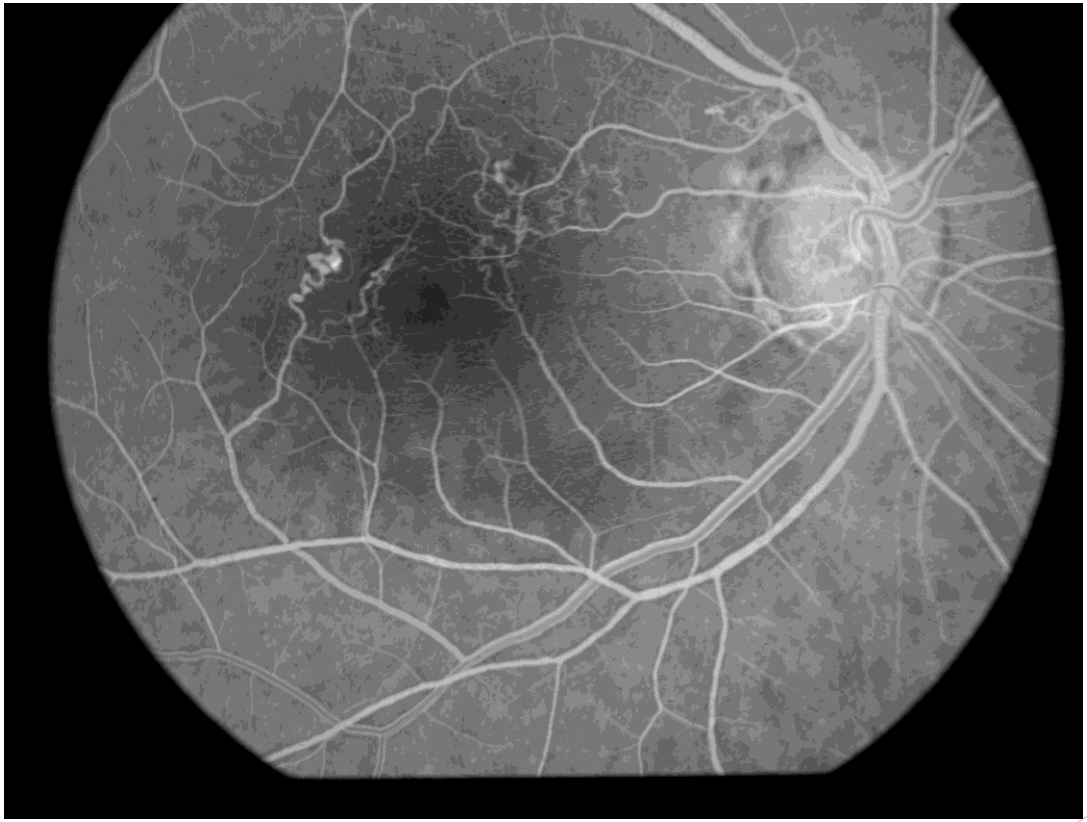


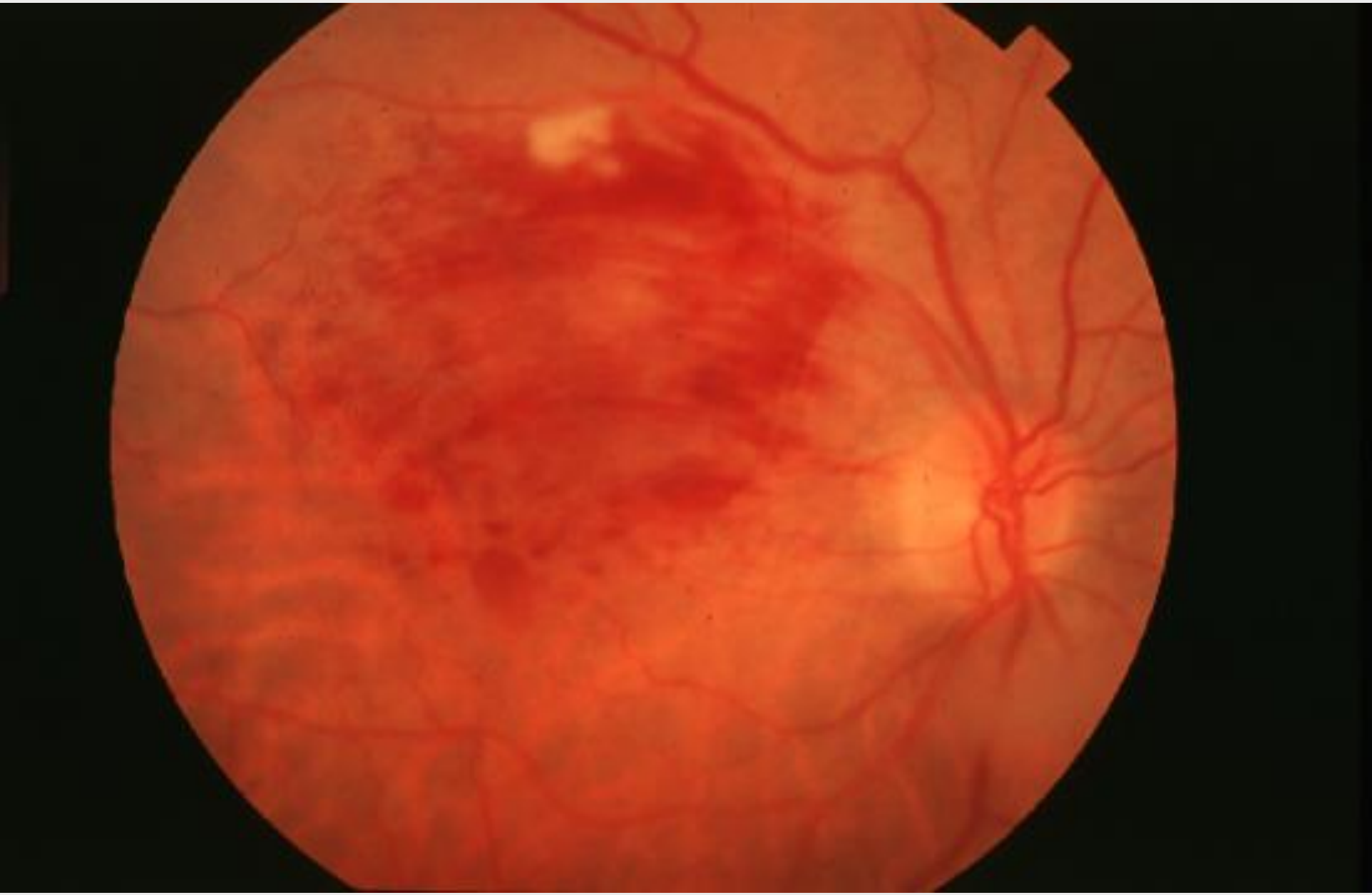
Collaterals: optic nerve

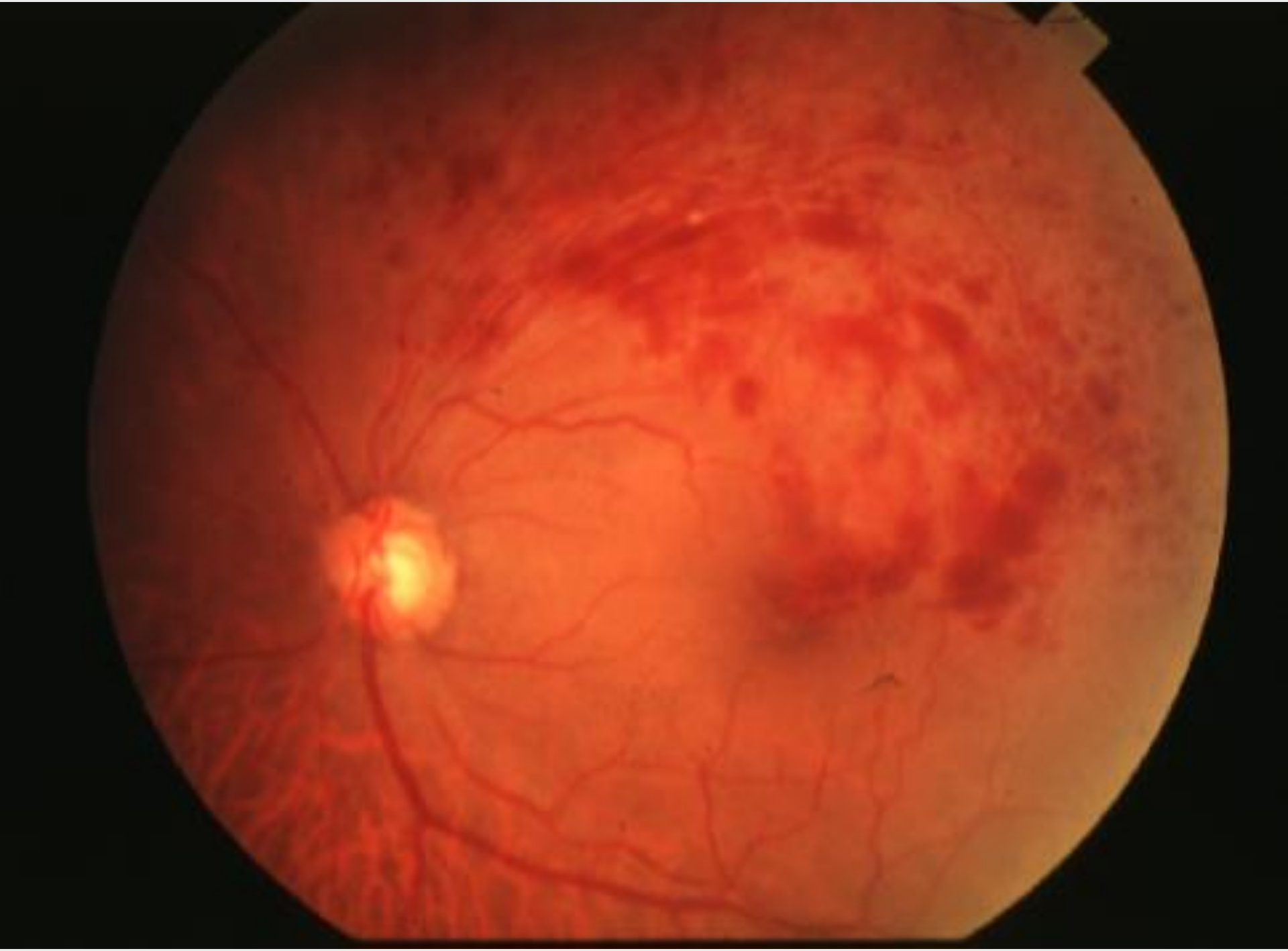


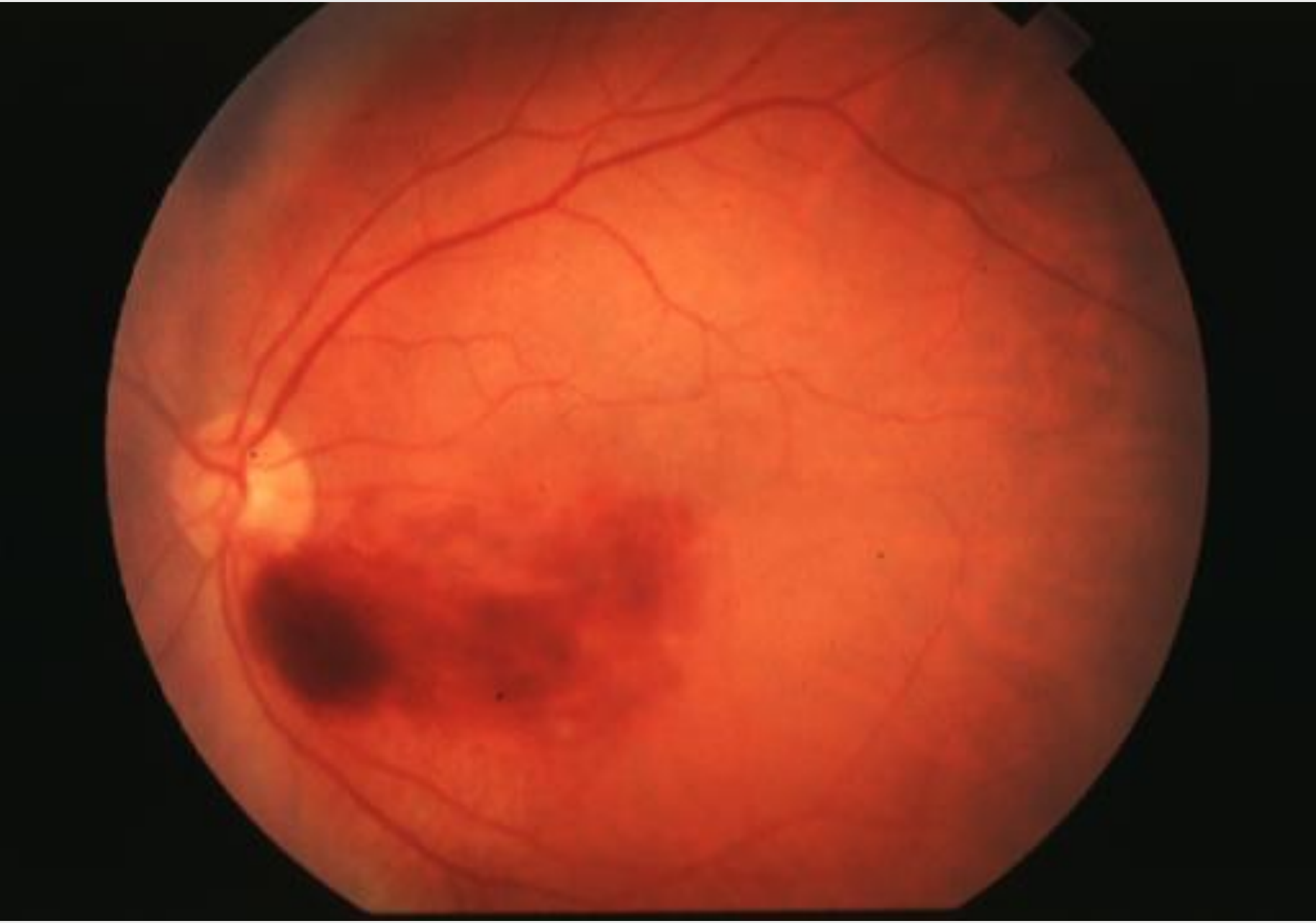
collaterals

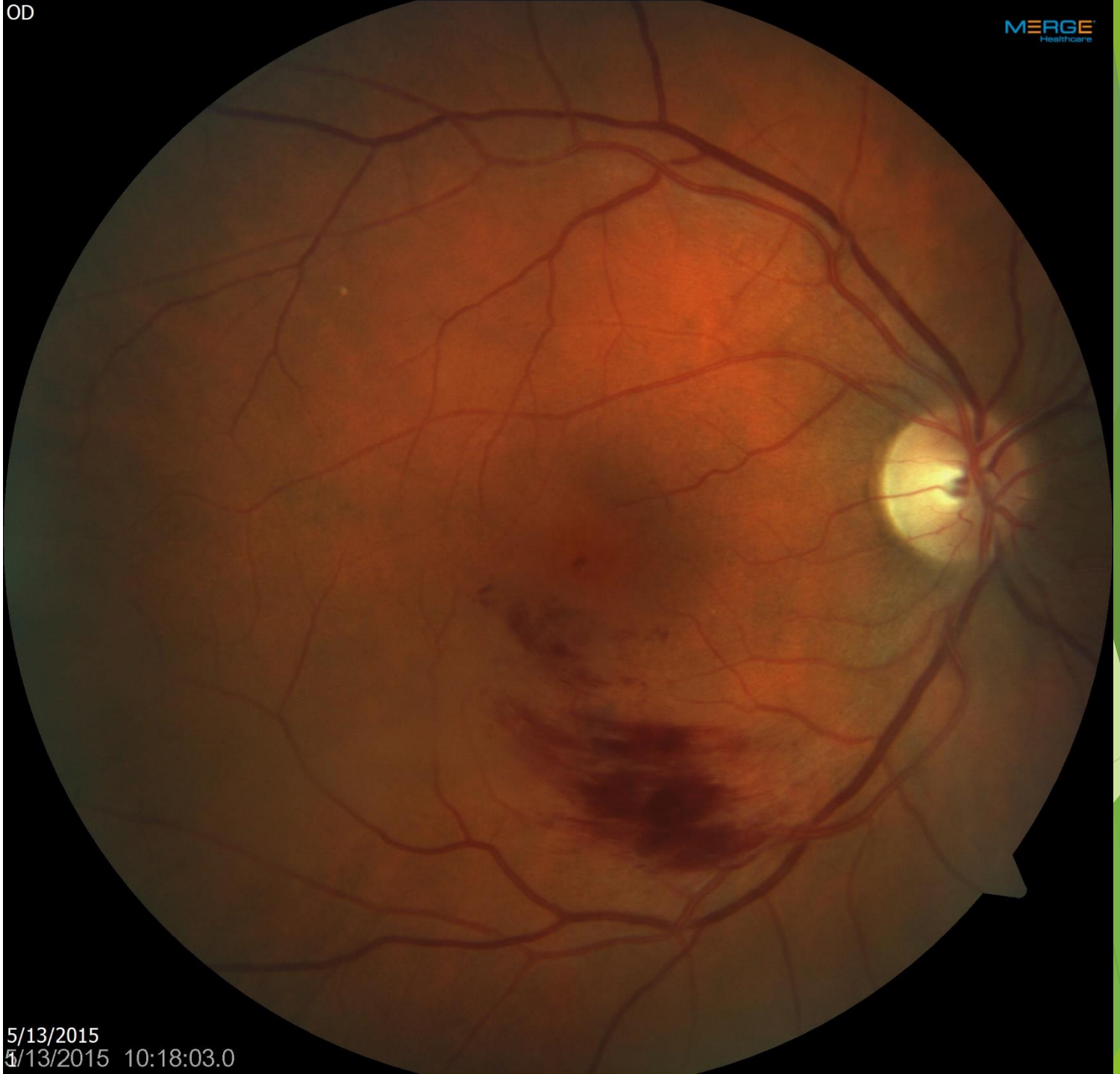
Collaterals IVFA courtesy Dr. Mohammad Rafietarry







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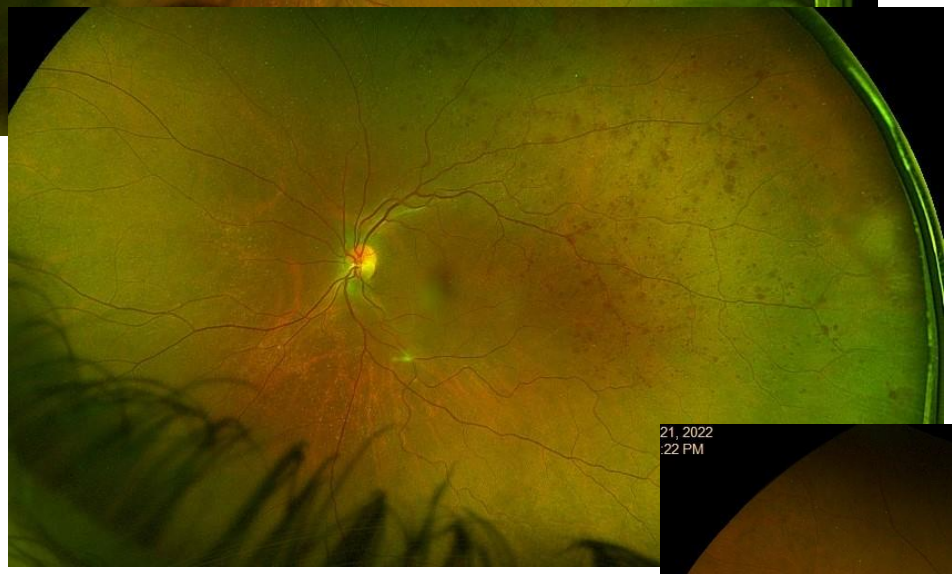
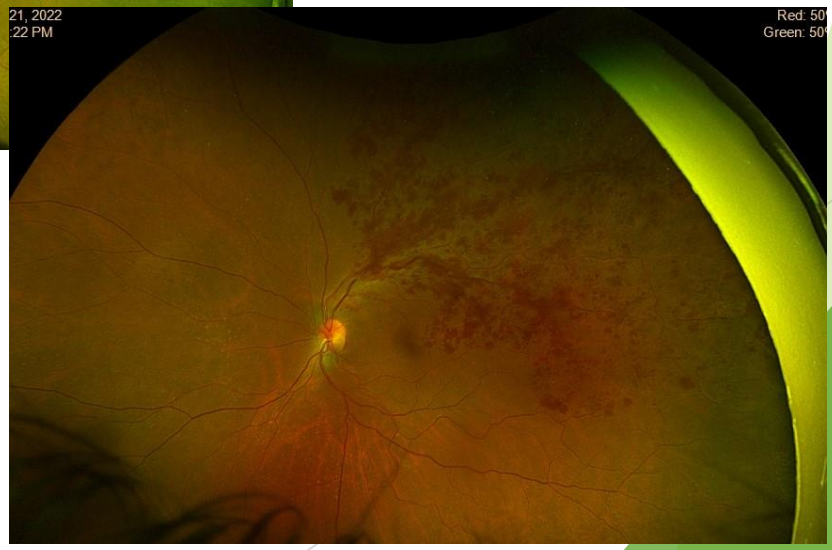
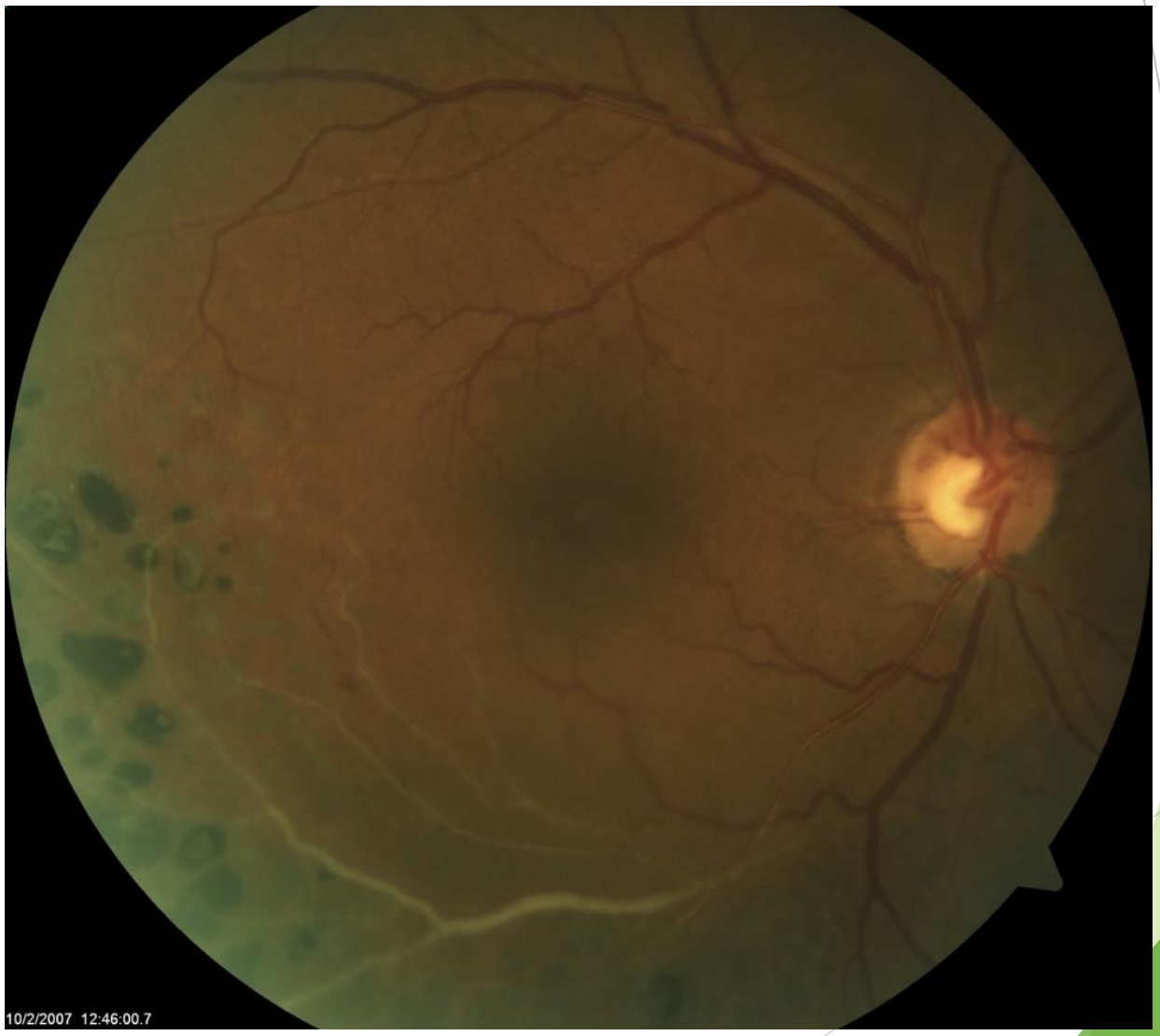


April



June

BRVO S/P laser Tx



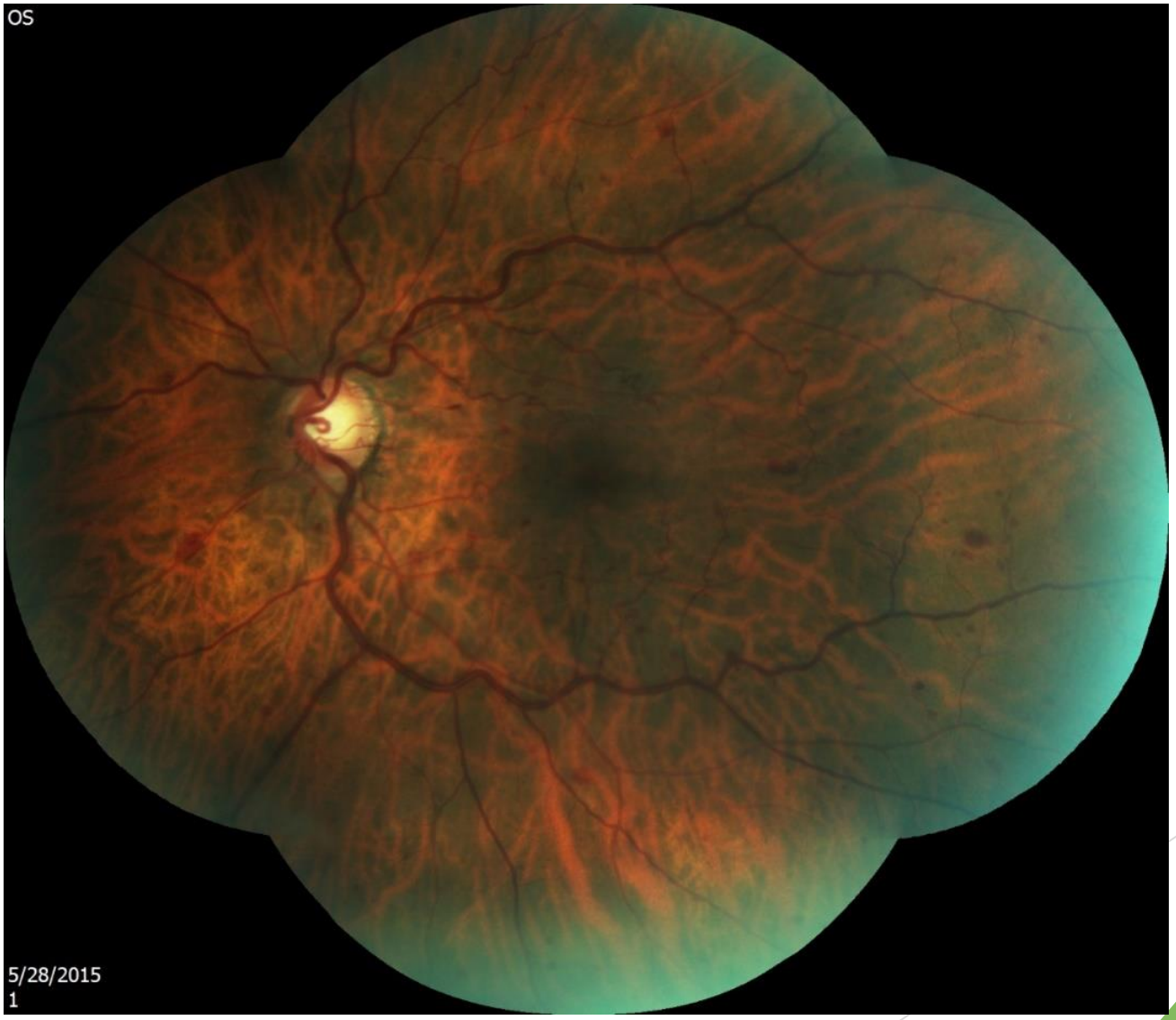
Papillophlebitis

- May be an inflammatory variant of CRVO (f 2x m). Often strikes at a younger age, but not always
- Some debate if it is even a separate condition or if it is just a very mild CRVO
- Disc edema typically out of proportion with retinal hemorrhaging, 4 quadrant hemorrhaging out to periphery
- Typically mild VA reduction to around the 20/30 level but can be worse

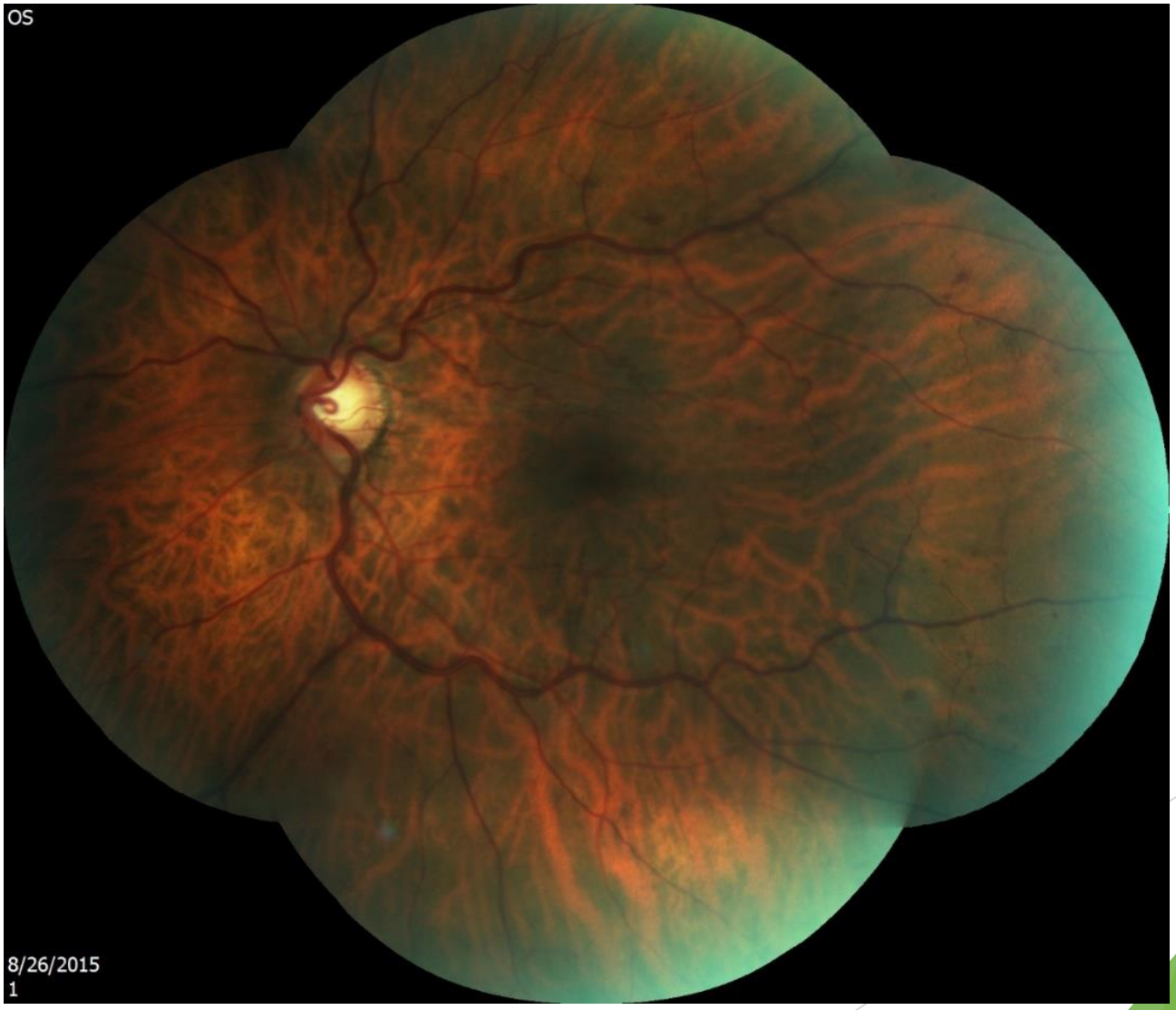
Papillophlebitis

- Often a vague prodrome of scintillating, colored lights with visual disturbances
- Enlarged blind spot on the visual field
- Dilated and tortuous veins
- Condition is self limiting over the course of several months and a complete recovery is the norm
- May be related to APA syndrome, other less common systemic conditions, or may have no systemic association

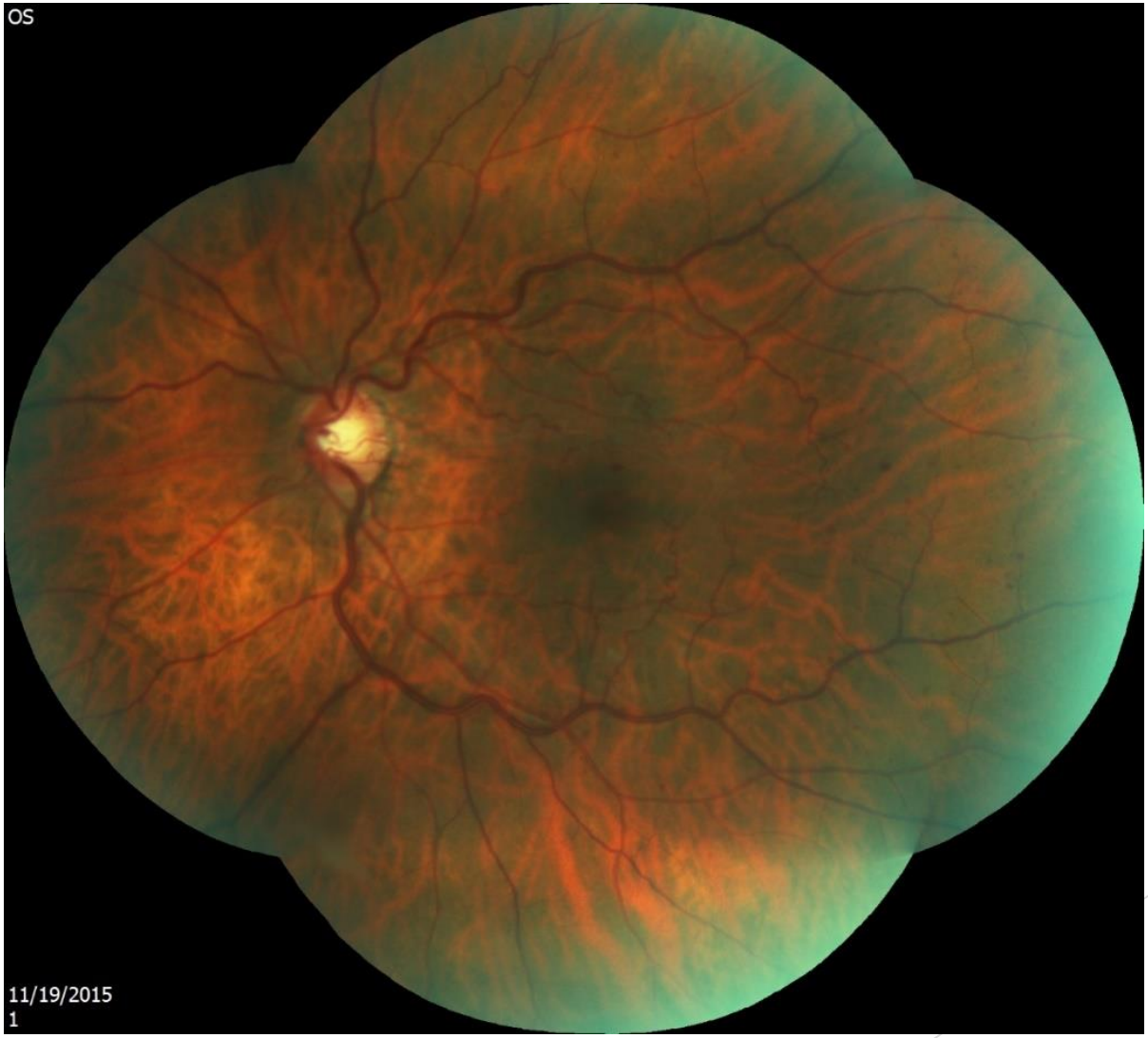
Papillophlebitis



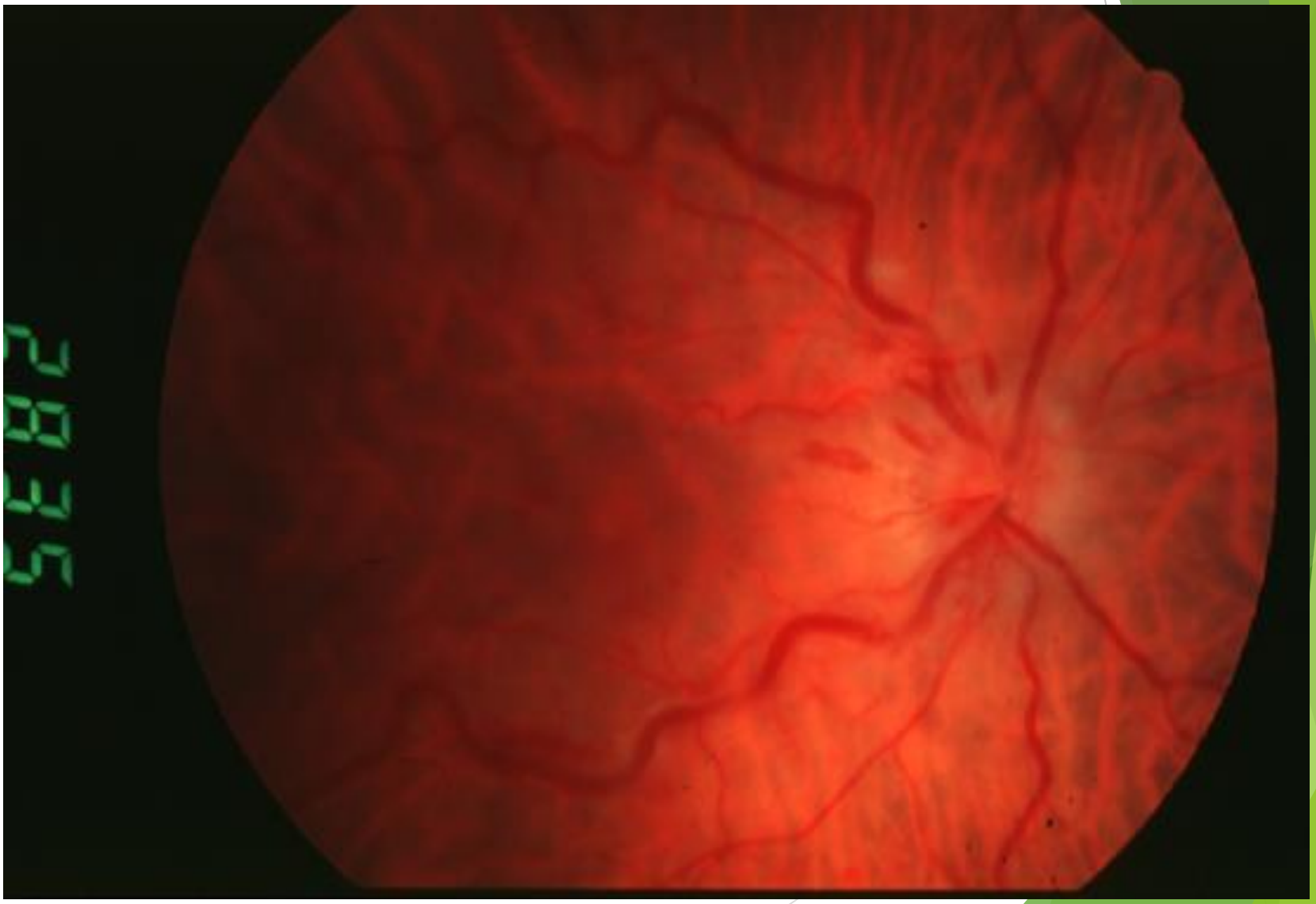
Papillophlebitis FU



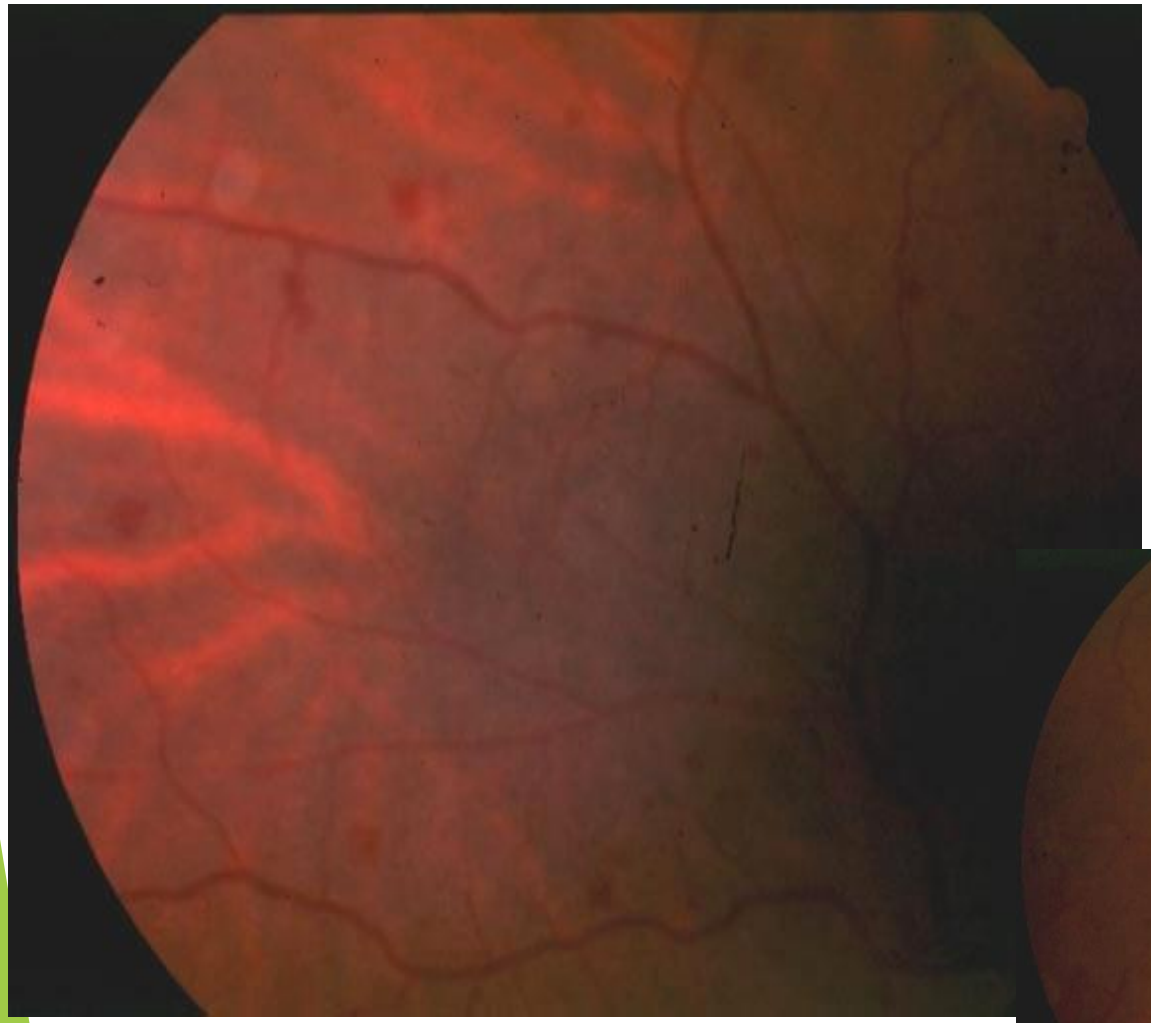
Papillophlebitis FU

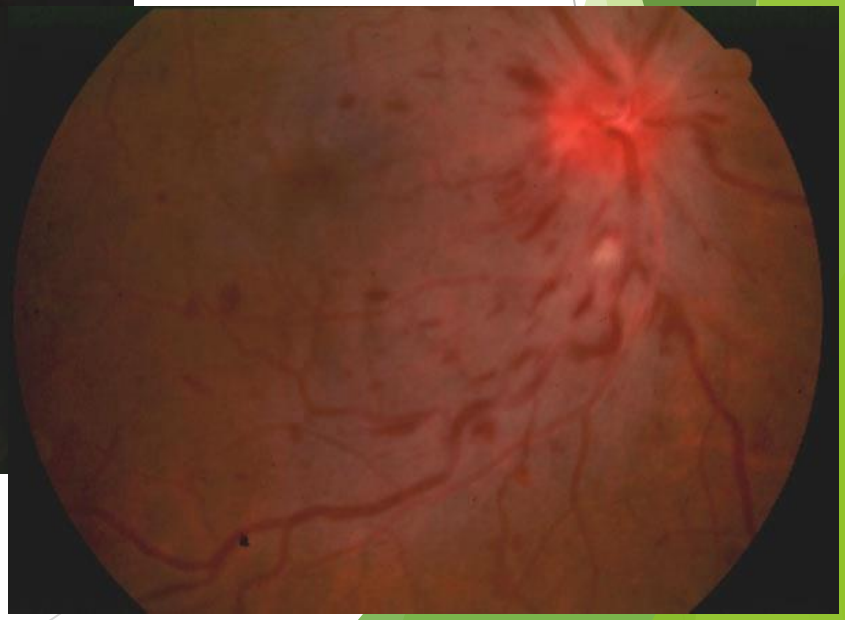


Papillophlebitis



Papillophlebitis





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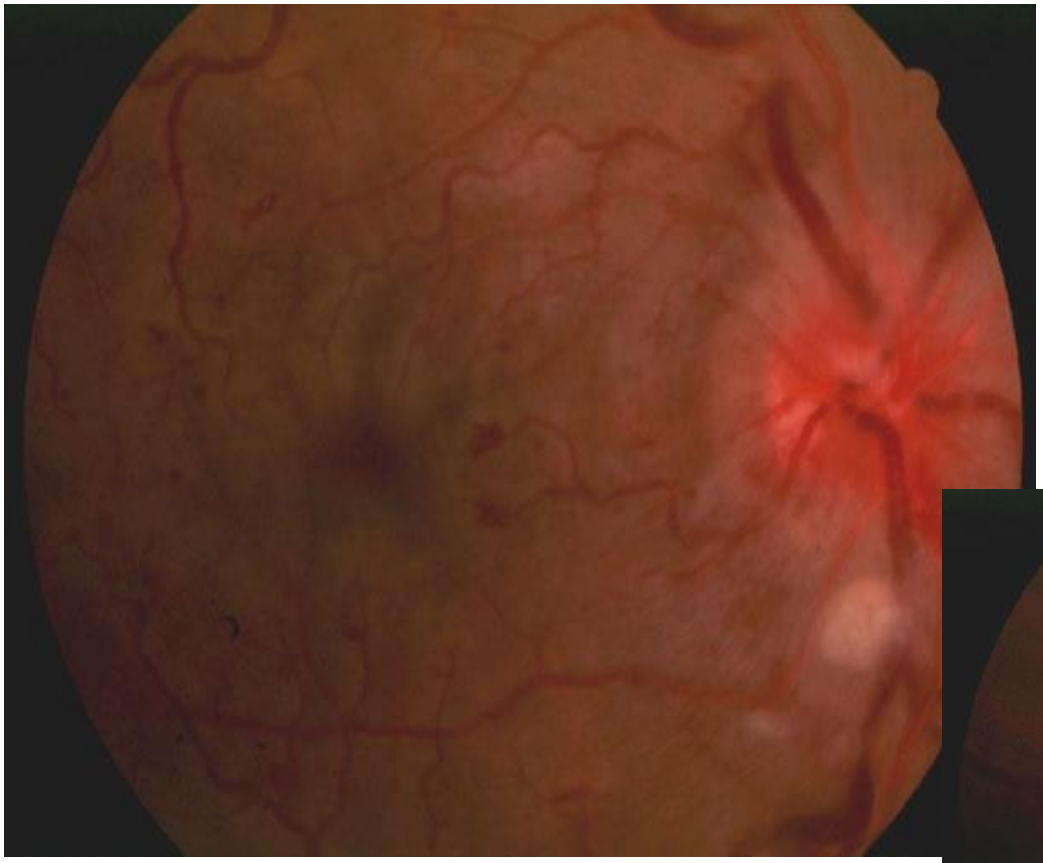

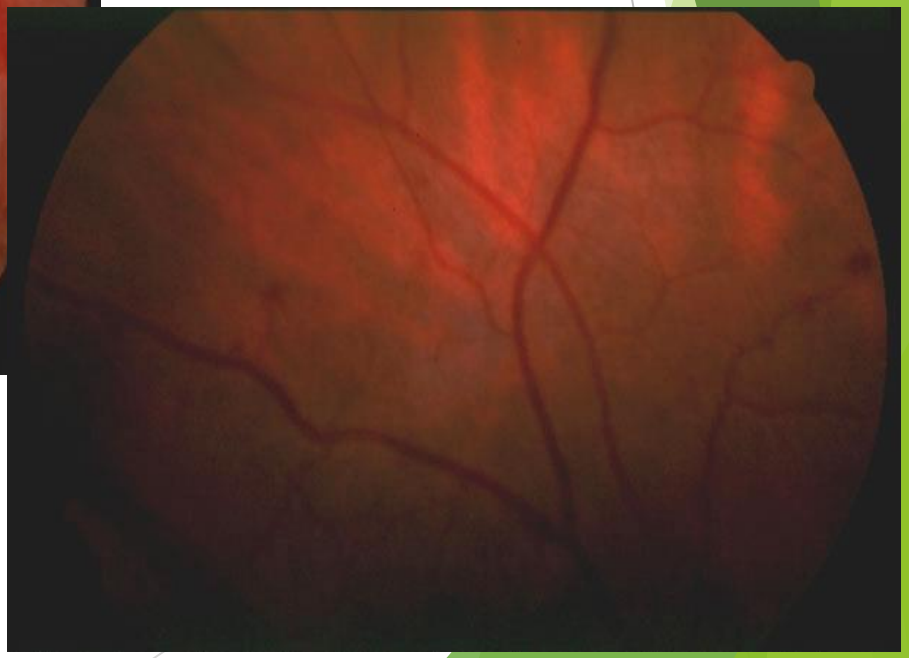

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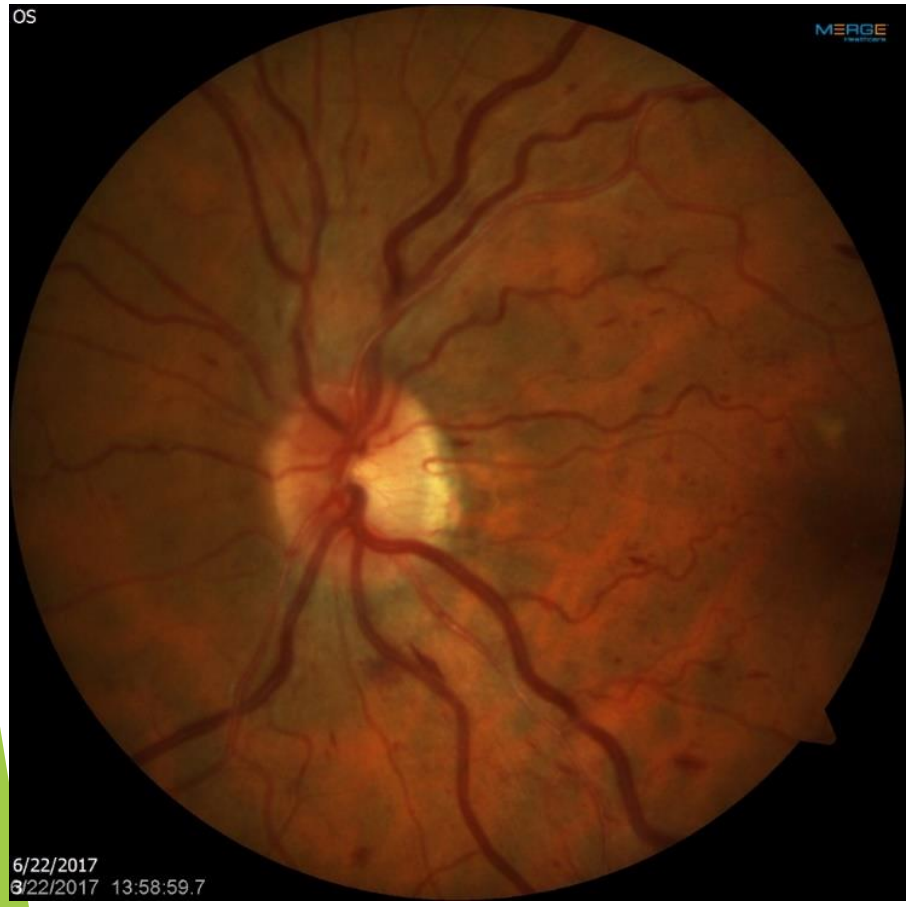

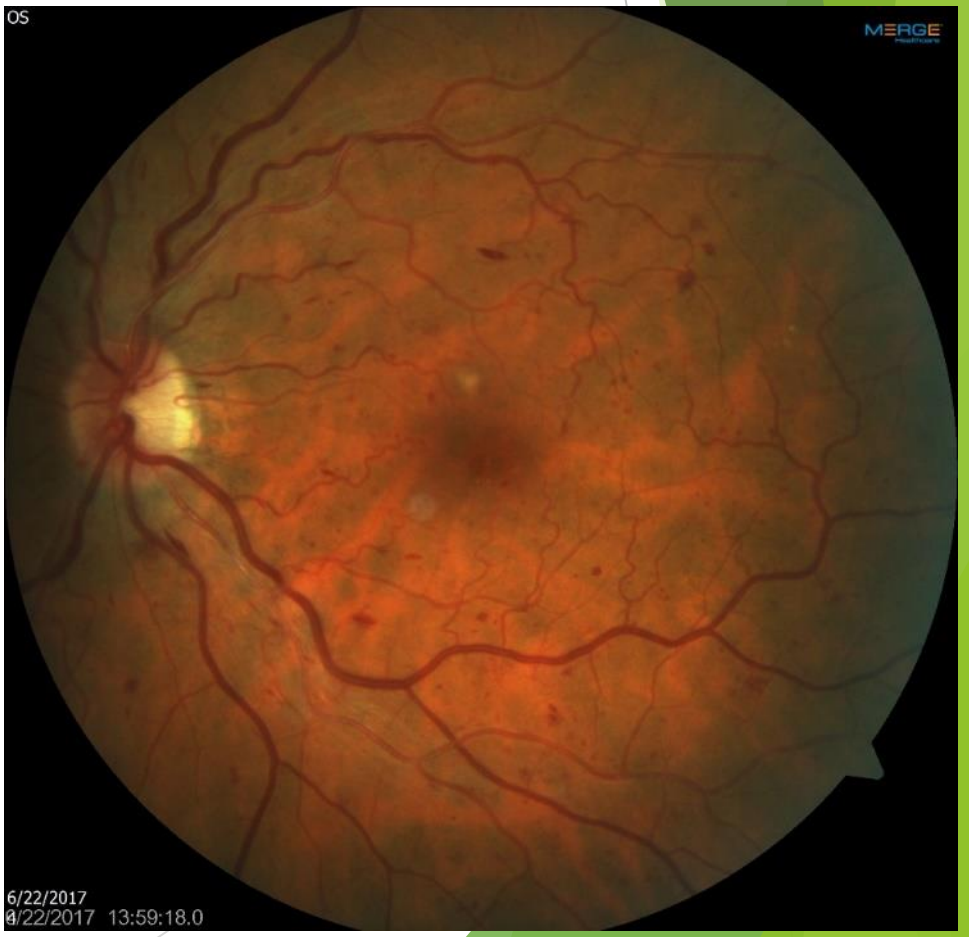

The End!