Angry Eyes

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61 B/M -first seen 1990

- Diagnosed Angioid Streaks* (20/20-)

*Angioid streaks:

2^0 to A1 Hemoglobinopathy

61 B/M -first seen 1990

- Diagnosed Angioid Streaks* (20/20-)

*Angioid streaks:

Algorithm for etiologies of AS

PEPSI T

What does the mnemonic PEPSI "T" remind us of?

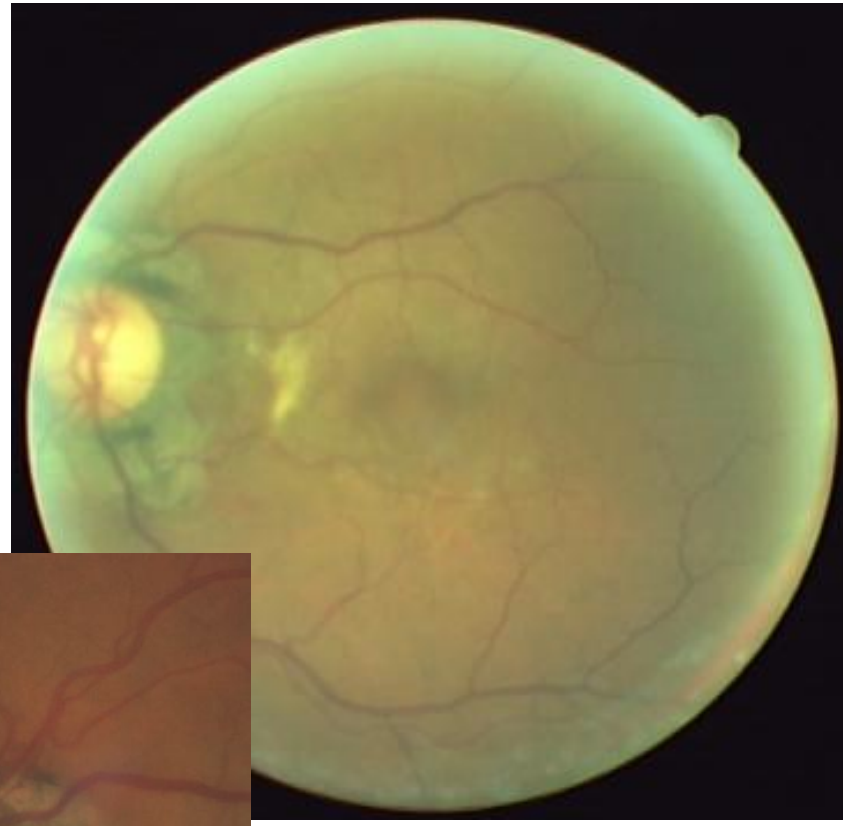
A newer mnemonic

- Psuedoxanthoma elasticum
- ~~Ehlers-Danlos syndr~~
- Paget's disease of bo
- Sickle-cell disease
- Idiopathic
- **H**omocysteinuria
- **A**cromegaly
- **M**arfans syndrome

What is the mechanism of CNV in each of these

61 B/M

- Followed X 12 years - Angioid Streaks 2^0 Al Hemoglobinopathy
- VA 20/25 (OS) – note less involvement

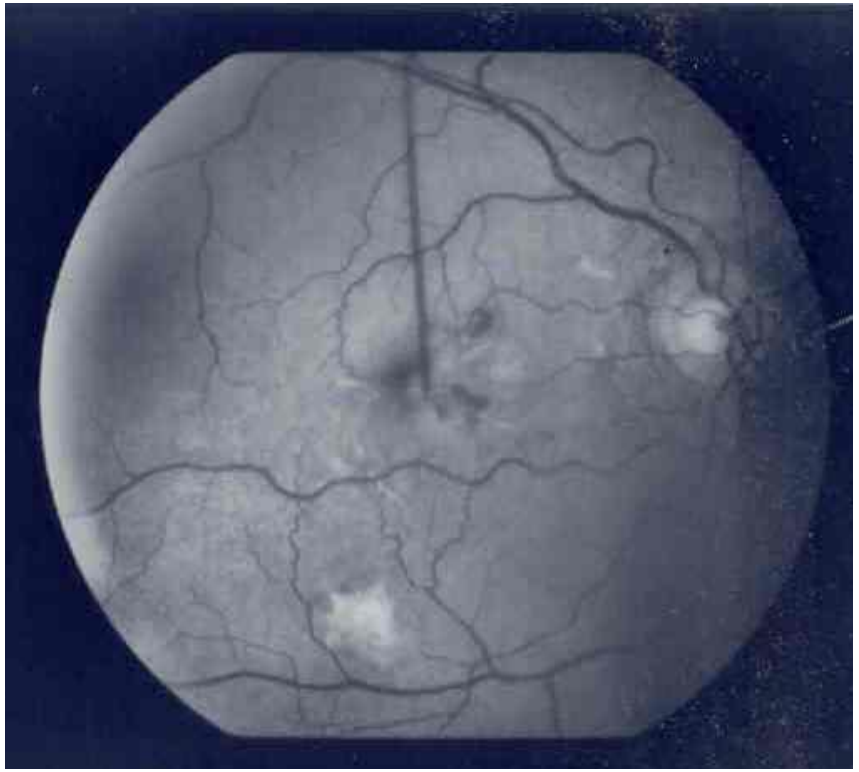


- 12/02

Angioid Streaks - CNVM

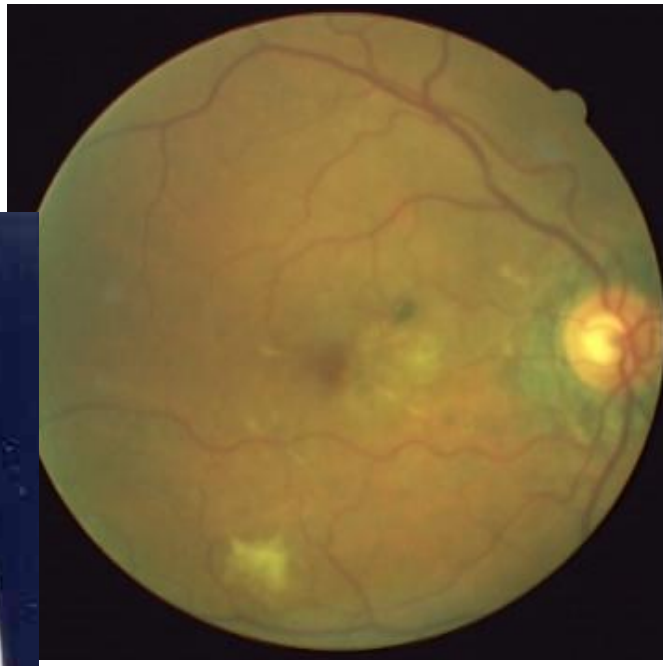
- 62 B/M (10/03)
- OS now symptomatic
- VA 20/30 (OD) note increased involvement
- RTC 4-5 mo.

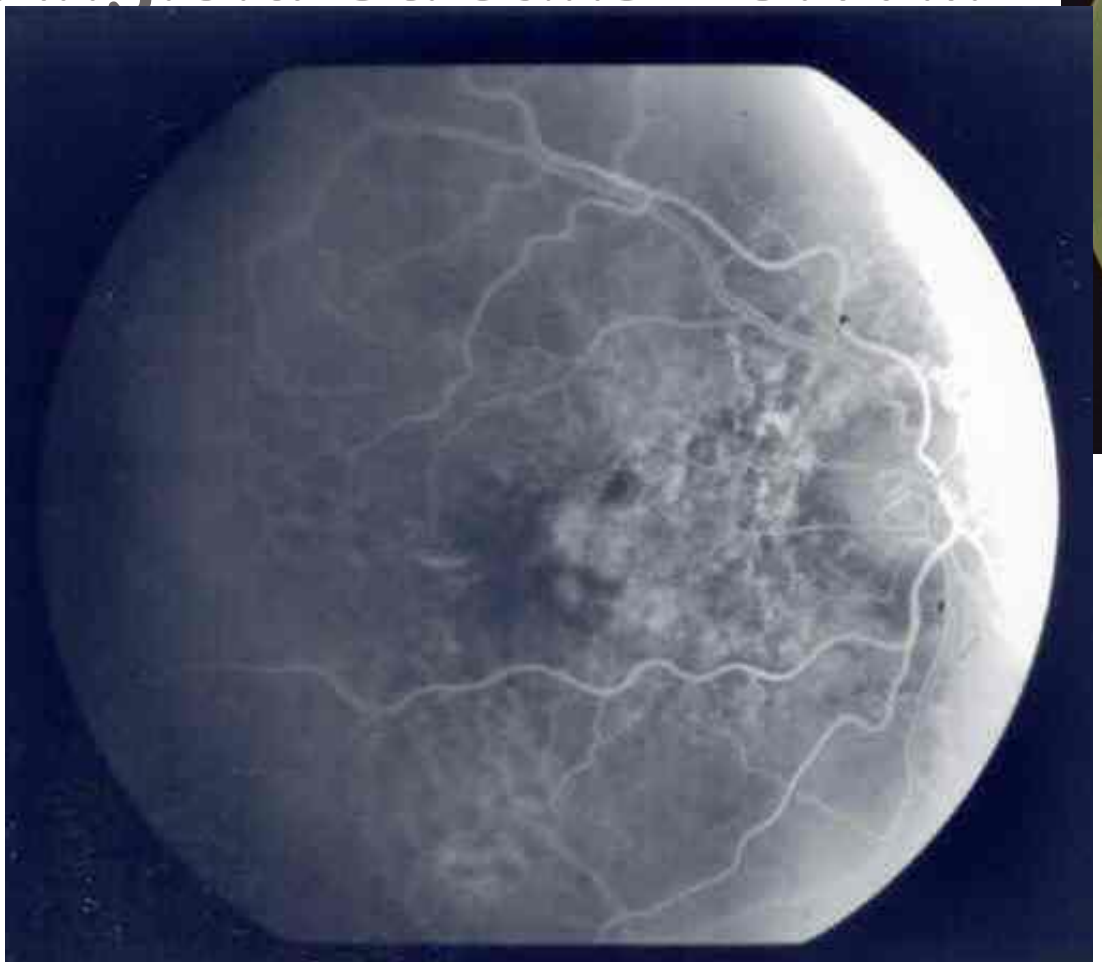
11/03 –compare RF and clinical (3 weeks later)



Red Free

Angioid Streaks - CNVM*

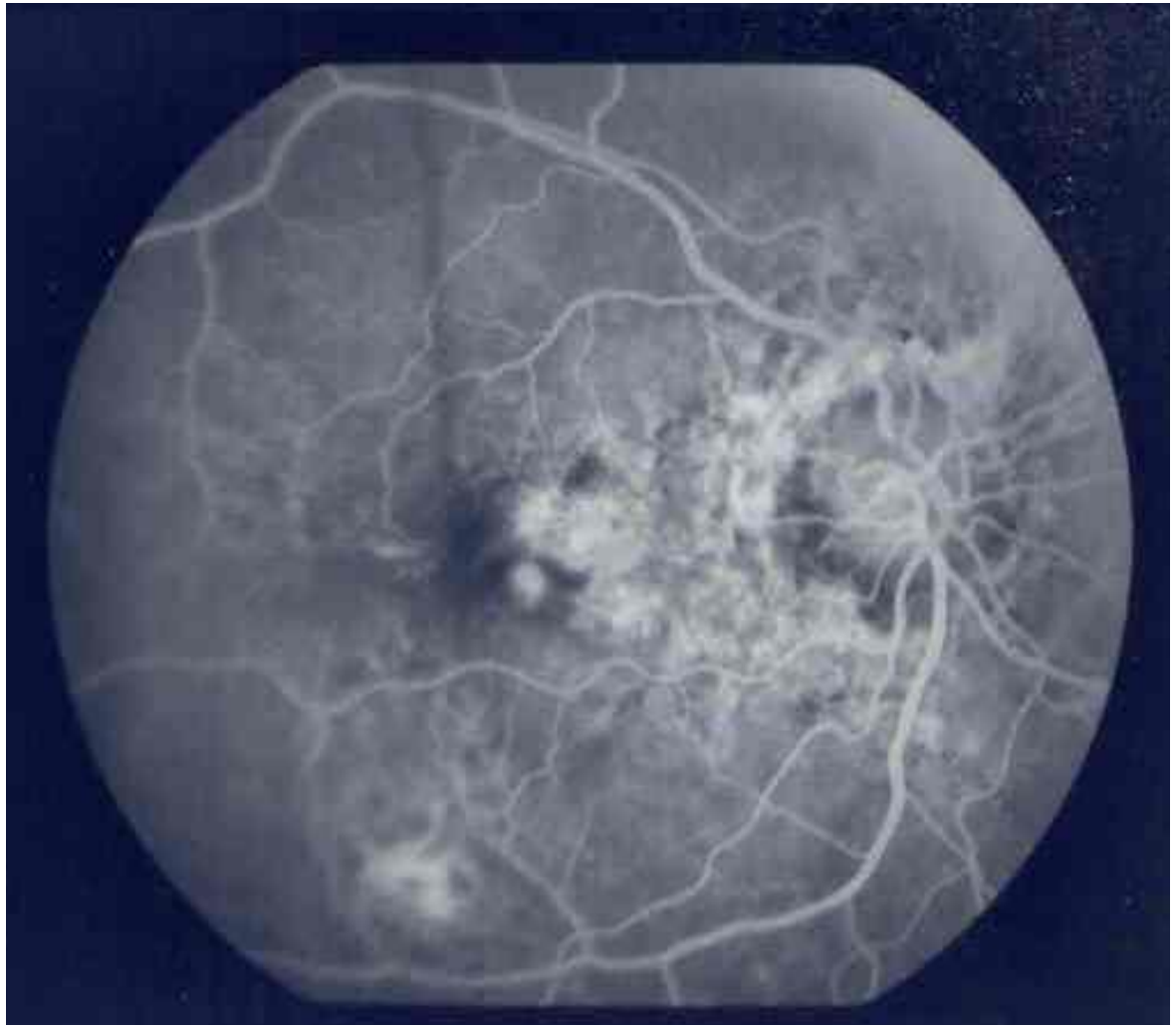




Early – note Hyperfluorescence (leakage)

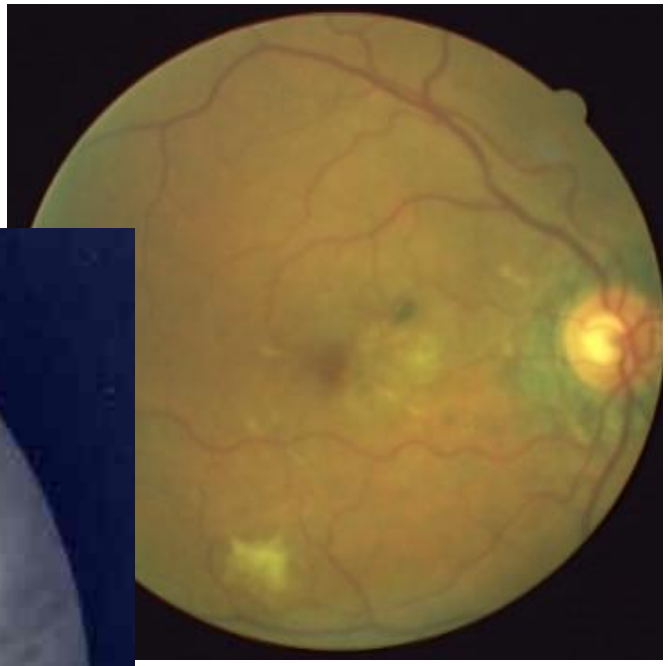
*CNVM mechanism is secondary to iron deposition at BM and consequent brittleness

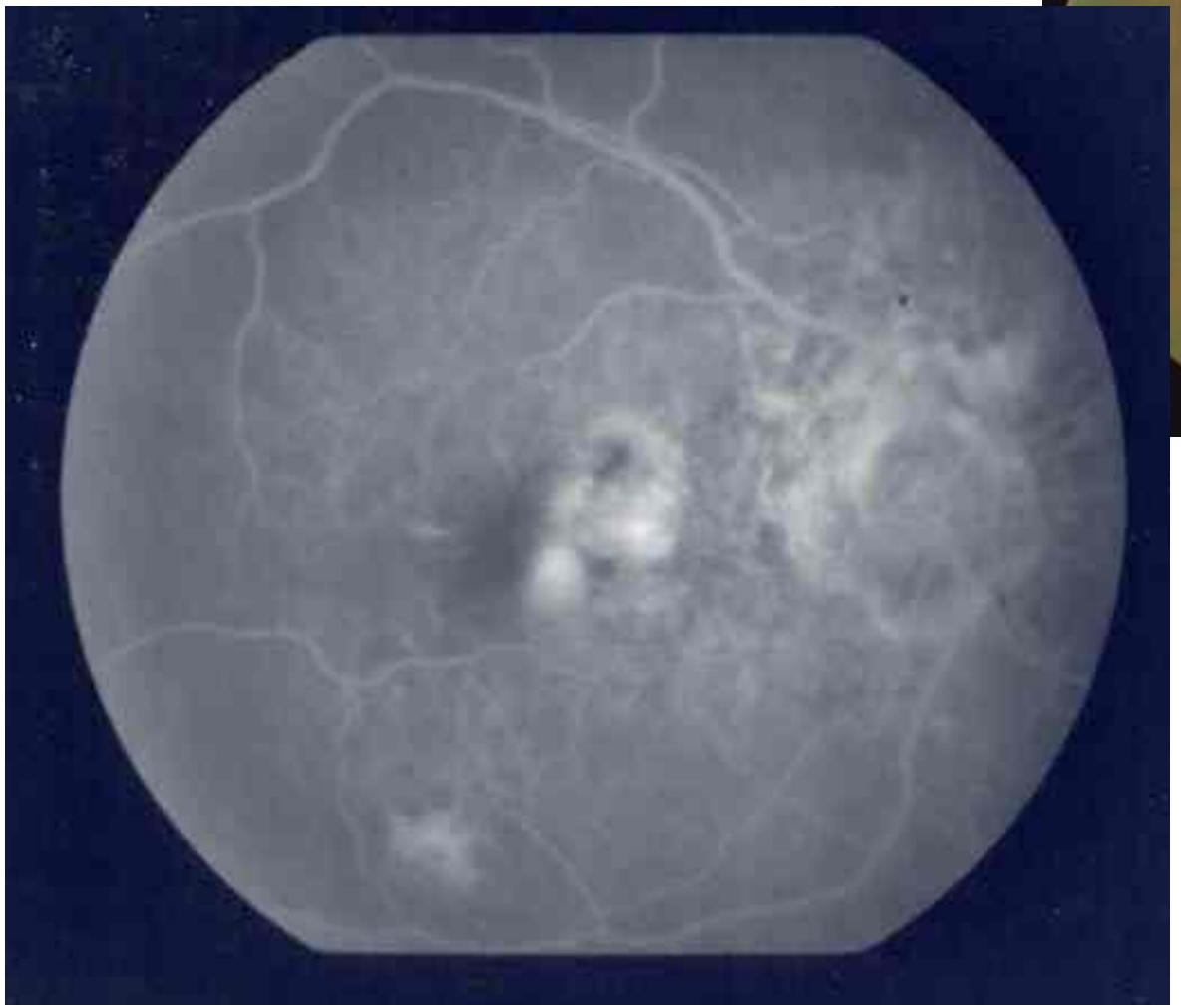
Angioid Streaks - CNVM



Midphase:
Note leakage associated
with Angioid Streaks = CNVM

Angioid Streaks - CNVM

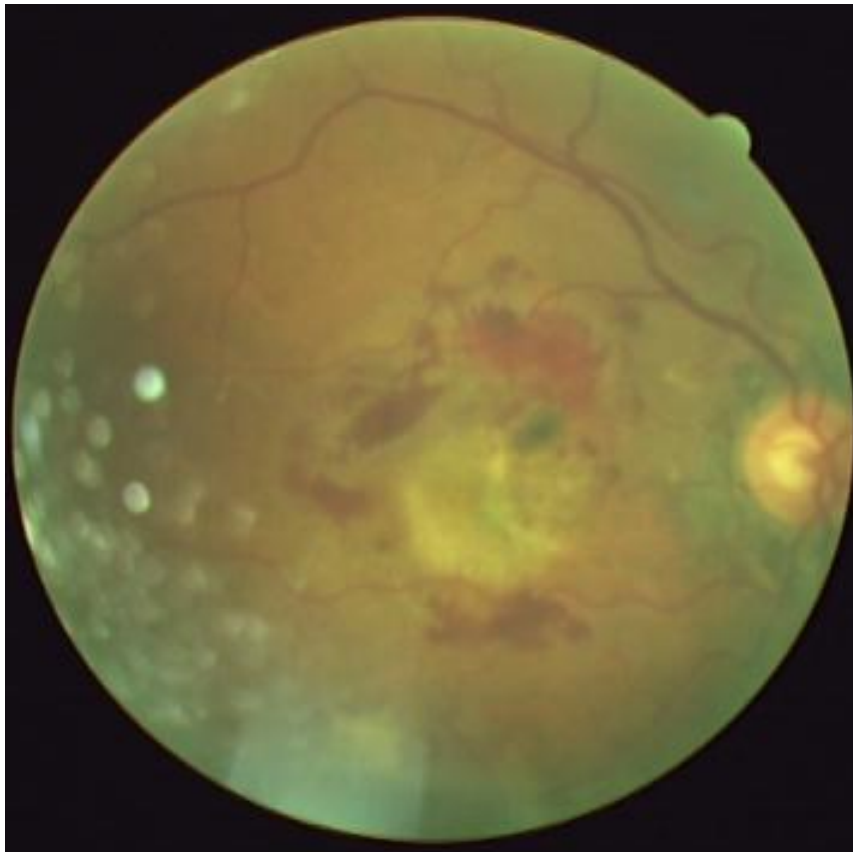




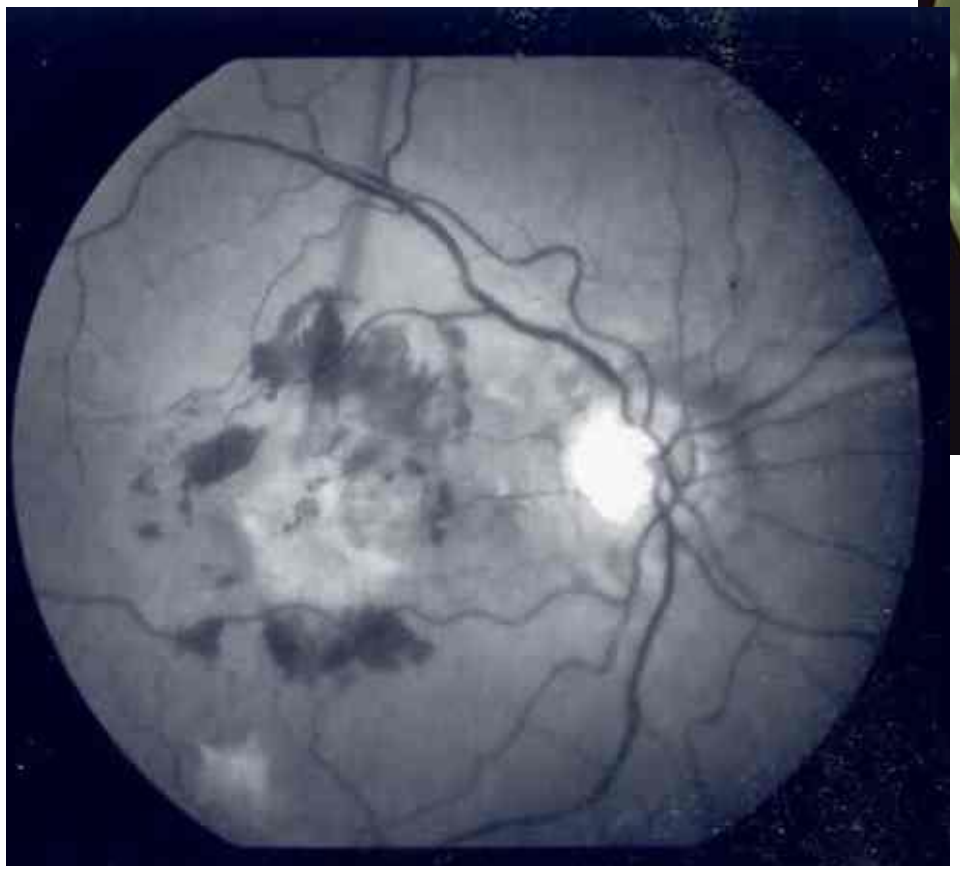
- Late phase leakage consistent with CNVM
- Verteporfin

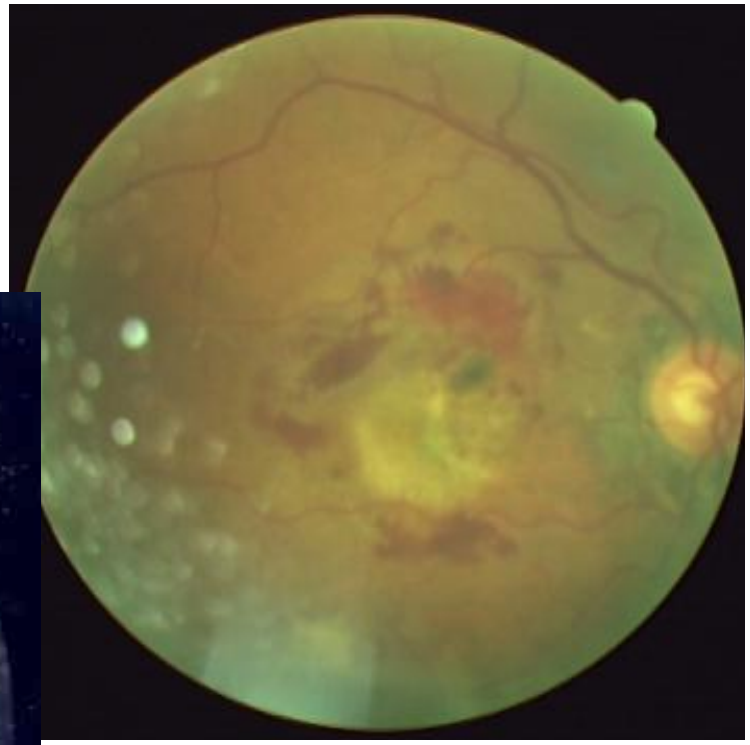
Angioid Streaks - CNVM

- 63 B/M (9/04)
- VA 20/80 (OD) note significant clinical picture consistent with remodeling following verteporfin (Visudyne©) treatment



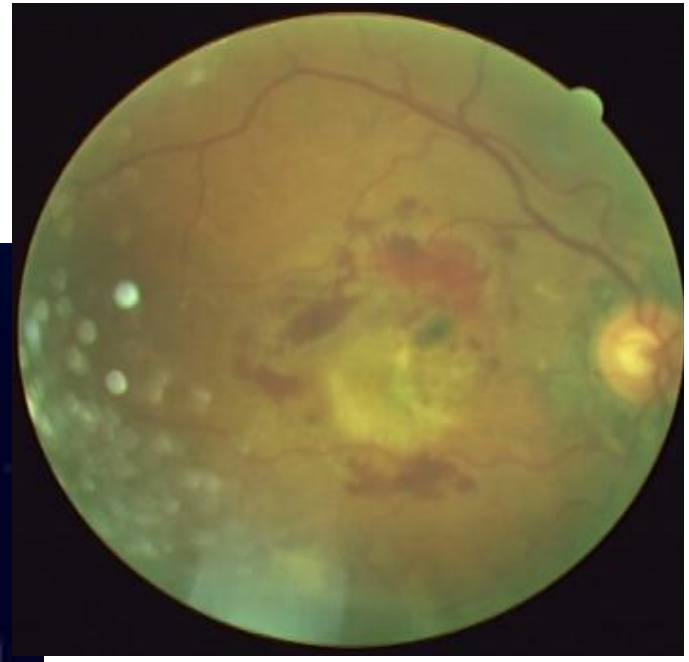
61 B/M (9/04)





RF

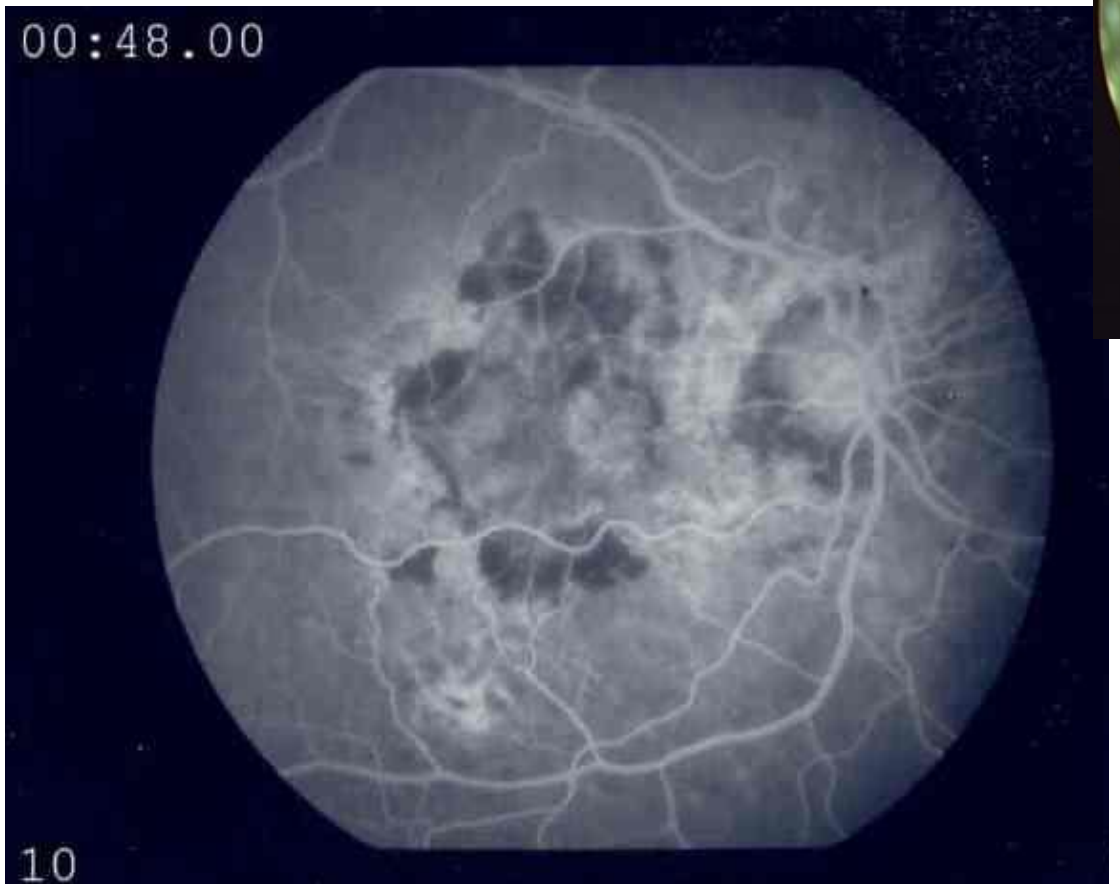
31 sec

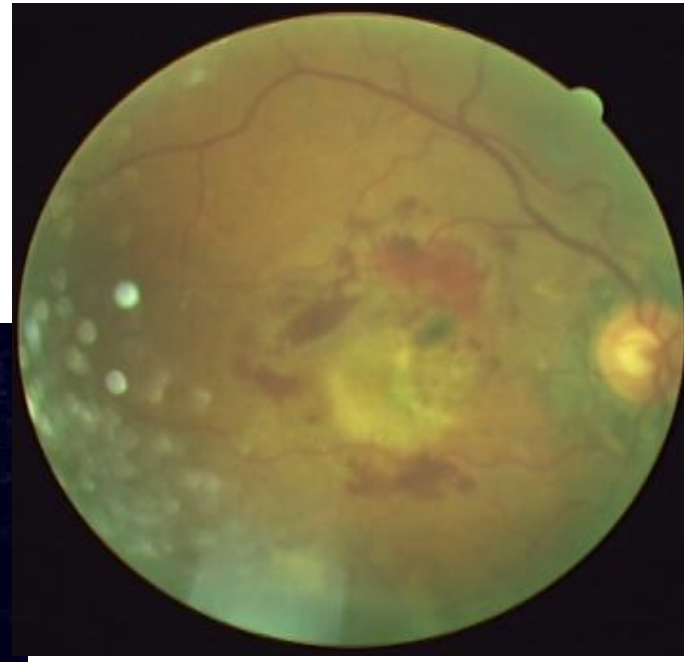


Note poor perfusion,
Lack of leakage

Indicating stable macula

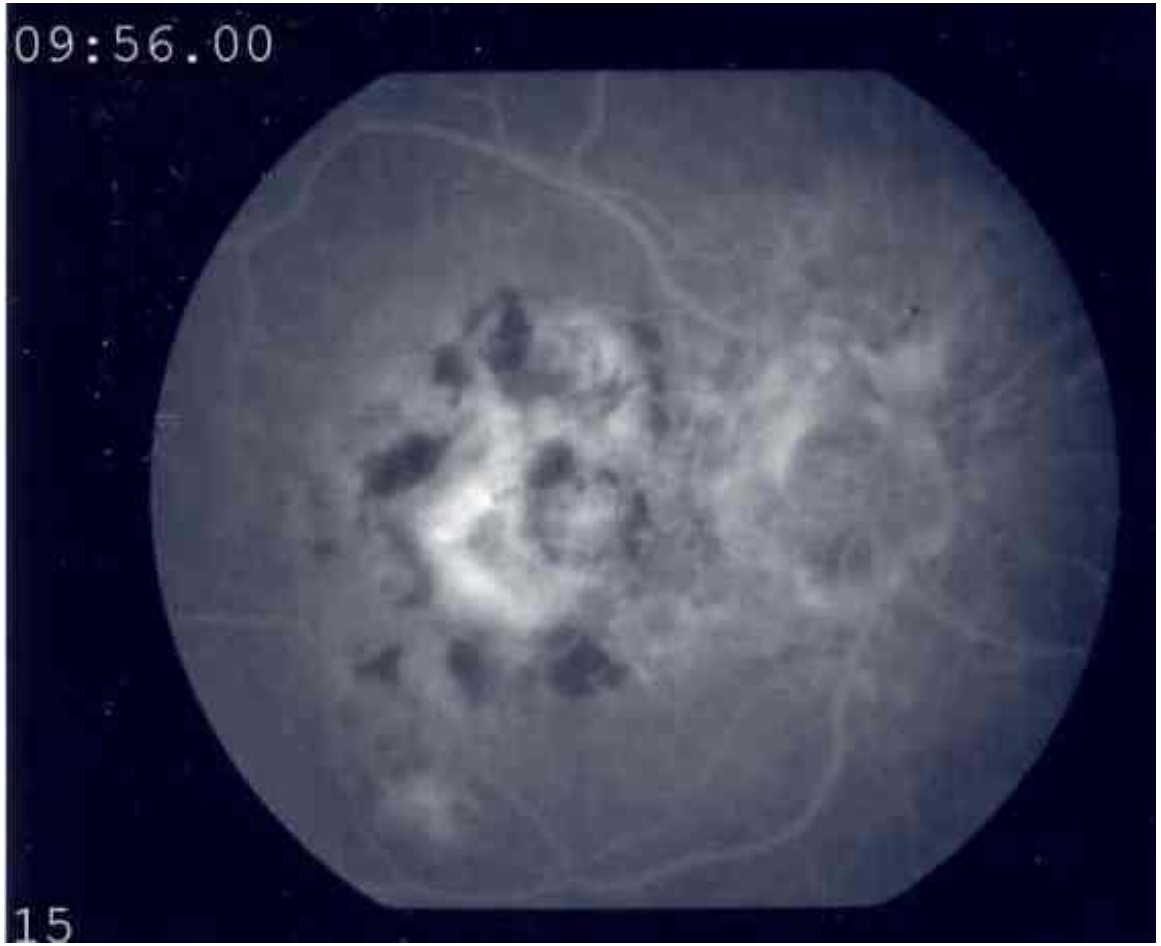
48 sec.

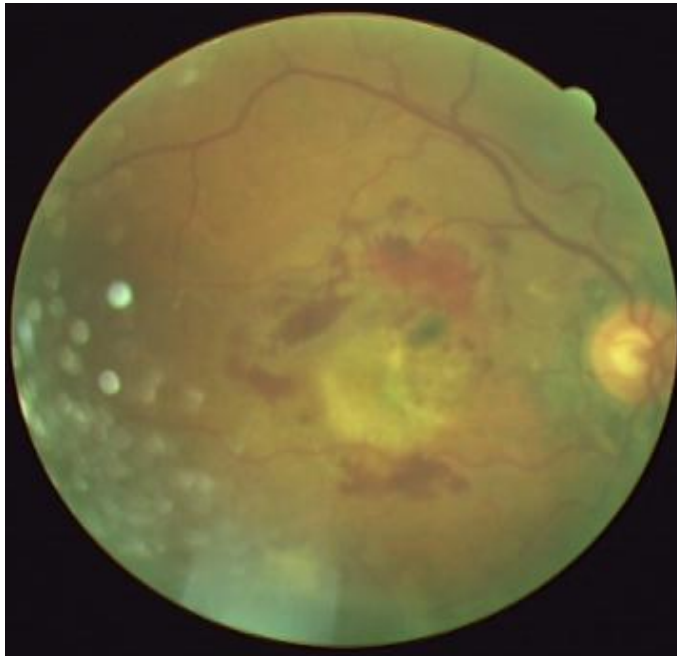




Note lack of leakage
(stabilized CNVM)

9:56 sec





Late-phase staining
No leakage

LS

OD – S/P verteporfin (Visudyne©) treatment

VA = 20/200 X 18 years

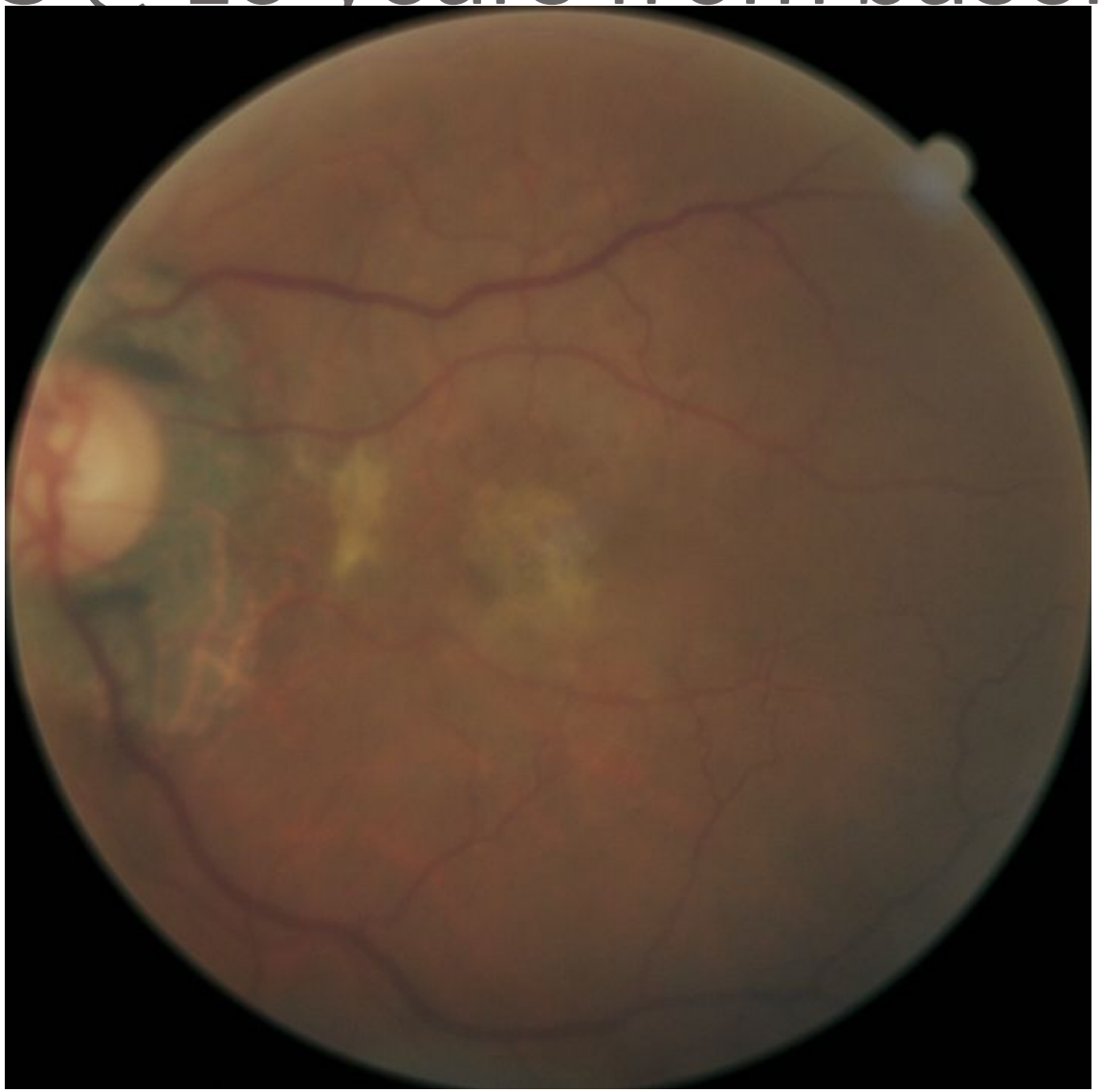
VA = 20/200 X 19 years

OS – a different story

18 years from baseline
– sub macular fluid
VA = 20/80

Treatment with
Avastin©

OS @ 19 years from baseline



S/P 3 Avastin injections

VA = 20/40 with GA; flat macula

- Questions
- Comments

86 YOWM

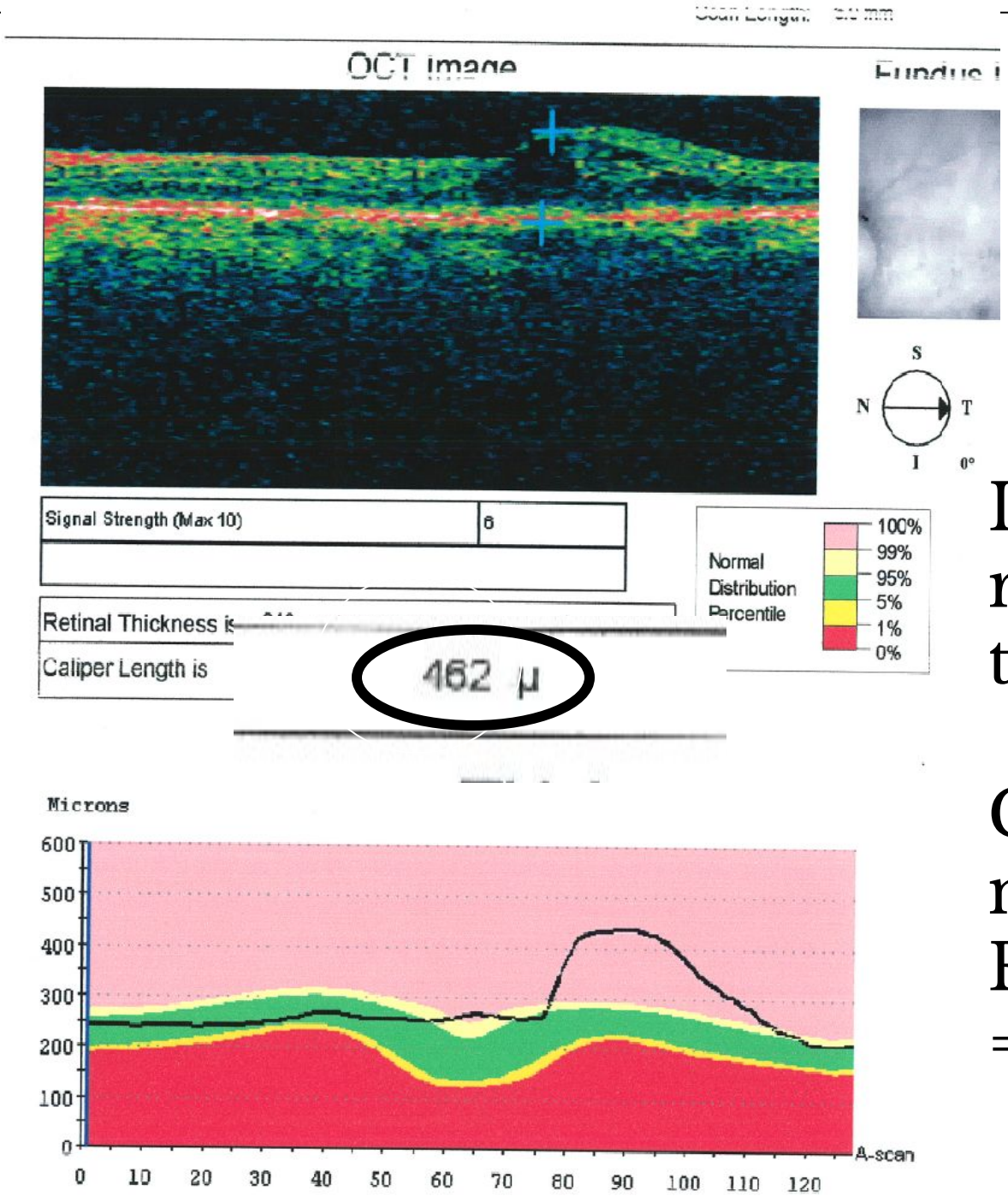
- Presents with reduced VA OS
- POH: repaired peripheral retinal hole SN OS X 11 yrs
- Pseudophakic in each eye
- Medicated for HTn X 20 yrs

20/80 - What's the diagnosis and management?

AMD - anti-VEGF injection

VS.

NV Type III (intraretinal neovasc.) - anti-VEGF injection



Basel ine

Intact RPE with retinal thickening

Caliper to measure Retinal thickness = 462 u

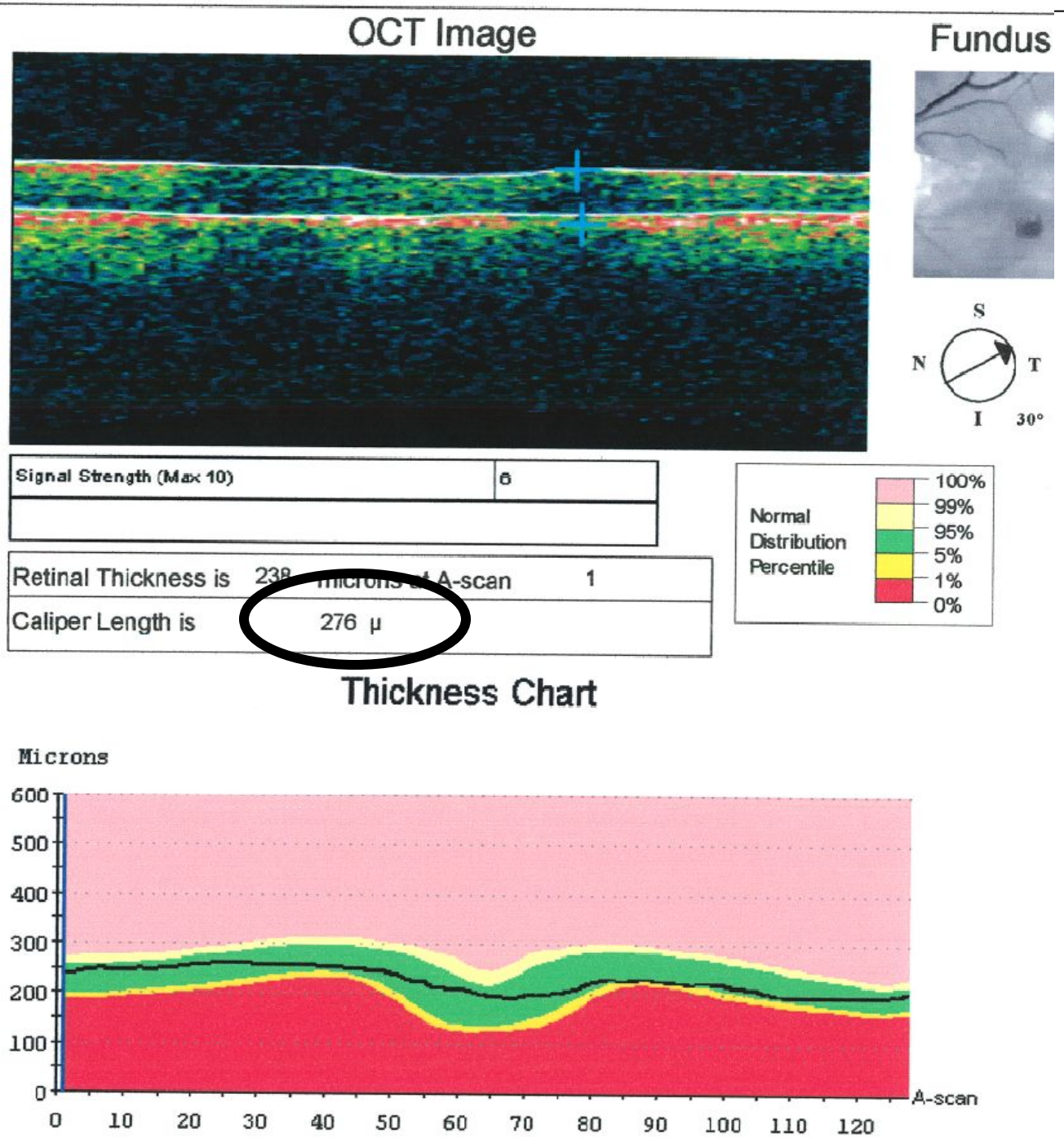
Retinal angiomatous proliferation

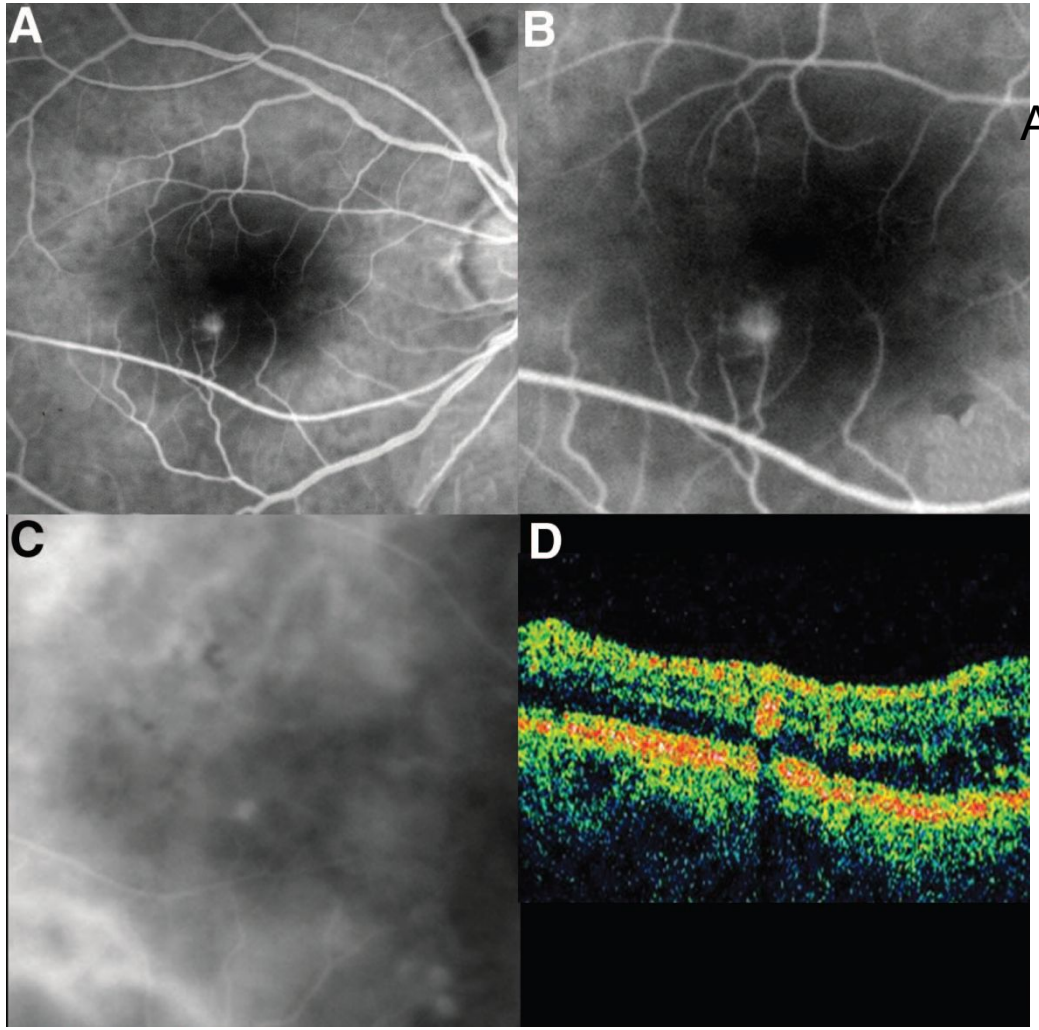
- Aka Type III neovascularization (intraretinal NV)
- Management
  - Avastin injection ⇒ 20/40 @ 3 weeks
  - 20/60 with 4 additional treatments @ 2 years

Neovascularization
Type I – CNVM
Type II – retinal teleangiectasia

3 weeks S/P 1 Avastin injection

Note retinal thickness response 276 u





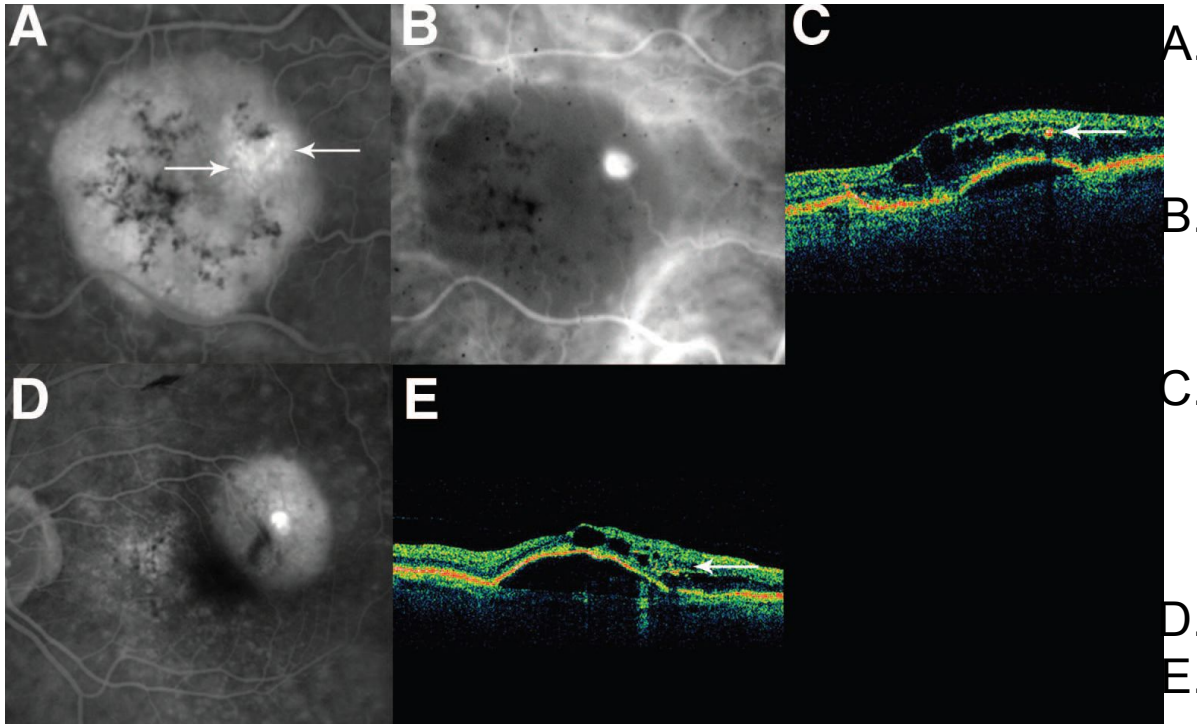
A. FA with early leakage of intraretinal vessels & accompanying teleangiectatic vessels.
B. Magnified view of RAP lesion

C. ICGA showing a single “hot spot.”

D. TD-OCT showing RAP lesion shadowing deeper structures.

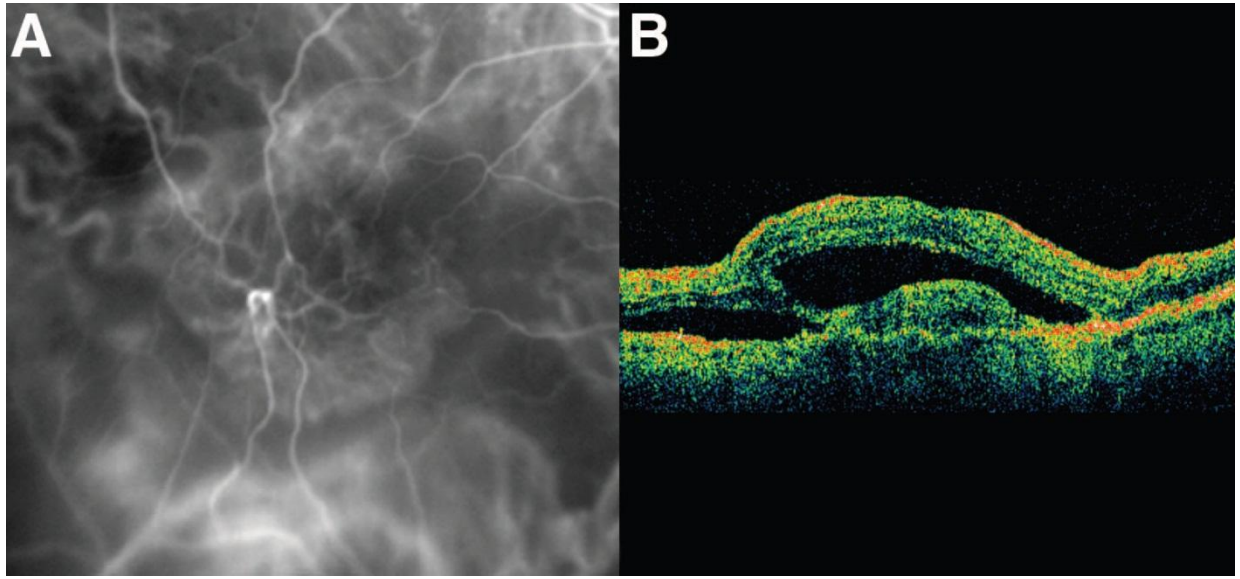
Yannuzzi LA, Freund KB, Takahashi BS. Review of retinal angiomatous proliferation or type 3 neovascularization. Retina. 2008 Mar;28(3):375-84.

Another example (Stage II, w/PED)



A. RAP with hyperfluorescent PED [arrows]
B. ICGA with RAP hot spot, and hypo fluorescent PED
C. OCT; intraretinal RAP [arrow], cystic spaces and PED
D. Another RAP with PED
E. Arrow indicates intraretinal lesion; cystic spaces overlay PED

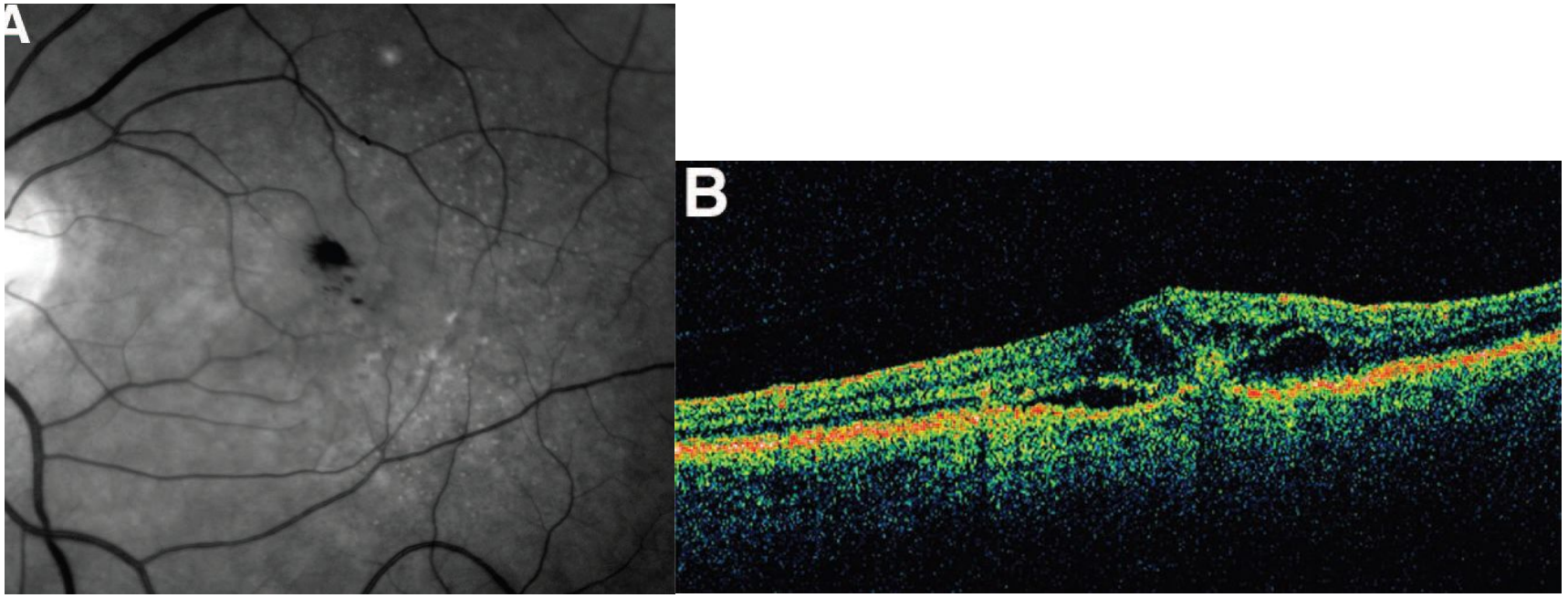
Yannuzzi LA, Freund KB, Takahashi BS. Review of retinal angiomatous proliferation or type 3 neovascularization. Retina. 2008 Mar;28(3):375-84.



A. ICGA showing communication among intraretinal, subretinal and choroidal NV.

B. OCT shows choroidal invasion into subretinal space; RAP not well defined

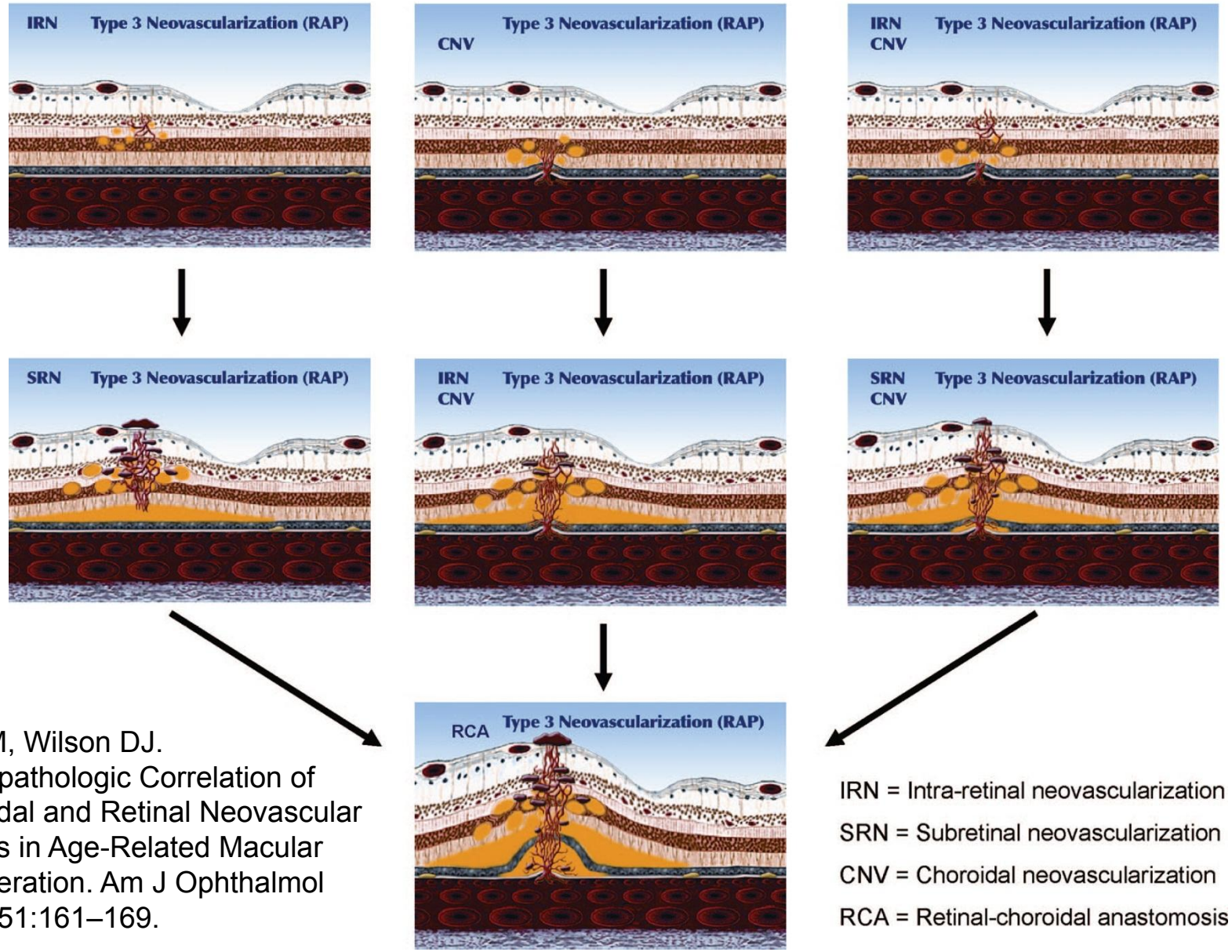
Yannuzzi LA, Freund KB, Takahashi BS. Review of retinal angiomatous proliferation or type 3 neovascularization. Retina. 2008 Mar;28(3):375-84.



A. RAP [note drusen]
B. OCT suggesting choroidal invasion of subretinal space

Yannuzzi LA, Freund KB, Takahashi BS. Review of retinal angiomatous proliferation or type 3 neovascularization. Retina. 2008 Mar;28(3):375-84.

TYPE 3 NEOVASCULARIZATION (RAP)



Klein M, Wilson DJ. Clinicopathologic Correlation of Choroidal and Retinal Neovascular Lesions in Age-Related Macular Degeneration. Am J Ophthalmol 2011;151:161–169.

IRN = Intra-retinal neovascularization

SRN = Subretinal neovascularization

CNV = Choroidal neovascularization

RCA = Retinal-choroidal anastomosis

The latest on RAP - OCTA evaluation

OPEN ACCESS
ARVO Annual Meeting Abstract | July 2018

Evaluating Retinal Angiomatous Proliferation With Optical Coherence Tomography Angiography

LIJUN SHEN; Jianbo Mao

Conclusions : Optical coherence tomography angiography is a noninvasive fast imaging modality for detecting vascular changes in RAP. Hot spot lesion and relative vessels showed more clearly on OCTA than on ICGA or FA. The OCTA patterns of hot spot maybe helpful in understanding the pathology of RAP. And OCTA would be a valuable tool to the follow up observation.

RAP

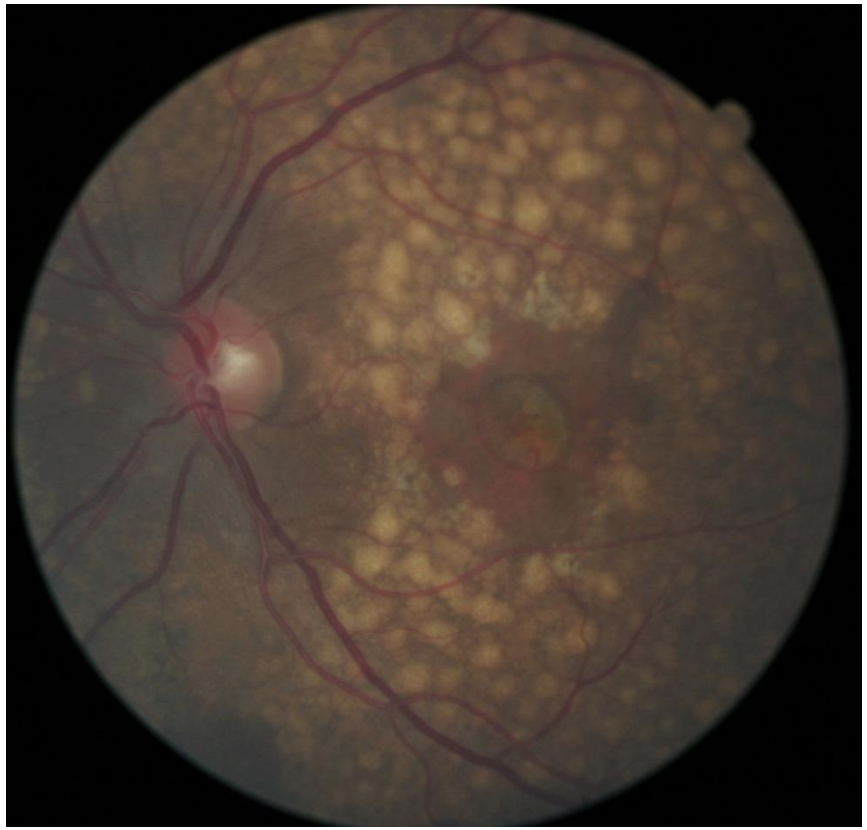
- Questions
- Comments

57 AA F

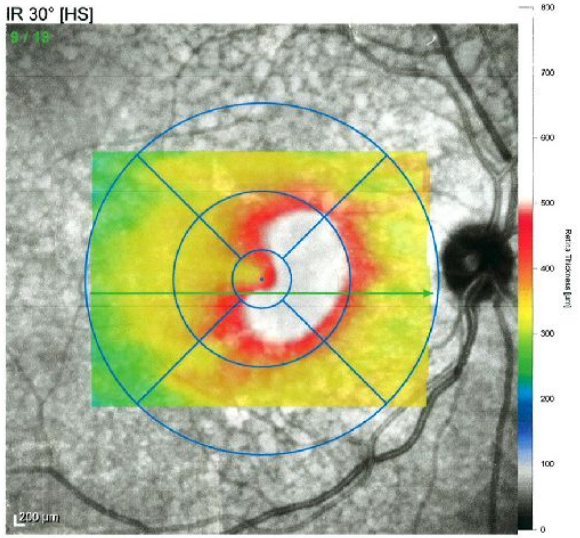
- Presents to UABSO with a history of reduced VA (OD> OS) because,

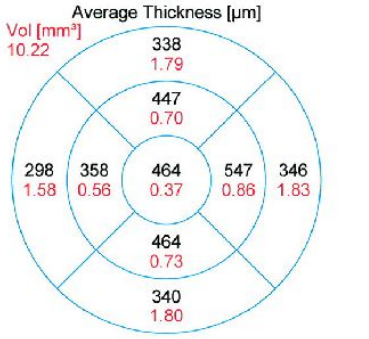
She had been told elsewhere that she had glaucoma.

- Ocular and family histories non-contributory
- Medical history: Tx for HTN X 12 yrs.
- VA 20/200, 20/80

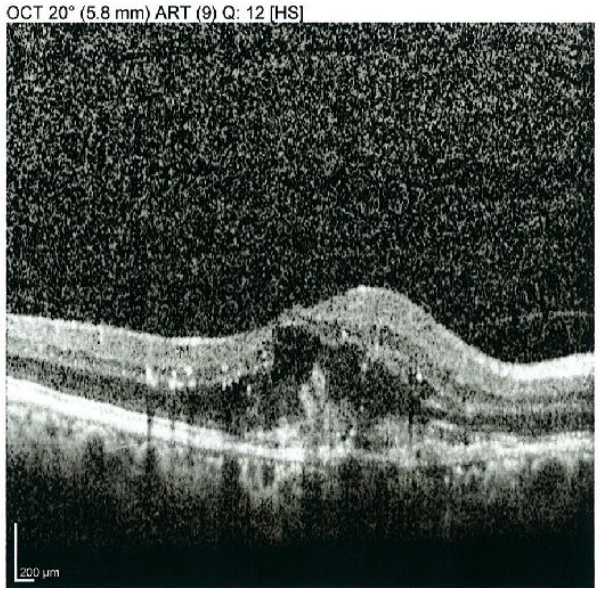


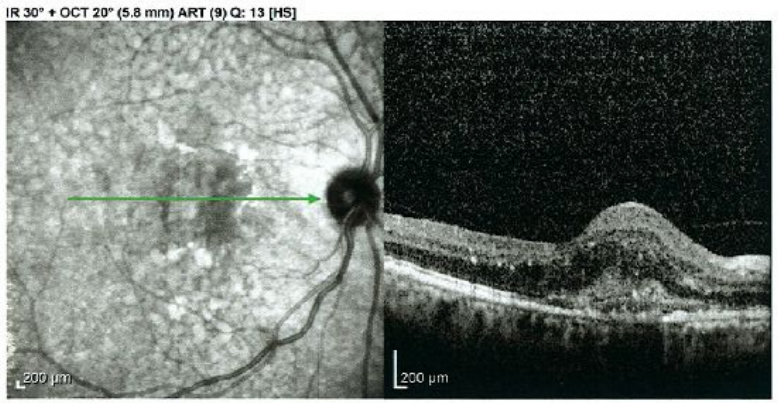
What's the cause of her reduced VA?
What's your diagnosis?
What's next?

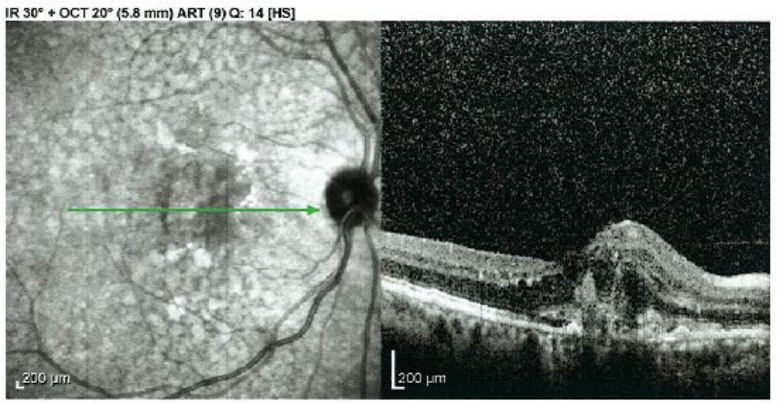




Center: 413 µm
Central Min: 336 µm
Central Max: 583 µm
Circle Diameters: 1, 3, 6 mm ETDRS




IR 30° + OCT 20° (5.8 mm) ART (9) Q: 13 [HS]
200 µm
200 µm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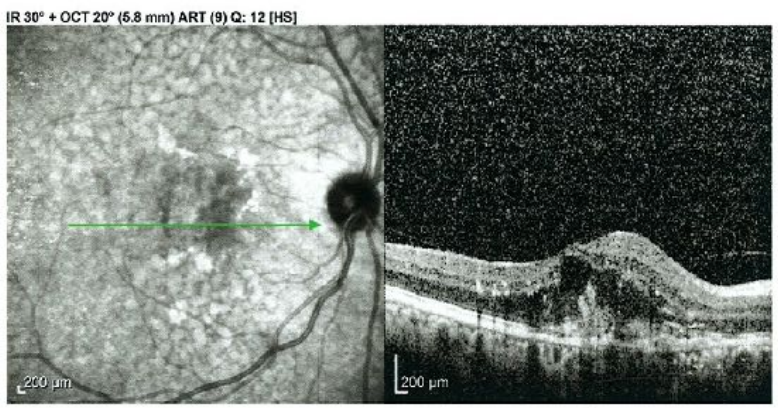
200 µm
200 µm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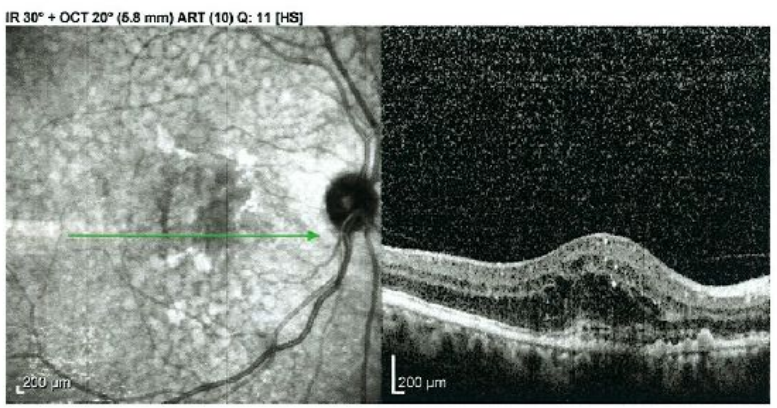
200 µm
200 µm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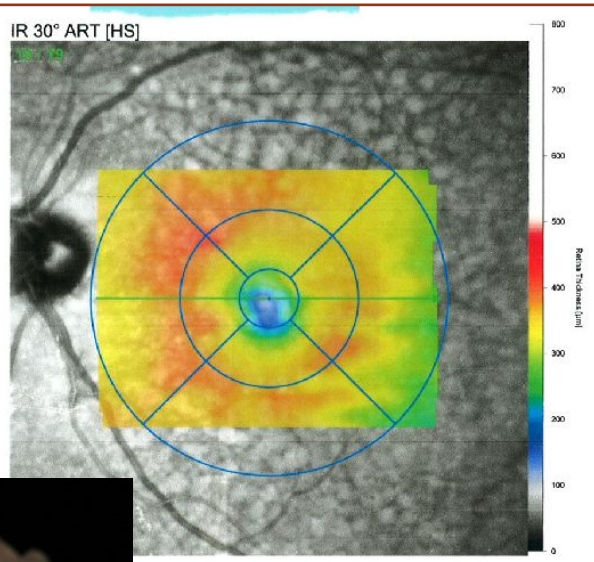
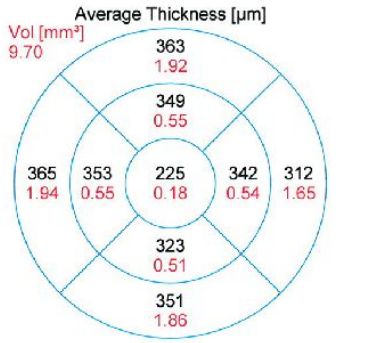
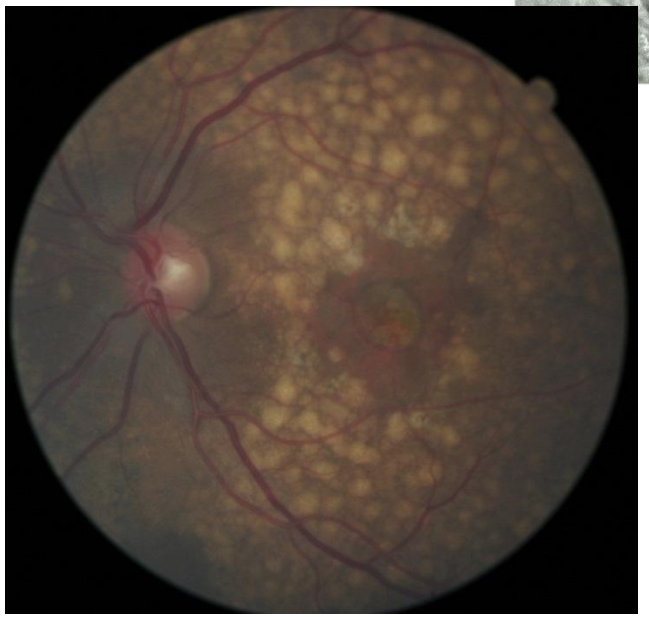
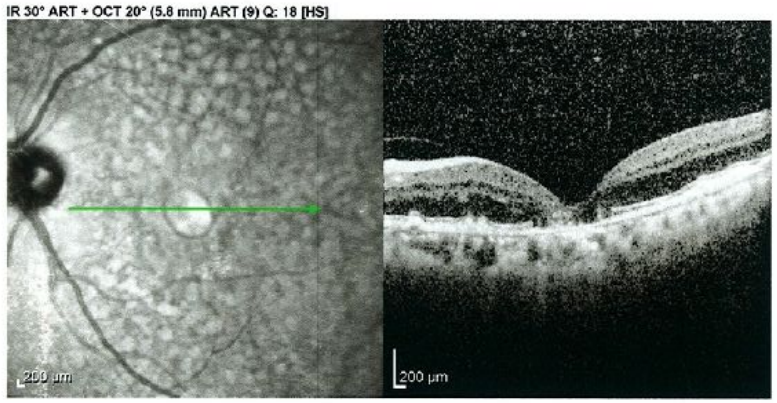
200 µm
200 µm

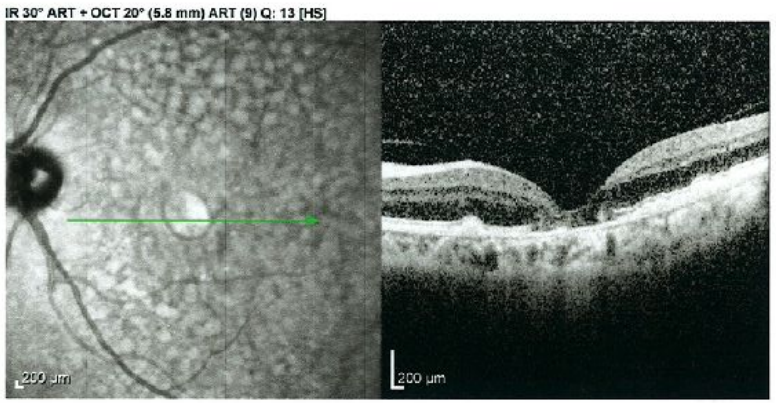


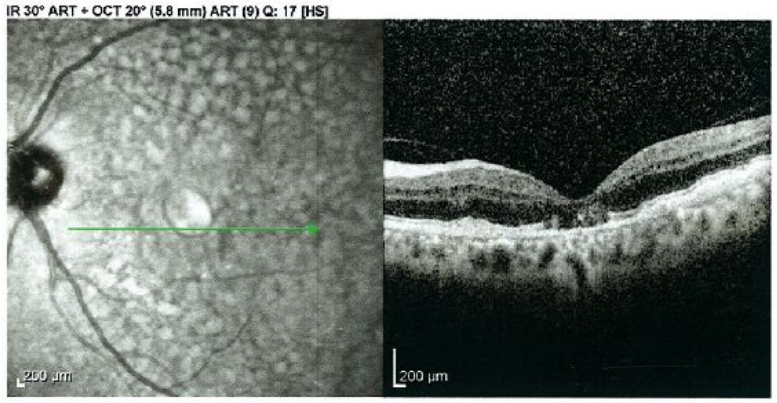


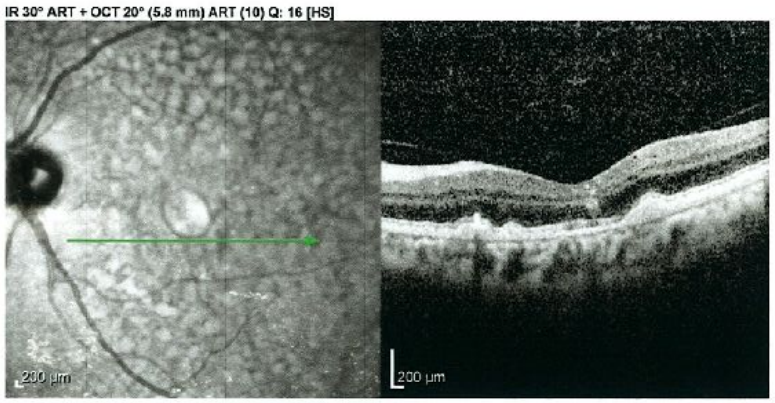
Center: 162 µm
Central Min: 146 µm
Central Max: 324 µm
Circle Diameters: 1, 3, 6 mm ETDRS




IR 30° ART + OCT 20° (5.8 mm) ART (9) Q: 18 [HS]
200 µm
200 µm


IR 30° ART + OCT 20° (5.8 mm) ART (9) Q: 13 [HS]
200 µm
200 µm


IR 30° ART + OCT 20° (5.8 mm) ART (9) Q: 17 [HS]
200 µm
200 µm


IR 30° ART + OCT 20° (5.8 mm) ART (10) Q: 16 [HS]
200 µm
200 µm

Rare presentation of *Malattia Leventinese*

(aka Doyne's honeycomb macular dystrophy)

- Eleonora Corbelli, et al. Optical Coherence Tomography Angiography Demonstration of Choroidal Neovascularization in Malattia Leventinese. Ophthalmic Surgery, Lasers and Imaging Retina. June 2016 - Volume 47 · Issue 6: 602-604.

- Serra R, Coscas F, Messaoudi N, Srour M, Souied E. Choroidal Neovascularization in Malattia Leventinese Diagnosed Using Optical Coherence Tomography Angiography. Am J Ophthalmol. April 2017Volume 176, Pages 108–117 DOI: http://dx.doi.org/10.1016/j.ajo.2016.12.027

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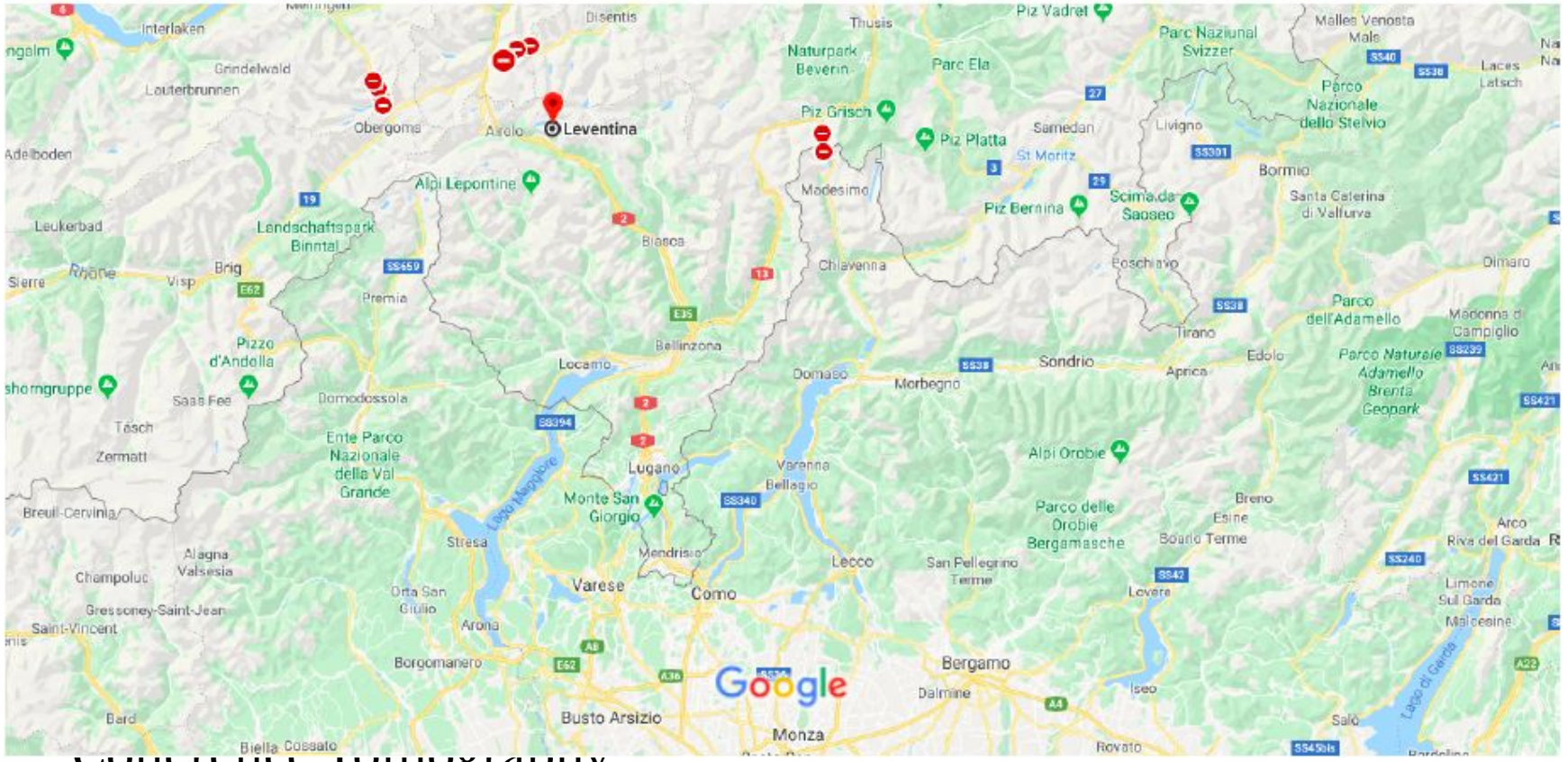

Conference Tomography Angiography. Am J Ophthalmol. April 2017Volume 176, Pages 108–117 DOI:

Rare presentation of *Malattia Leventinese*

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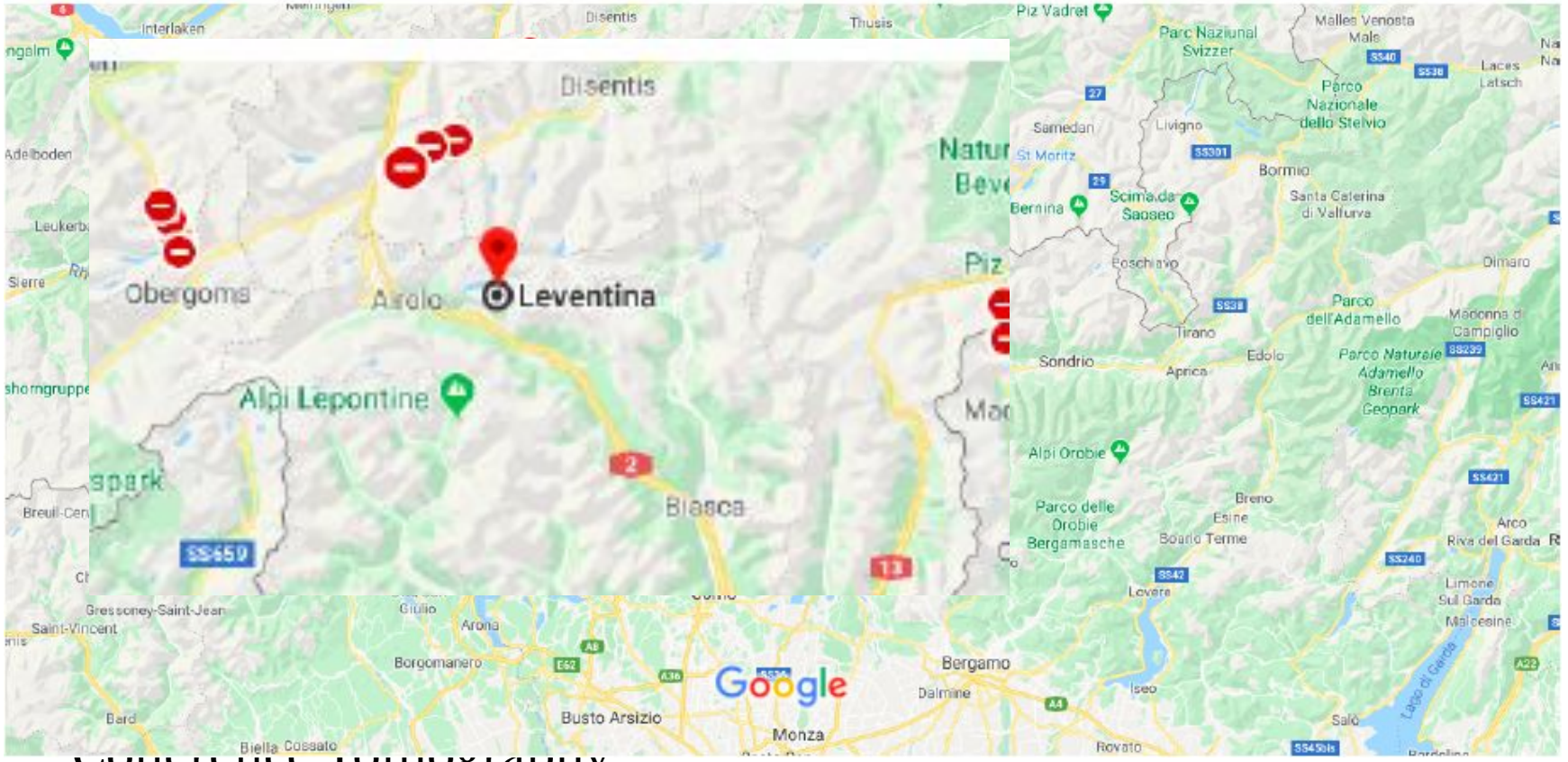


Angiography. Am J Ophthalmol. April 2017Volume 176, Pages 108–117
DOI:

From the literature…

WF fundus image showing drusenoid deposits and subretinal neovascularizaion

FAF demonstrating intense autofluorescense of large drusen

SD-OCT X-section demonstrating subretinal fluid (hyporeflective area) and RPE elevations

Corbelli E, et al. Optical Coherence Tomography Angiography Demonstration of Choroidal Neovascularization in Malattia Leventinese. OSLI Retina. 2016 47 (6) : 602-604

Malattia Leventinese

- Questions
- Comments

Case: 39 W/M

- Initial presentation:
  - Desires second opinion on treatment for retinal problem
  - History: taking clindamycin X 6 weeks PO + steroids PO X 3 weeks (D/C)
  - VA (OD) 20/200, mild vitritis, no A/C rxn., all else unremarkable; OS uninvolved

39 W/M

- OD Fundus appearance @ initial presentation

Polling questions

- Should this patient's treatment be modified?
- Is the treatment consistent with your diagnosis?
- Does this patient need an anti-VEGF injection?

39 W/M

- Initial treatment
  - *Bactrim DS* (160 mg. Trimethoprim + 800 mg. Sulfmethoxazole)* PO bid X 2 weeks
  - RTC X 2 wks

Current recommended treatment when offered.
Lima GSC, Saraiva PGC, Saraiva FP. Current therapy of acquired ocular toxoplasmosis: a review. J O P T. 2015 (Sep): 511-517.

39 W/M

- 2- week return visit (fundus appearance)

39 W/M

- VA unchanged
  - Vitritis slightly diminished
  - Fundus appearance essentially unchanged
  - Options
    - Continue meds???
    - Change meds???
    - Add meds???
    - Refer???

39 W/M

- 4-week return visit
  - VA unchanged
  - Vitritis resolved; 1+ A/C reaction
  - Fundus appearance essentially unchanged
  - Options
    - Continue meds???
    - Change meds???
    - Add meds???
    - Refer???

Case: 39 W/M

- Fundus appearance at discharge visit (8 weeks following initial presentation).

Note retinal vessel communicating with choroidal circulation (*karyolysis retinalis*)

Treatment indications [1]

- Involvement within the macular arcades
- Proximity of lesion to optic nerve
- Relative indications
  - infection in immunocompromised patients
  - marked vitreous reaction (posterior uveitis)
  - active lesion in an only eye
  - presentation in an elderly patient (shown to be more aggressive)

1. Stanford MR, Gilbert RE. Treating ocular toxoplasmosis: current evidence. Mem Inst Oswaldo Cruz. 2009;104(2):312-5.

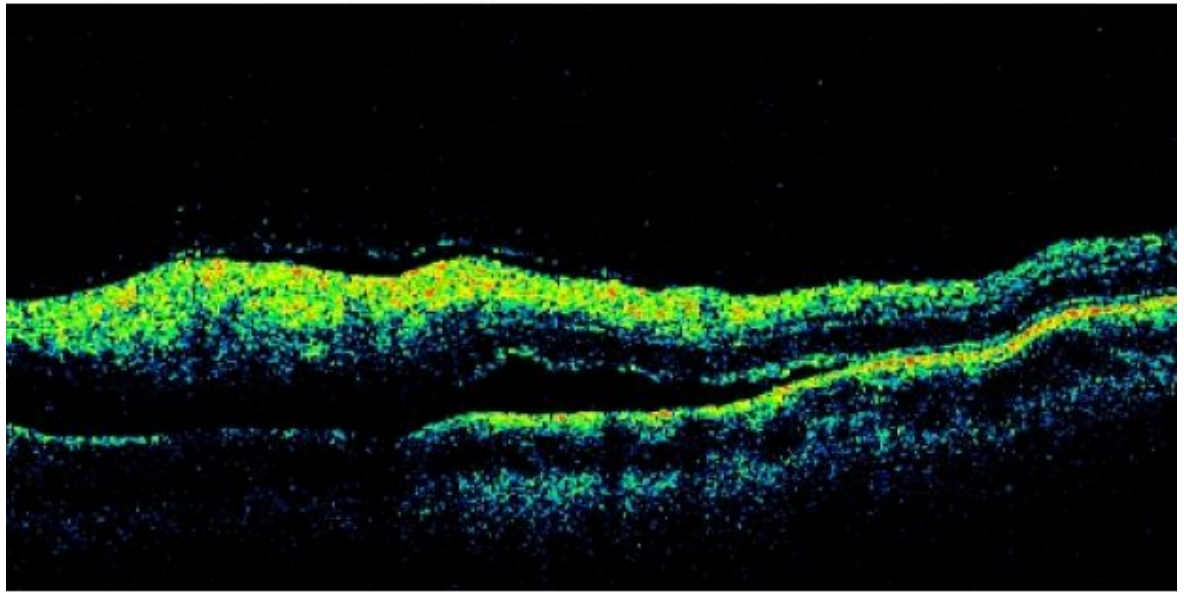
Additional features [1]

- Punctate outer retinal toxoplasmosis
- Retinal vasculitis
- Retinal vascular occlusions
- Rhegmatogenous RD with serous RD
- Unilateral pigmentary changes simulating RP
- Neuroretinitis and other forms of optic neuropathy
- Peripheral retinitis and scleritis
- In children:
  - Cataract, CNV, glaucoma, RD
- Has been associated with Fuch's heterochromic iridocyclitis

1. Commodaro AG, Belfort RN, Rizzo LV, et al. Ocular toxoplasmosis: an update and review of the literature. Mem Inst Oswaldo Cruz. 2009;104(2):345-50.

OCT features of Toxoplasmic retinochoroiditis

- Reflective inner retina in active presentations
- Posterior hyaloid thickened and detached over the lesion
- Shadowing of the underlying choroid
- May have serous fluid



Monnet D, Averous K, Delair E, Brézin AP. Optical coherence tomography in ocular toxoplasmosis. Int J Med Sci. 2009;6(3):137-8.

Management of Ocular Toxoplasmosis

- Clinical Presentation / Diagnosis
  - Observation of a yellowish lesion with overlying inflammatory cells is almost diagnostic
  - Vitritis/choroiditis may accompany
  - Blood tests are definitive (systemic) but not in ocular
    - Sabin-Feldman methylene blue dye test, IgG, IgM, ELISA)
  - Treatment is indicated when the posterior pole is involved

Ocular Toxoplasmosis

- 30% - 50% of all cases of posterior uveitis
- Clinical presentation (same for cong. and acq.)
  - Focus of necrotizing retinitis
  - Moderate to severe vitritis
    - In immunocompromised patients, there may be multiple foci or extensive necrosis

Other Toxoplasmic Retinochoroiditis Risks

- *Eating locally produced cured, dried, or smoked meat*
- *Working with meat*
- *Drinking unpasteurized goat's milk*
- *Having 3 or more kittens*
- *Eating raw oysters, clams, or mussels was significant in a separate model among persons asked this question.*

Jones JL, et al. Risk factors for *T. gondii* infection in the United States. Clin Infect Dis. 2009 Sep 15;49(6):878-84.

From the NAHANES -2018

Am. J. Trop. Med. Hyg., 98(2), 2018, pp. 551–557
doi:10.4269/ajtmh.17-0677

Toxoplasma gondii Infection in the United States, 2011–2014

Jeffrey L. Jones,[1*] Deanna Kruszon-Moran,[2] Scott Elder,[1] Hilda N. Rivera,[1] Cindy Press,[3] Jose G. Montoya,[3,4] and Geraldine M. McQuillan[2]

[1]*Division of Parasitic Diseases and Malaria, Center for Global Health, Centers for Disease Control and Prevention, Atlanta, Georgia;* [2]*Division of Health and Nutrition Examination Statistics, National Center for Health Statistics, Centers for Disease Control and Prevention, Hyattsville, Maryland;* [3]*Palo Alto Medical Foundation, Toxoplasma Serology Laboratory, Palo Alto, California;* [4]*Division of Infectious Diseases and Geographic Medicine, Department of Medicine, Stanford University School of Medicine, Stanford, California*

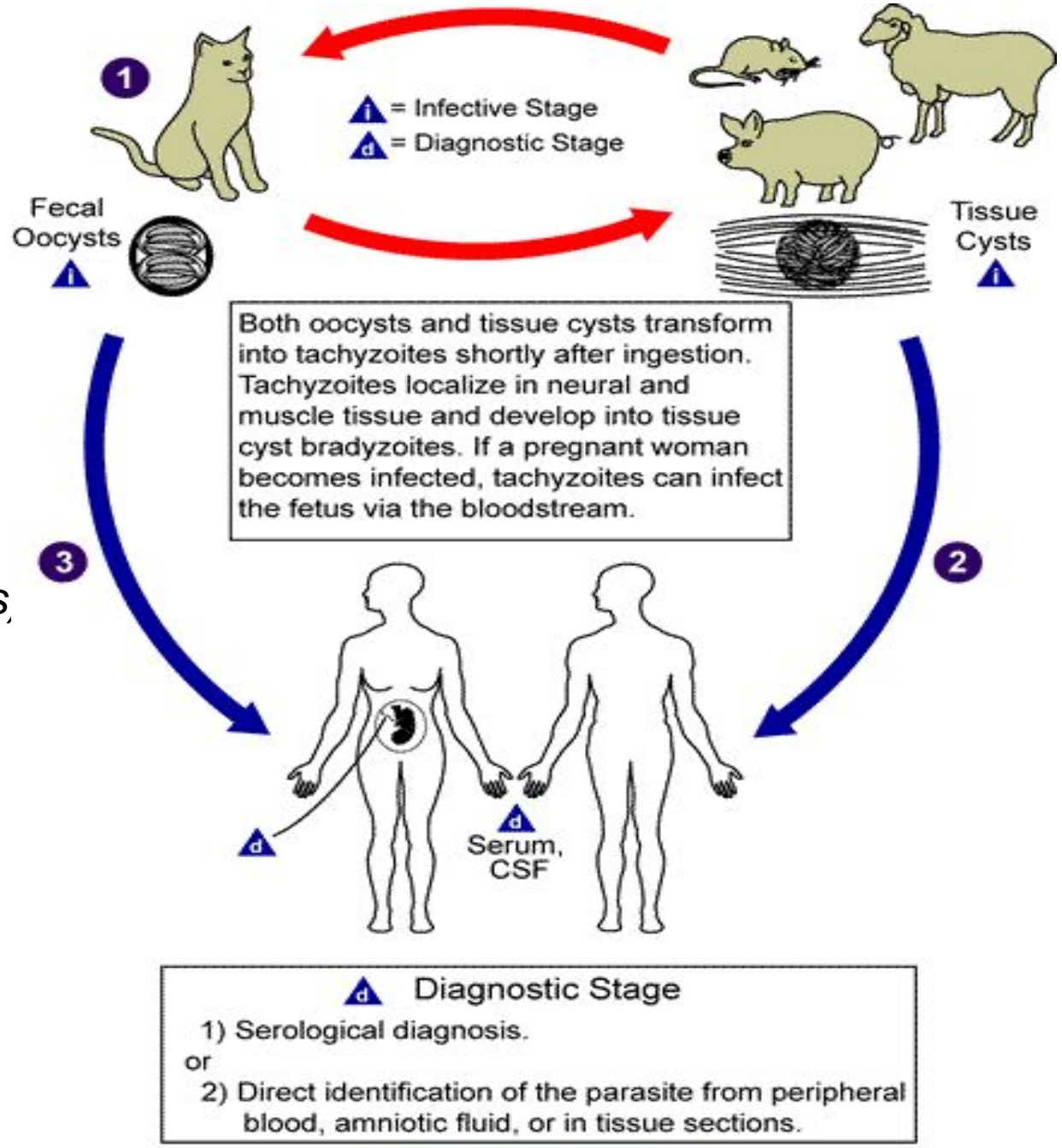
Abstract. *Toxoplasma gondii* can cause severe neurologic and ocular disease when transmitted congenitally and in immunosuppressed persons. Sera collected in the National Health and Nutrition Examination Survey 2011 through 2014 in 13,507 persons ≥6 years old were tested for *T. gondii* immunoglobulin (Ig) G and IgM antibodies, and in those both IgG and IgM antibody positive, for IgG avidity. Overall, 11.14% (95% confidence limits [CL] 9.88%, 12.51%) were seropositive for *T. gondii* IgG antibody (age-adjusted seroprevalence 10.42% [95% CL 9.19%, 11.76%]); in women aged 15–44 years, the age-adjusted *T. gondii* IgG seroprevalence was 7.50% (95% CL 6.00%, 9.25%). In multivariable analysis, risk for IgG seropositivity increased with age and was higher in males; persons living below the poverty level; persons with ≤ a high school education compared with those with > a high school education; and non-Hispanic black, Mexican American, and foreign born non-Hispanic white persons compared with U.S.-born non-Hispanic white persons. Overall, 1.16% (95% CL 0.94%, 1.42%) were *T. gondii* IgM antibody positive and 0.71%, (95% CL 0.54%, 0.92%) were both IgM and IgG antibody positive. In multivariable analysis, the significant risk factors for being both IgM and IgG positive were older age, crowding, and non-U.S. birth origin compared with U.S.-born persons. Among those positive for both IgM and IgG antibody, almost all had high avidity (all women aged 15–44 years had high avidity). *Toxoplasma gondii* antibody prevalence remains relatively low in the United States, although it is higher in non-U.S.–born persons, males, and some minority and socioeconomically disadvantaged groups.

Conclusions . . .

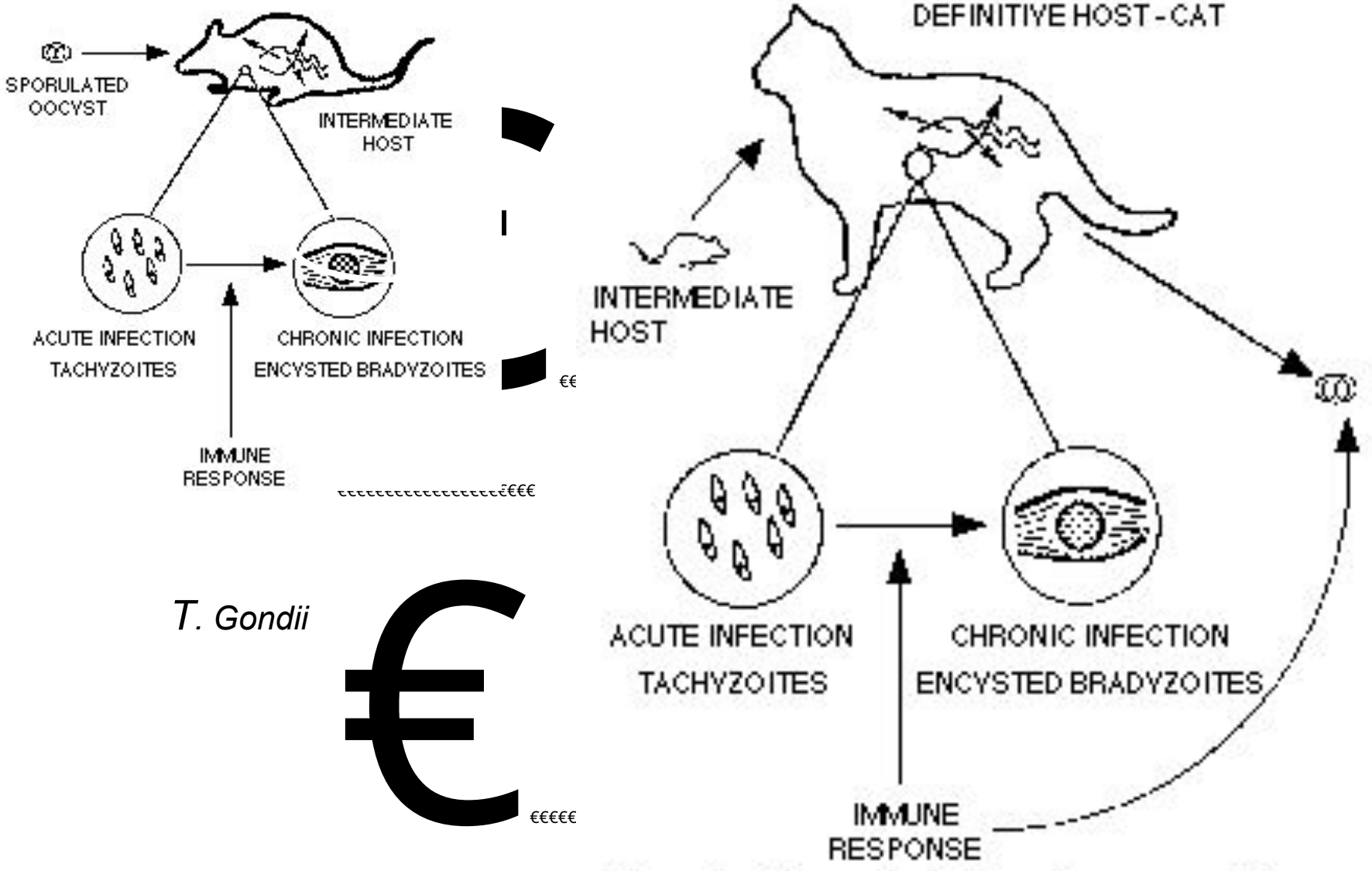
- Older age
- Exposure to soil-borne oocytes
- non-USA born individuals
- Certain minorities and socially disadvantaged individuals (males > females)

T. Gondii

(multiple pathway risks)



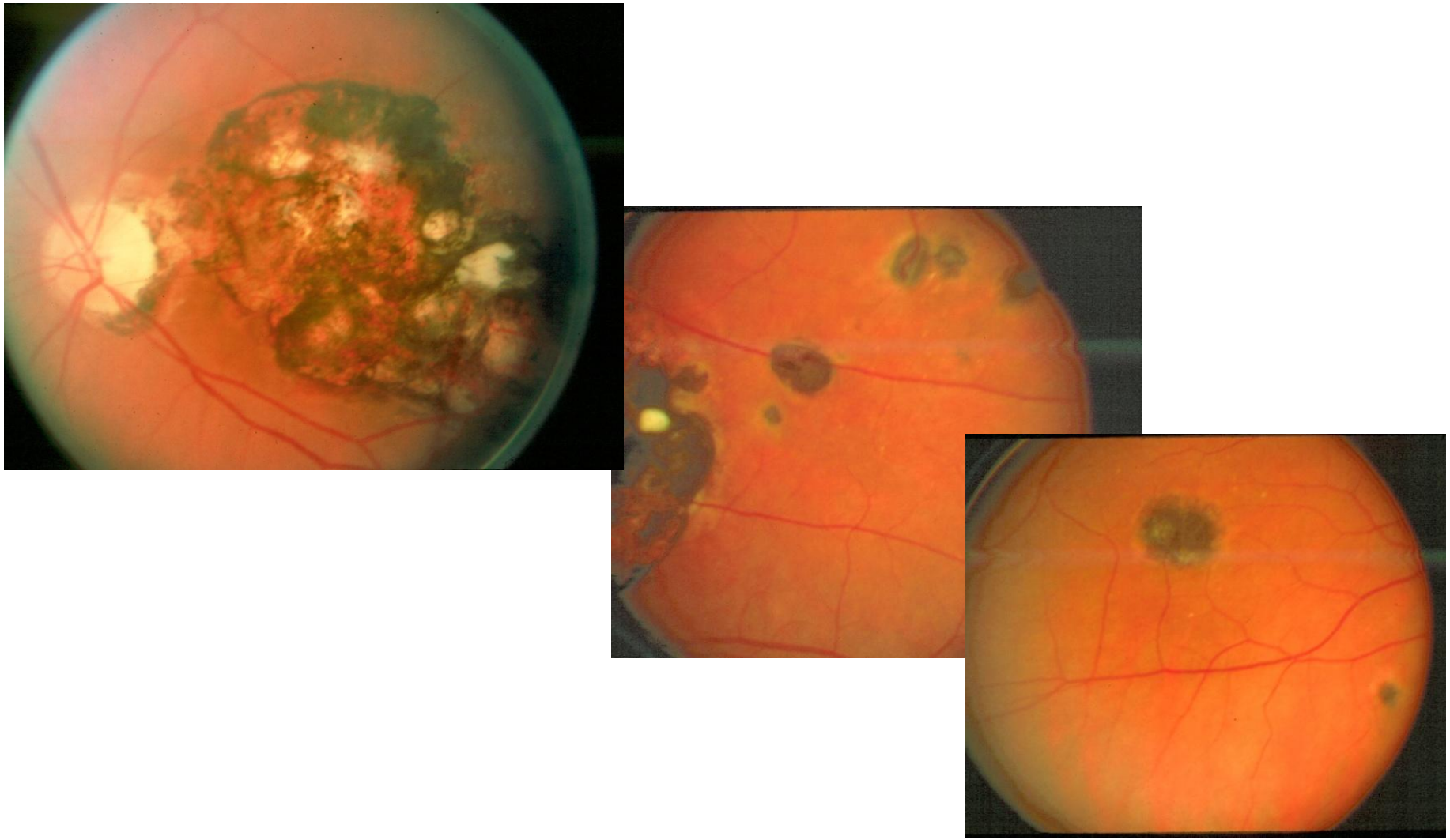
http://www.dpd.cdc.gov/dpdx/HTML/Toxoplasmosis.asp?body



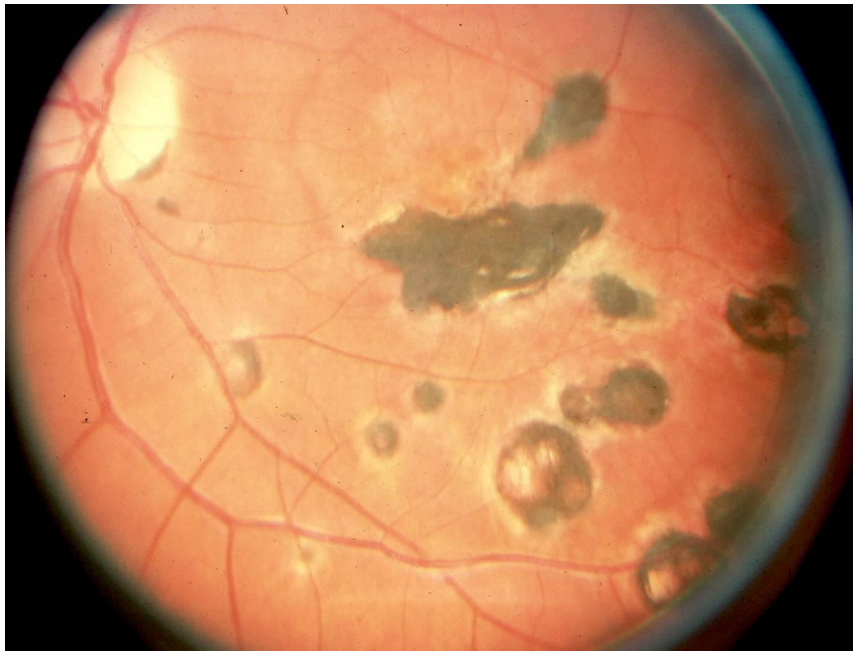
T. Gondii

http://www.fabcats.org/toxoplasmosis.html Feline advisory board

Post-inflammatory lesions



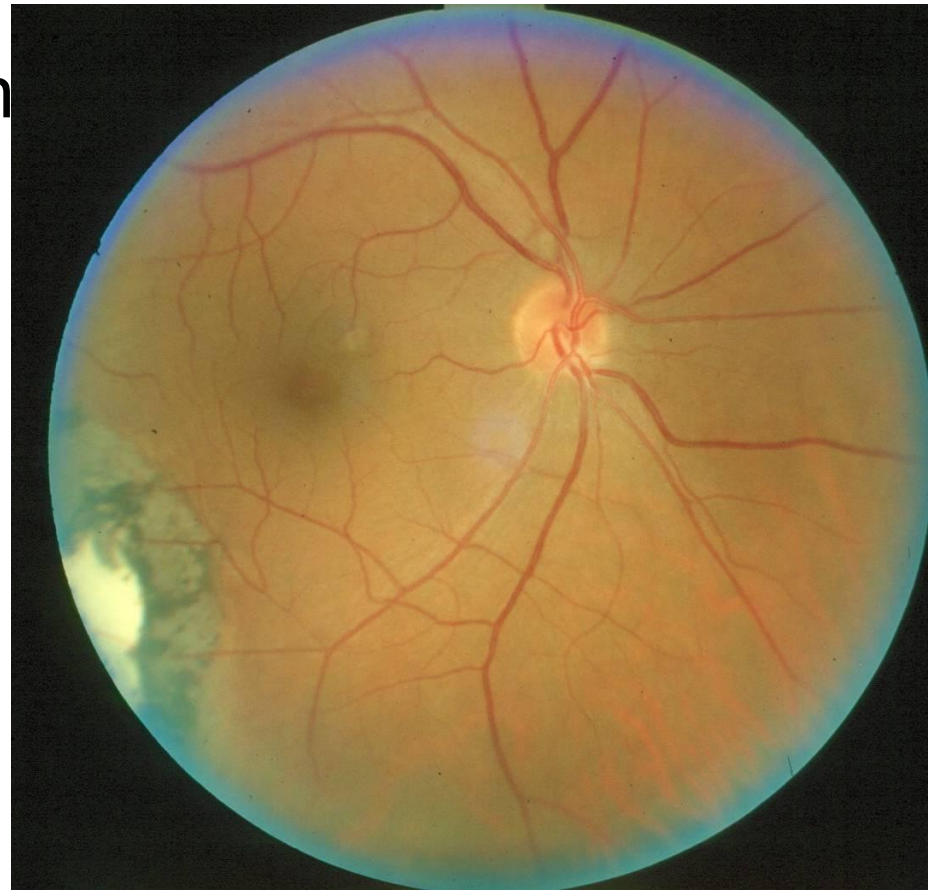
Post-inflammatory lesions (“congenital toxo”)



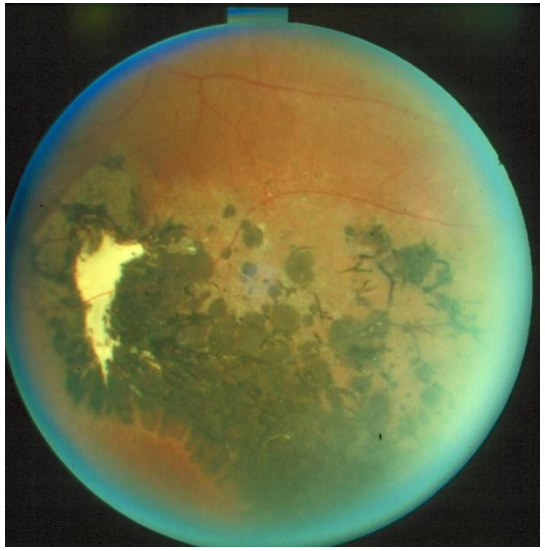
Congenital is generally bilateral, and involves posterior pole
Reactivation is not unexpected…

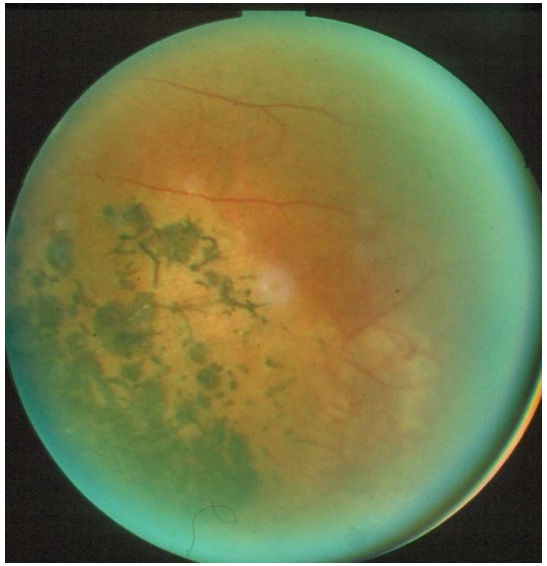
Inactive retinochoroiditis

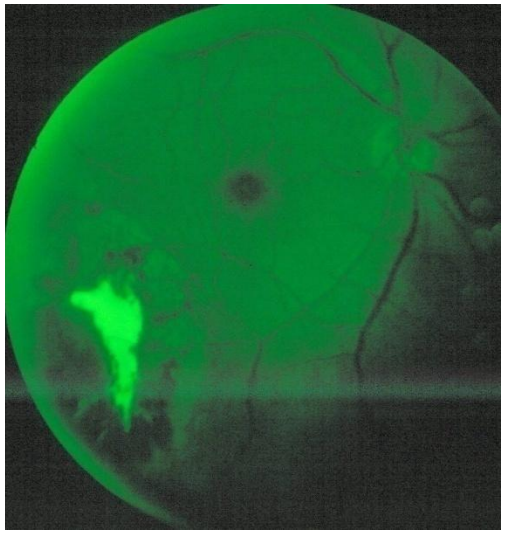
- 58 B/M presents with near complaints
  - VA – 20/20



Inactive Retinochoroiditis

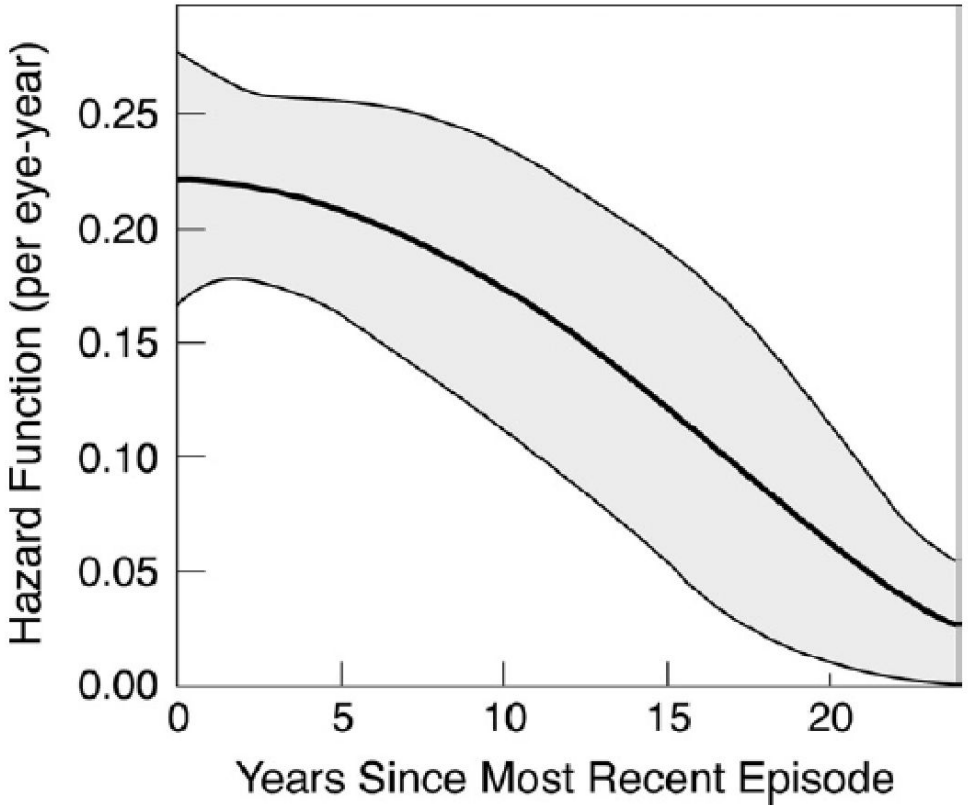






Inactive

Recurrences (risk)



- While risk appears to decline with increasing time from most recent episode, risk may be accelerated by increasing age (interactive factors).

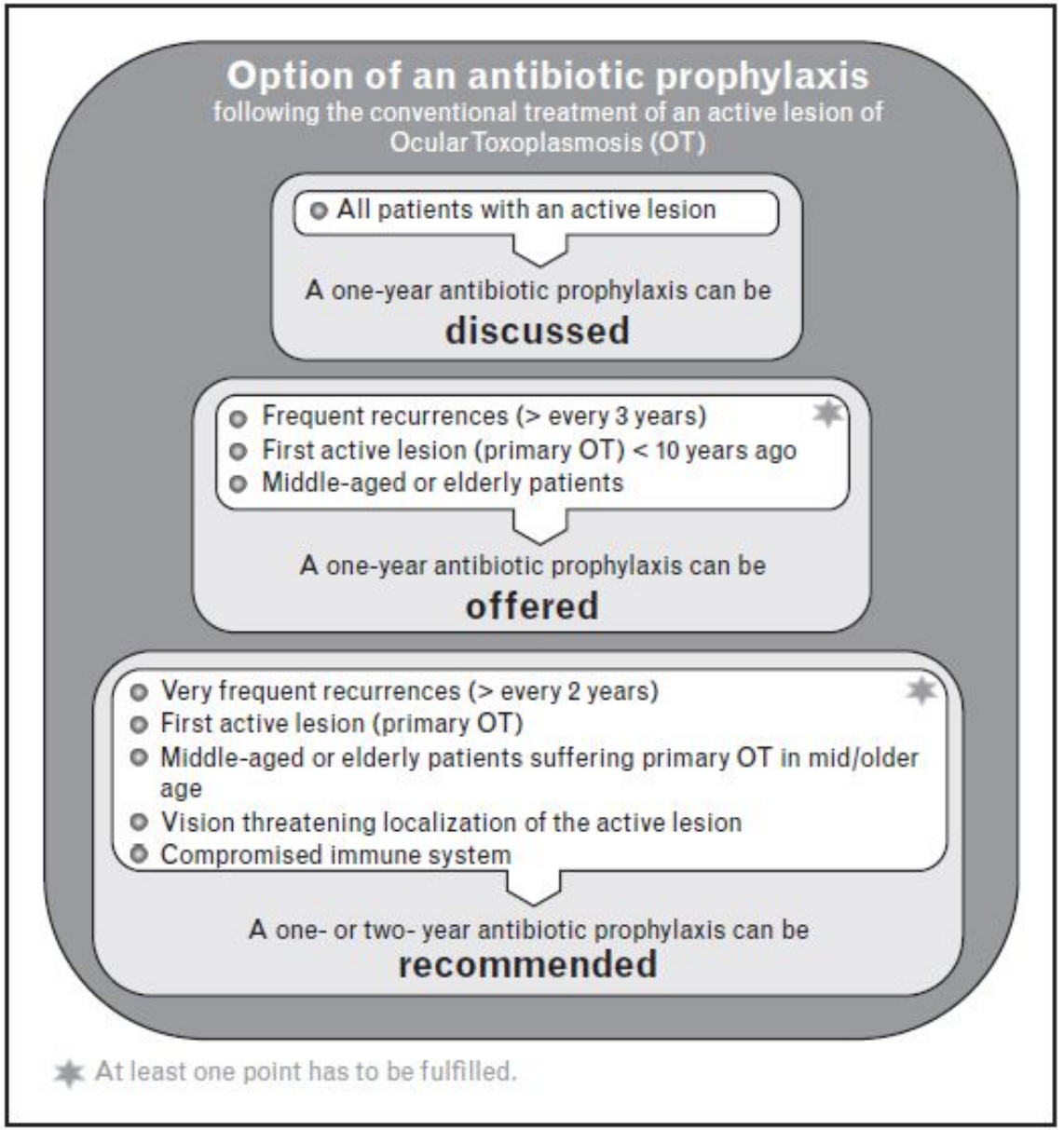
- Holland GN, et al., Analysis of recurrence patterns associated with toxoplasmic retinochoroiditis. Am J Ophthalmol. 2008 ;145(6):1007-1013.

Prophylaxis ?

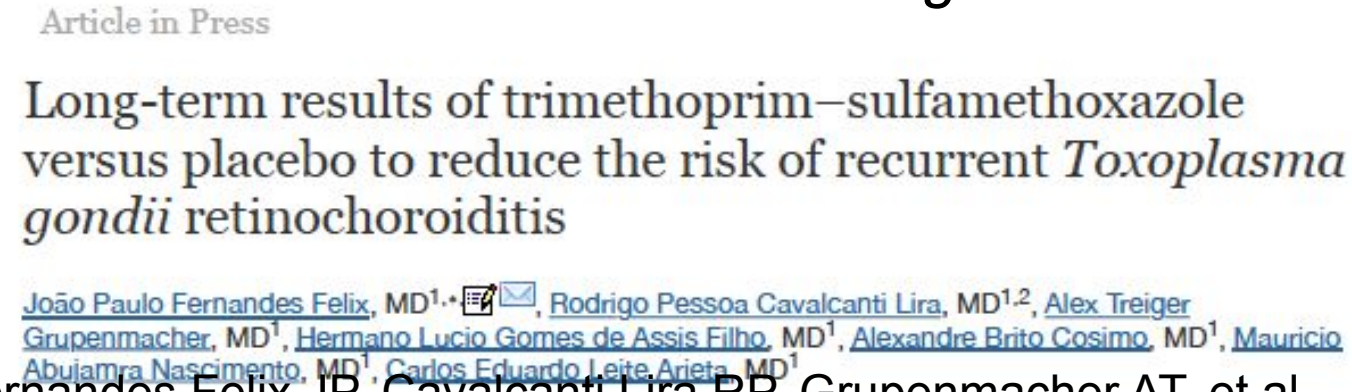
- Evolving guidance

Reich M, Mackensen F. Ocular toxoplasmosis: background and evidence for antibiotic prophylaxis. Curr Opin Ophthalmol 2015; 26 (Nov): 498-505.

Fernandes Felix JP, Cavalcanti Lira RP, Cosimo AB, Cardeal da Costa RL, Nascimento MA, Leite Arieta CE. Trimethoprim-Sulfamethoxazole Versus Placebo in Reducing the Risk of Toxoplasmic Retinochoroiditis Recurrences: A Three-Year Follow-up



Latest recommendation - endorsing Bactrim™



Fernandes Felix JP, Cavalcanti Lira RP, Grupenmacher AT, et al.. Long-term results of trimethoprim-sulfamethoxazole versus placebo to reduce the risk of recurrent Toxoplasma gondii retinochoroiditis. Am J Ophthalmol. 2020 Jan 9.

Results / clinical guidance

Cumulative recurrence rates

- Placebo vs. TMP-SMZ

Years		Rate (%)	
1	13	0	
2	17.4		0
3	20.3		0
4	23.2		0
5	26.1		0
6	27.5		1.4

3 cases of multiple recurrences occurred in the same individual in the placebo group

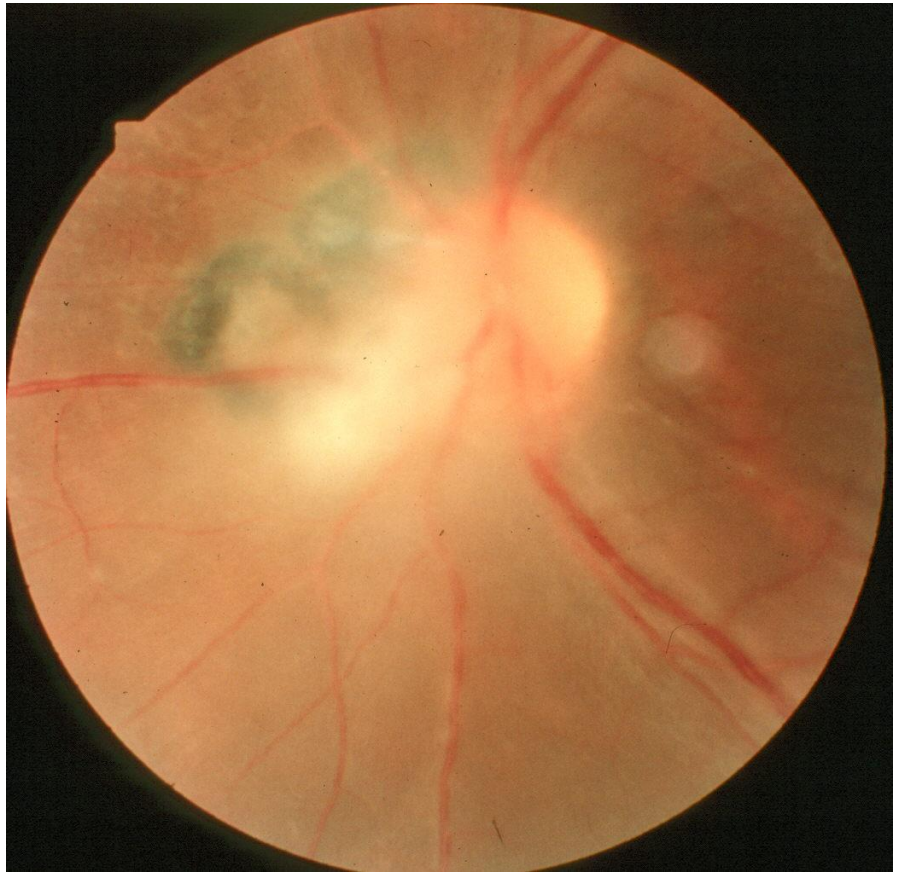
No treatment-limiting toxicity or SEs were observed in either group

Recurrence frequency: ♂ > ♀

Fernandes Felix JP, Cavalcanti Lira RP, Grupenmacher AT, et al.. Long-term results of trimethoprim-sulfamethoxazole versus placebo to reduce the risk of recurrent Toxoplasma gondii retinochoroiditis. Am J Ophthalmol. 2020 Jan 9

QUIZ TIME

- 16 W/M
- Slight decrease in VA recently
- VA = 20/40 (OS), DX = ?
- DDx = ?



Active lesions are white and fluffy with vitritis.
(a controversial indication to all oral prednisone)

Ocular Toxoplasmosis

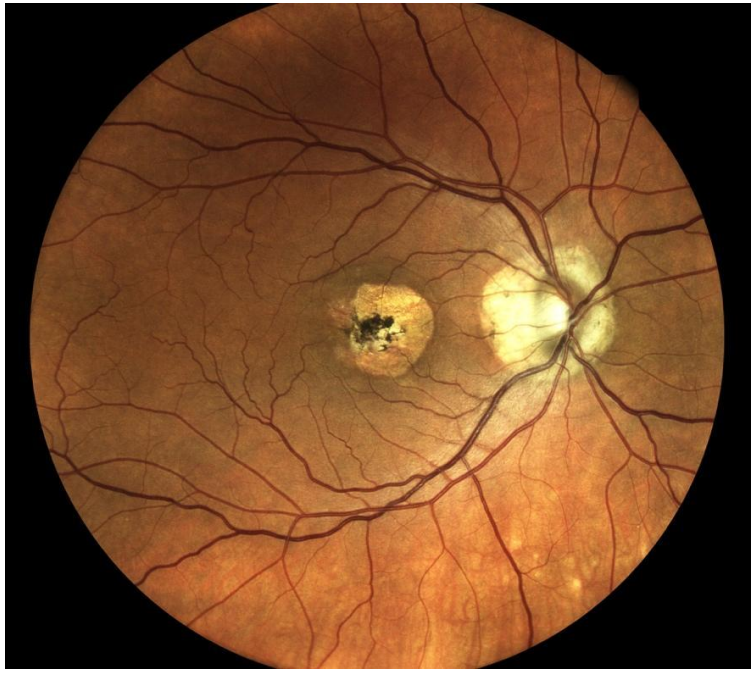
- Questions
- Comments

65 yo white male

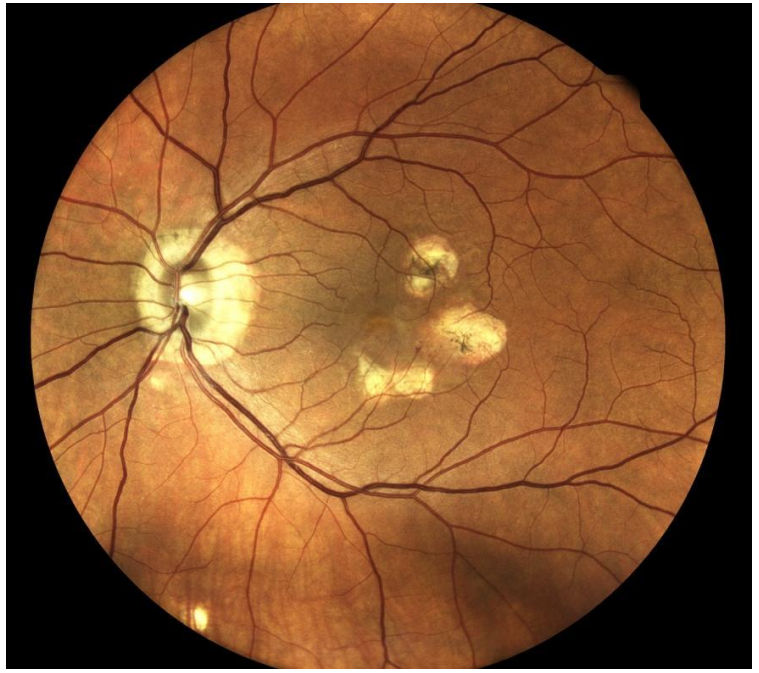
- h/o OHS for many years
  - Laser OD 20 + years ago with scarring and poor acuity ≈CF
  - CNVM OS x 10 years, with 3 injections, VA stable since, ≈20/25
- In for 9 month follow up. Reports stable vision with no active issues
- CF OD
- 20/25 OS

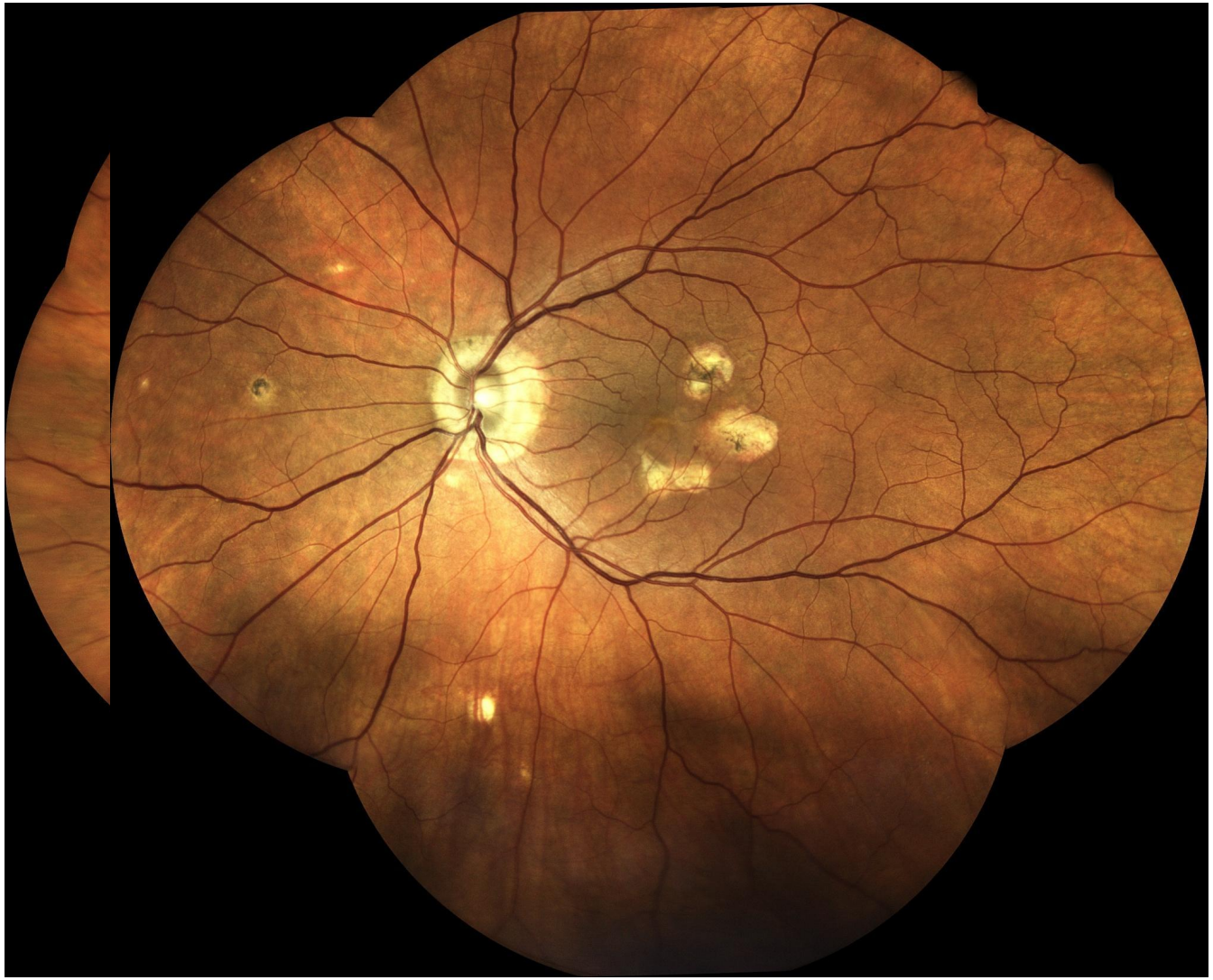
OHS

CF

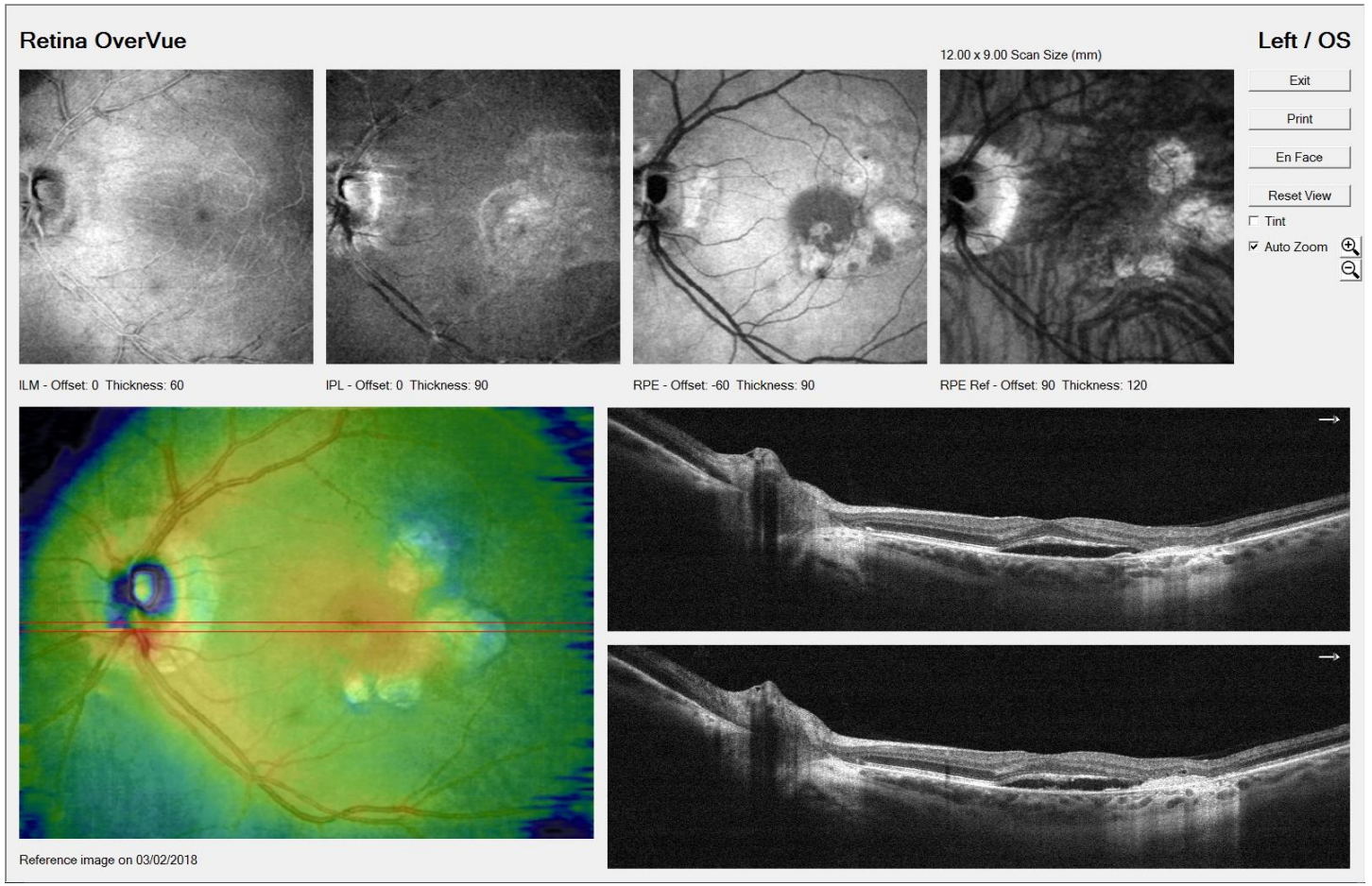


20/25





WIDEFIELD


Retina OverVue
Left / OS
12.00 x 9.00 Scan Size (mm)
Exit
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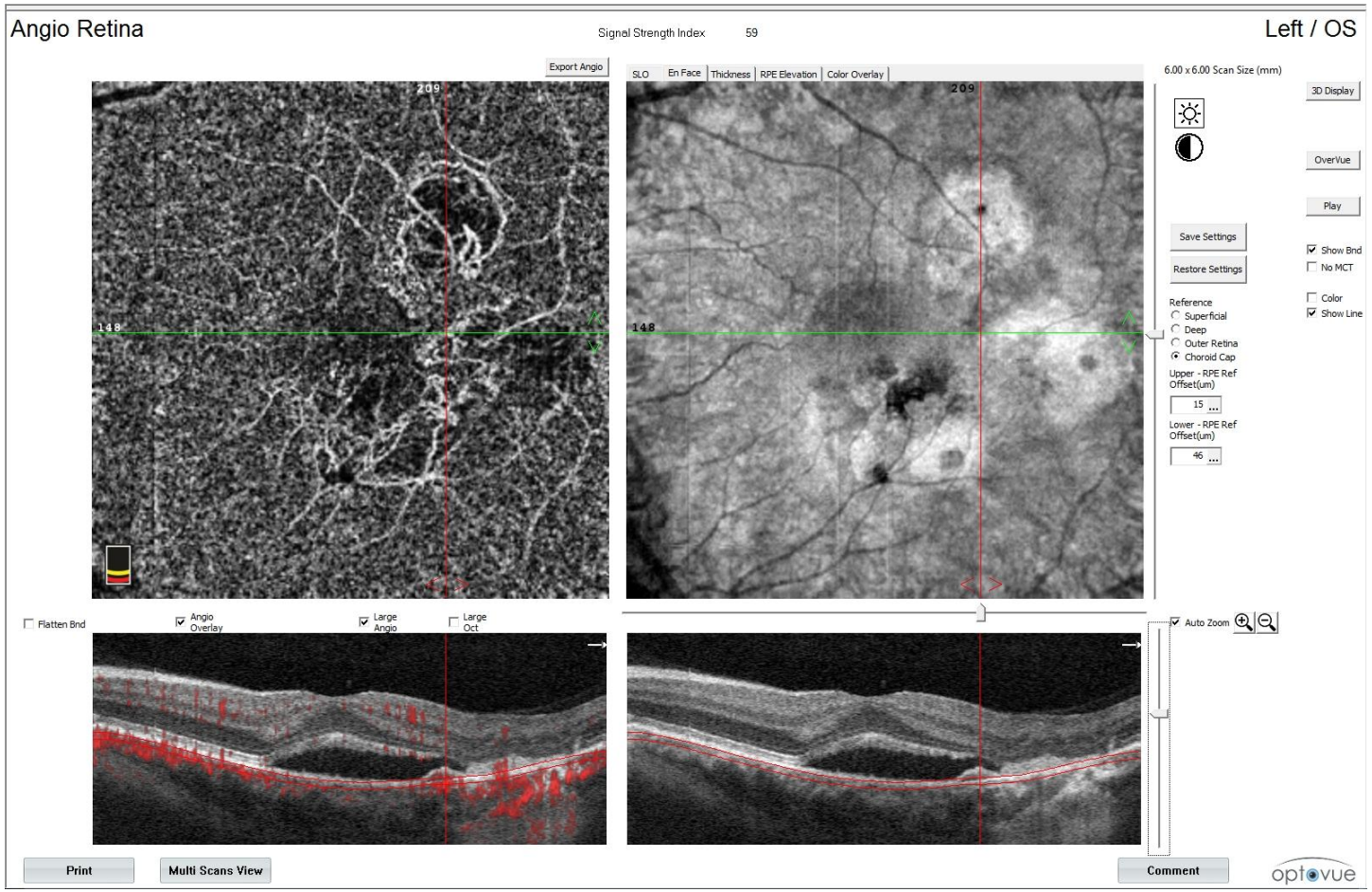
ILM - Offset: 0 Thickness: 60
IPL - Offset: 0 Thickness: 90
RPE - Offset: -60 Thickness: 90
RPE Ref - Offset: 90 Thickness: 120
Reference image on 03/02/2018


Angio Retina
Signal Strength Index 59
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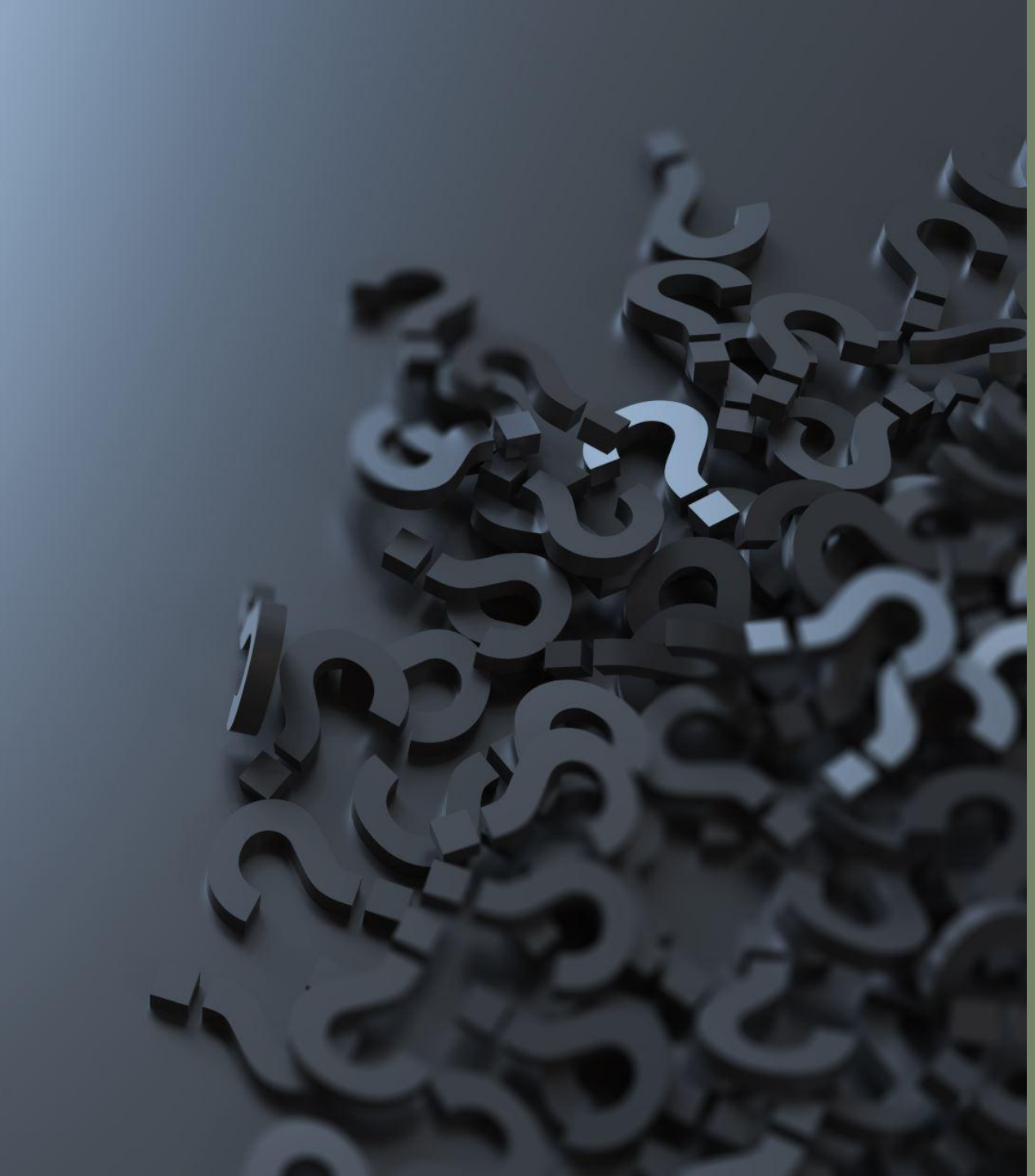
Color Overlay
209
148
6.00 x 6.00 Scan Size (mm)
3D Display
OverVue
Play
Save Settings
Restore Settings
Show Bnd
No MCT
Color
Show Line
Reference
Superficial
Deep
Outer Retina
Choroid Cap
Upper - RPE Ref Offset(um)
15
Lower - RPE Ref Offset(um)
46
Flatten Bnd
Angio Overlay
Large Angio
Large Oct
Auto Zoom
Print
Multi Scans View
Comment
optovue

65 yo white male

- ASSESSMENT
  - OHS with reactivation CNVM OS
- PLAN
  - Avastin x 3
  - RTC for repeat OCT, OCTA after 3rd injection
  - Pt agrees with plan
  - Monoc precautions including Polycarbonate lenses

All you need to know about differentiating ocular histo from toxo

- Histoplasmosis
  - Fungal organism
  - Vector is fowl guano with geographic predispositions
  - Affects Lungs OR Eyes
  - Triad of ocular signs
    - perihpheral "histo spots"
    - parapapillary atrophy
    - macular involvement
  - Histologically is a primary choroiditis
  - Ocular involvement - treat locally
- Toxoplasmosis
  - Obligate intracellular parasite
  - Vectors are multi (but NOT fowl) with regional predictions
  - Systemic involvement
  - Ocular signs
    - peripheral scarring
    - REACTIVATION
  - Histologically is a primary retinitis
  - Ocular involvement – treat systemically



Histo *vs.* Toxo

Questions

Comments

28 W/M

- History / RFV
  - Healthy Dental Student II
  - 2-day observation of “floater” OD only

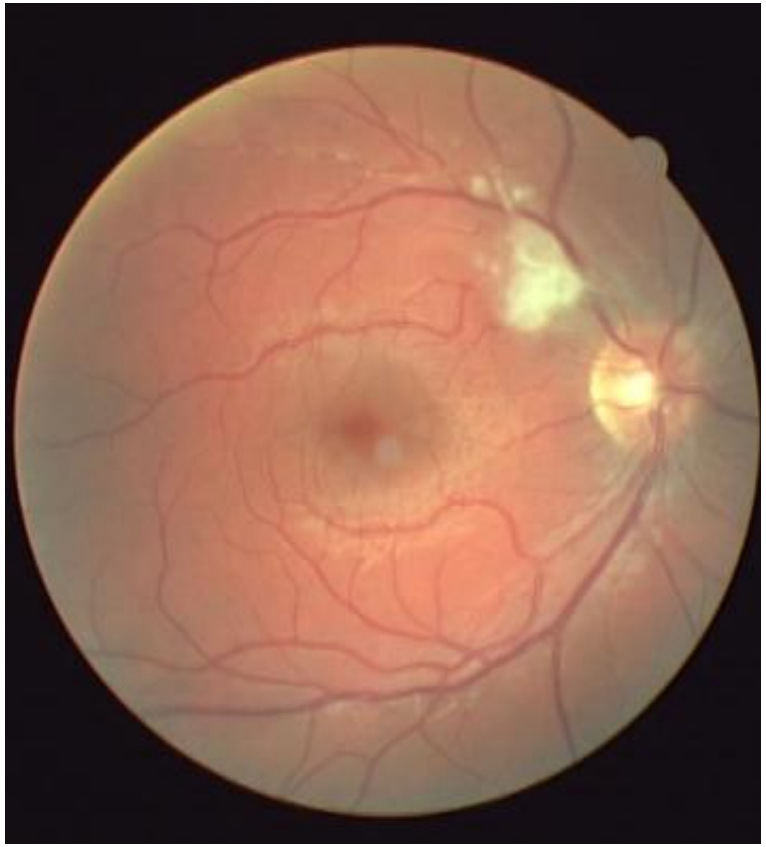
  - No current medications/allergies
  - No chronic or acute medical problems

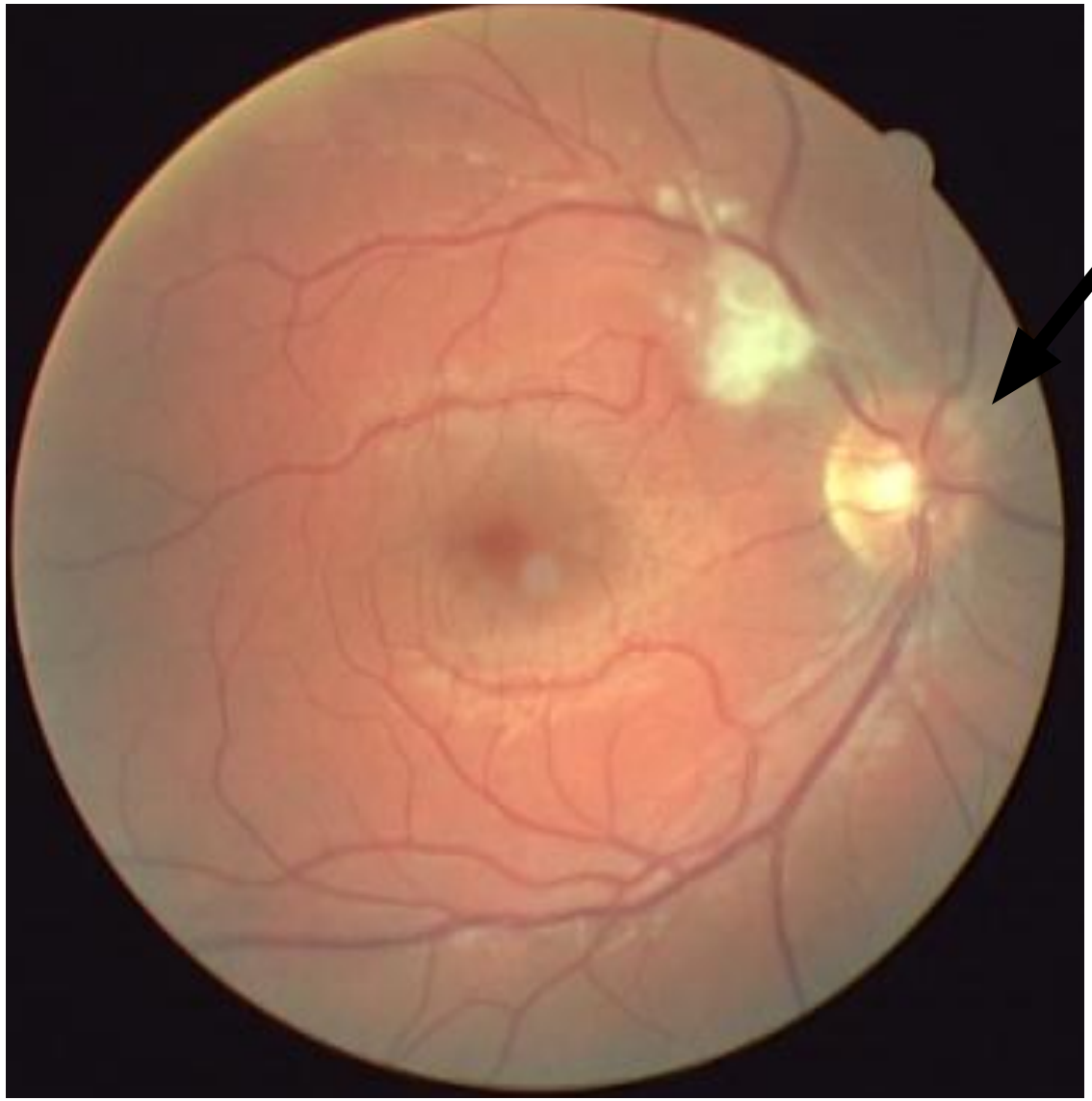
28 W/M

- Findings
  - UCVA 20/20 in each eye
  - Normal motility
  - VF - FFCF
  - PERLA (-) APD
  - Anterior segment unremarkable OD/OS
  - T_A 14/14 mm Hg (OD/OS)

28 W/M

- Findings (con't) DFE
  - 1-2^{+} Vitritis (OD)
  - Granuloma




Note disc
swelling

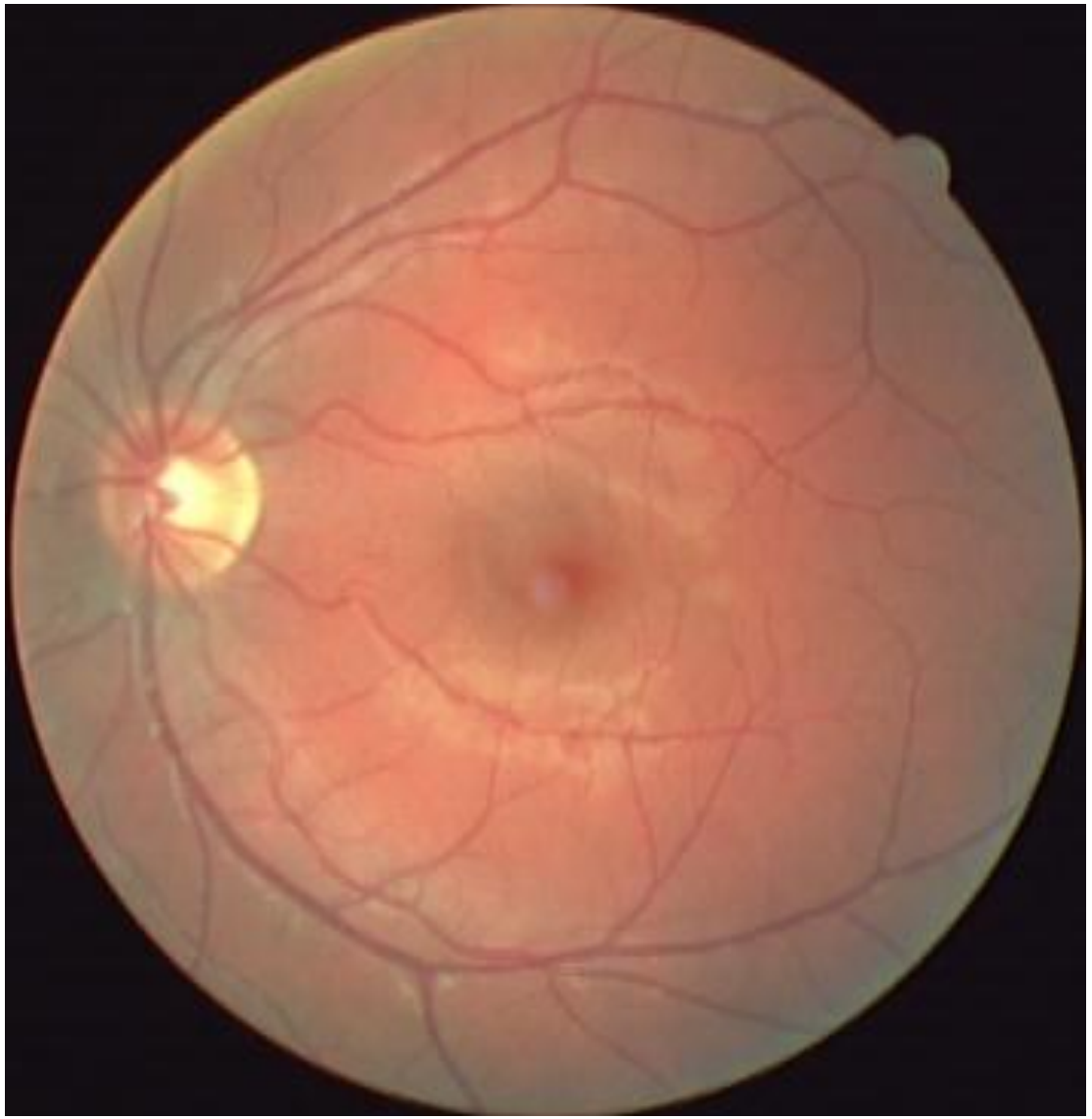
28 W/M

- What are some differential diagnoses?
  - Toxoplasmosis
  - Histoplasmosis
  - Toxocariasis
  - Cat-scratch neuroretinitis

28 W/M

- DFE (OD)
  - Granuloma +
  - Elevated nasal disc margin +
  - parapapillary retinal edema

    = Neuroretinitis !



Uninvolved Fellow eye

28 W/M

- Cat-scratch neuroretinitis
  - Granuloma @ optic disc (OD)
  - Slight optic nerve head edema with spread to retina on nasal side (OD)
  - *History of new kittens with scratch (still healing) on back of left hand*

28 W/M

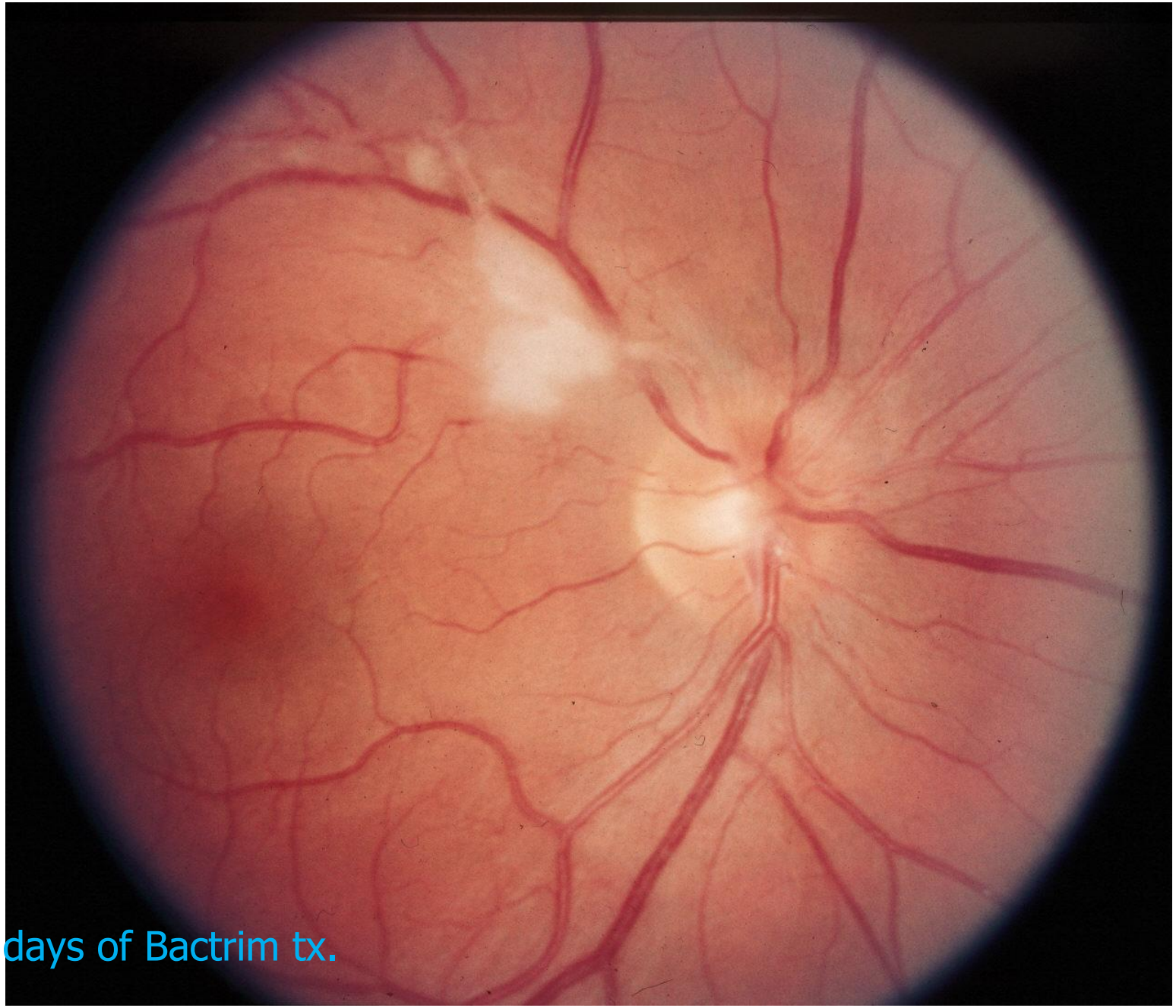
- Treatment
  - Bactrim DS
  - 1 tab, PO, bid X 4 wks.

28 W/M

Discussion

- Optic nerve swelling as an early sign in cat-scratch disease

Wade NK, Levi L, Jones MR, et al. Optic disk edema associated with peripapillary serous retinal detachment: an early sign of systemic *Bartonella henselae* infection. Am J Ophthalmol 2000*;* 130: 327-334.



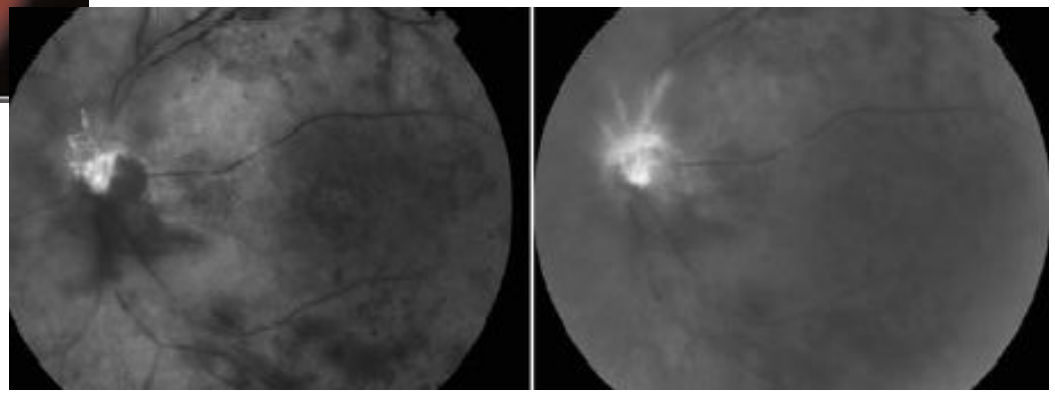
S/P 2 days of Bactrim tx.

Case Example 28 W/M

- Cat-scratch neuroretinitis
  - Treatment with Bactrim DS [sulfmethoxazole + trimethoprim] bid X 2 weeks and re-check
  - Serology to confirm diagnosis
  - Resolution is sometimes spontaneous without ocular consequences

Neuroretinitis with CRAO, CRVO, (& NVG)

Arterial attenuation, sclerosis, disc edema, pale retina, resolving macular exudates



FA @ 49 sec fails to reveal retinal perfusion

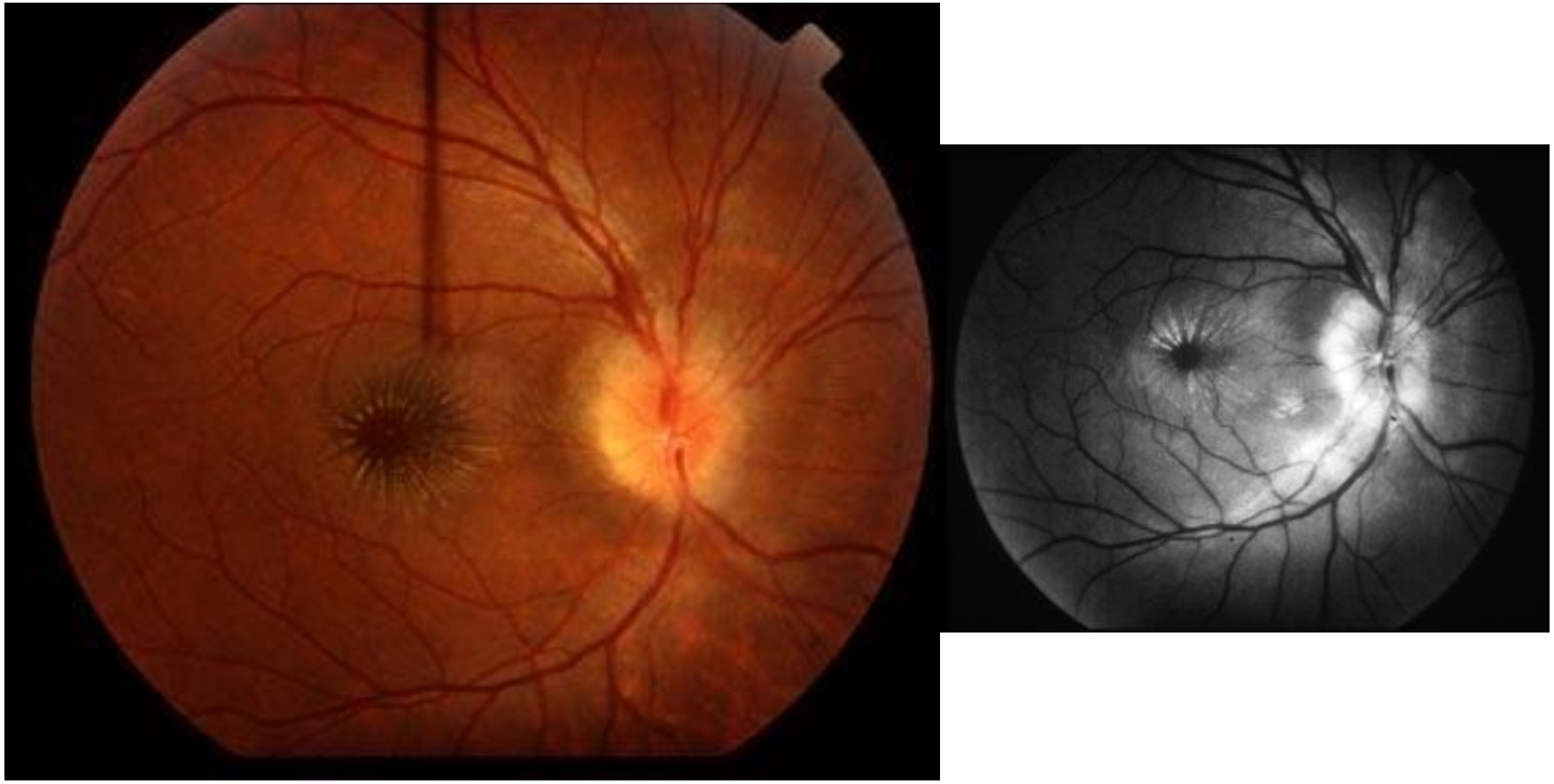
Neuroretinitis with CRAO, CRVO, & NVG

@ 1 month

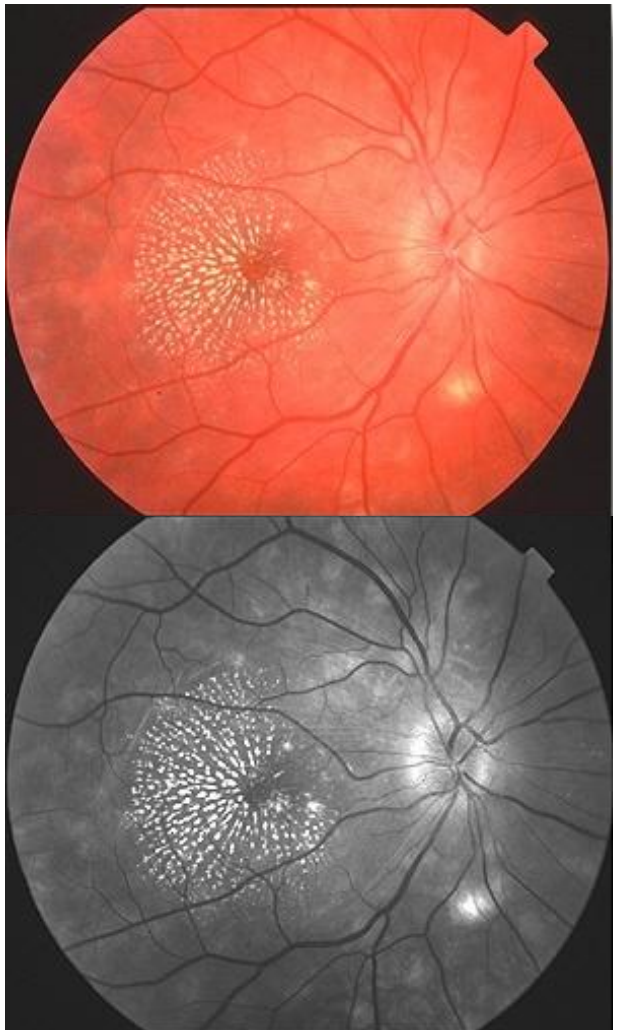
- Disc edema
- Dilated tortuous veins
- Thin arteries
- Intraretinal hemorrhages
- Retinal pallor

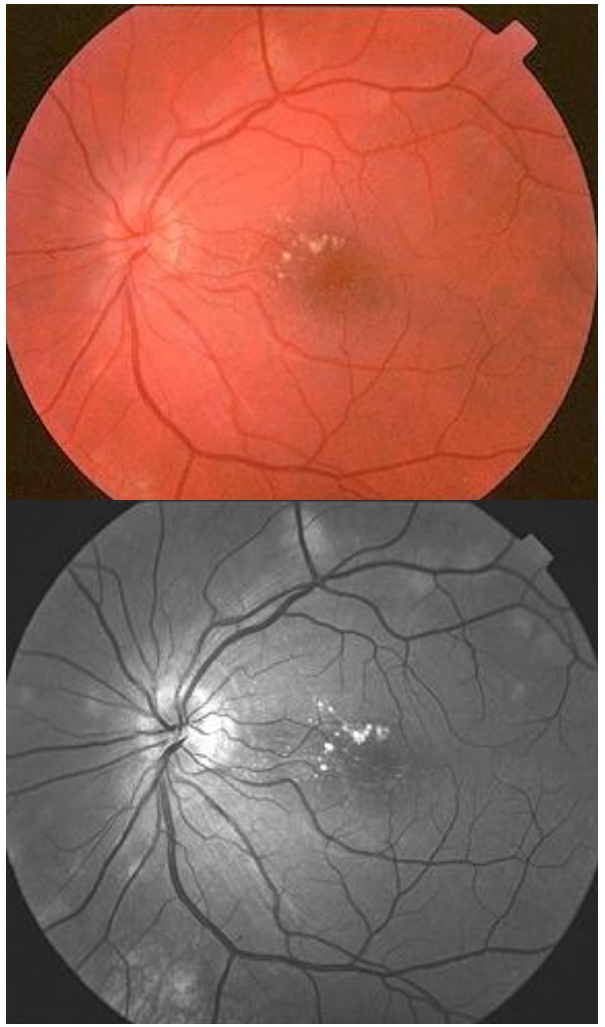
Neuroretinitis (optic disc edema with macular star, ODEMS)

…



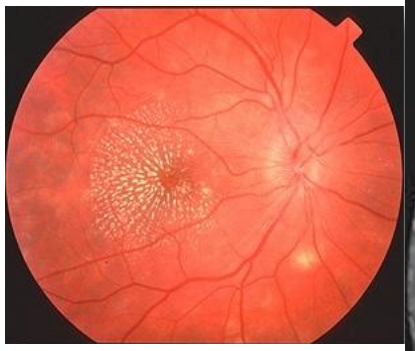
ODEMS (May be bilateral)

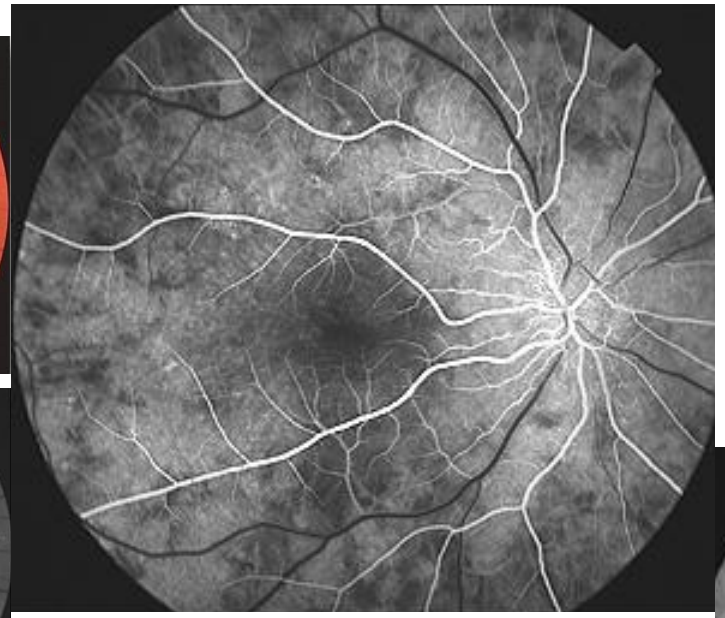




Note choroidal lesions on the RF photo

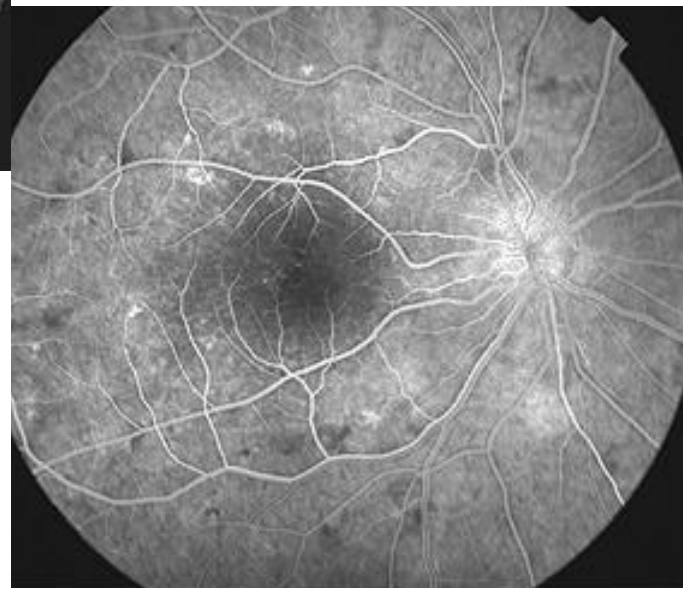
ODEMS: FA



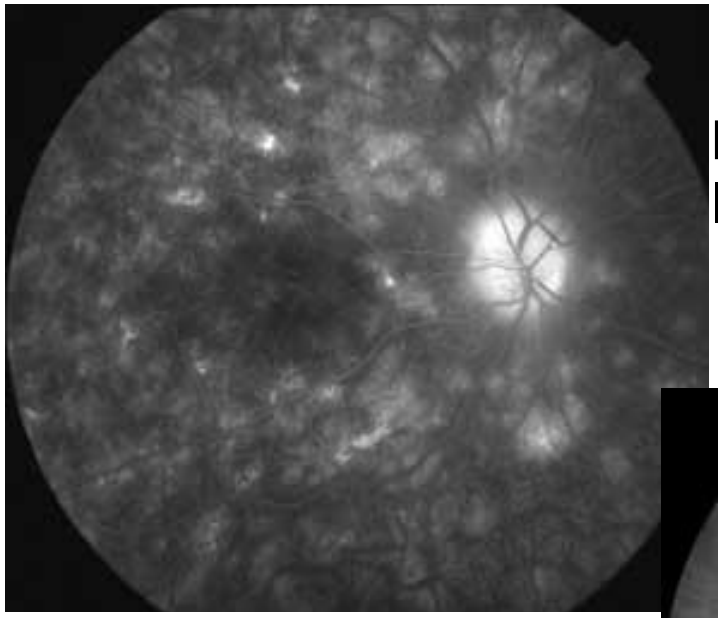


Early: Retinal arterioles filled; patchy choroidal filling

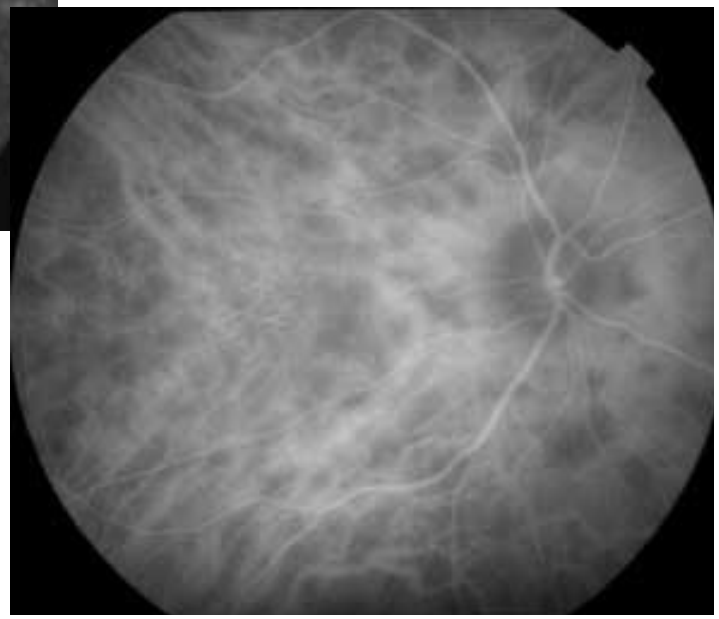
Mid: Dilated disc vessels and hypofluorescence in choroid



ODEMS: FA



Late: Patchy choroidal lesions



Mid phase ICG: many hypofluorescent choroidal lesions.

http://www.southeasterneyecenter.com/cases/catscratch.htm

The NEW ENGLAND JOURNAL of MEDICINE

IMAGES IN CLINICAL MEDICINE

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This article was published on March 6, 2021, at NEJM.org.

- 55 YOF presented with blurry VA and pain, OD, for several days (“CF”)
- + RAPD (OD)
- Anterior Segment inflammation
- Swollen ONH
- Macular star

- Positive *Bartonella* titer. elevated IgG
- Treatment:
  topical steroid drops
  oral doxycycline
  oral prednisone
- Outcome:
  Resolution of RAPD, macular edema
  VA recovered to 20/80

Bartonella Neuroretinitis

- Questions
- Comments

46 Asian Male

- “blurry vision”
- X 3 mo OS; began only last night OD
- Began new BP med last week
- Has never had eye exam
- Central blur in OS has improved somewhat
- + floaters X 1 yr
- - flashes, discharge, pain

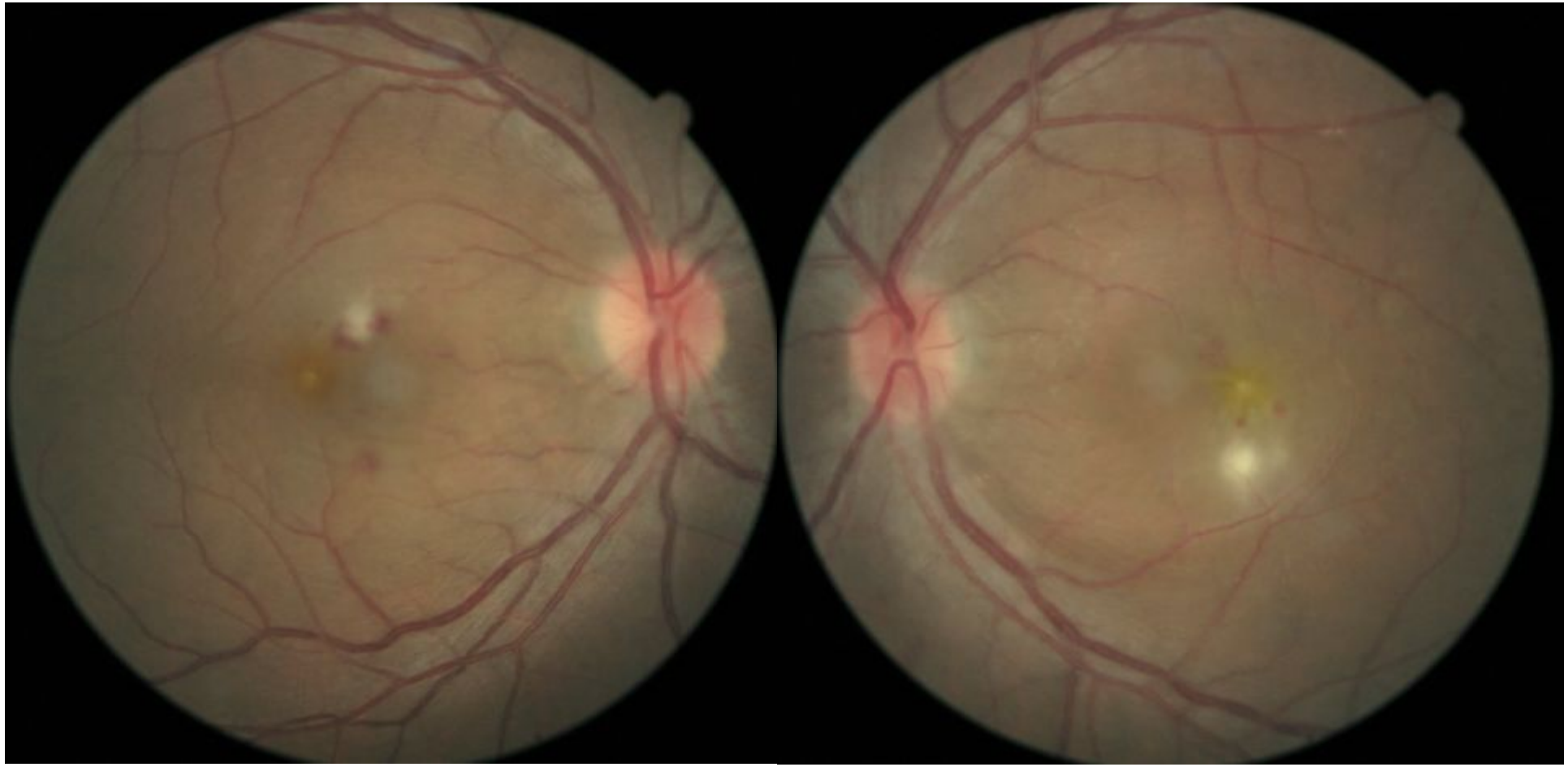
46 Asian Male

- Previous ocular history is negative for refractive correction, injury, glaucoma, cataract, strabismus, amblyopia, etc.
- Family medical / ocular histories negative
- No known allergies
- Began lisinopril qD X 1 wk. [ACE inhibitor]
- BP 150/100

46 Asian Male

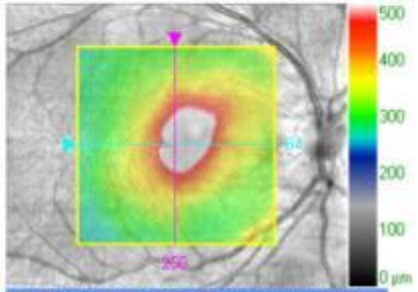
- VA 20/40- 20/400 (PHNI)
- -RAPD
- IOP: 14/14
- No EOM restrictions
- Confrontation FTFC OD, OS
- -1.50 / -2.25 -0.50 X 070 VA NI
- Anterior segment unremarkable OD, OS

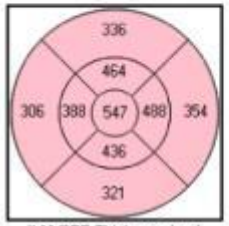
Baseline



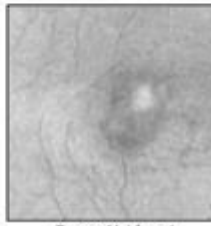
Doctor: Signal Strength: 9/10

Macula Thickness : Macular Cube 512x128 OD ● ○ OS

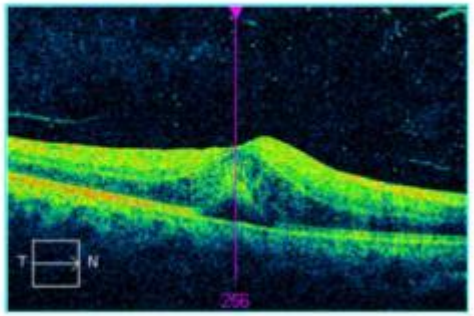


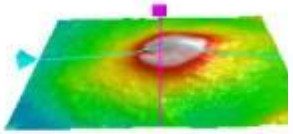


ILM-RPE Thickness (µm)

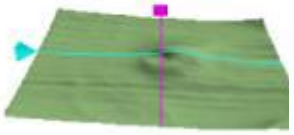


Fovea: Not found

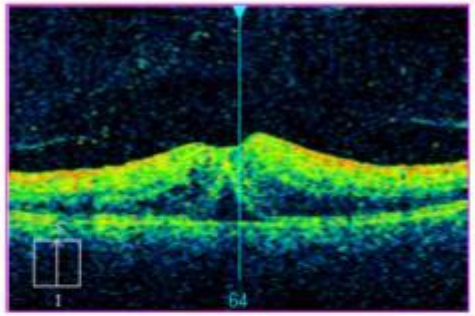


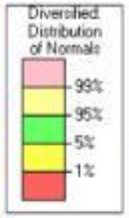


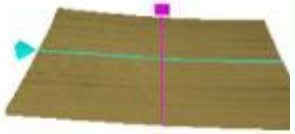
ILM - RPE



ILM





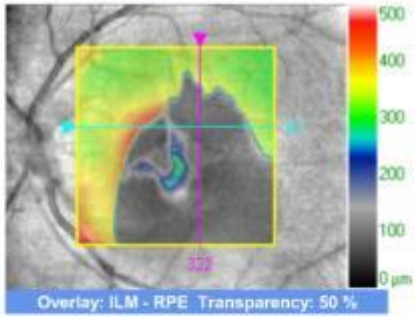


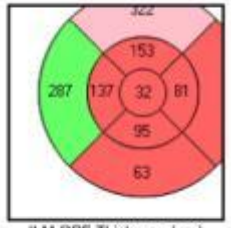
RPE

	Central Subfield Thickness (µm)	Cube Volume (mm³)	Cube Average Thickness (µm)
ILM - RPE	547	12.4	348

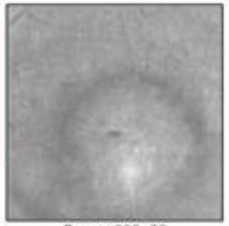
Doctor: Signal Strength: 7/10

Macula Thickness : Macular Cube 512x128 OD ○ ● OS

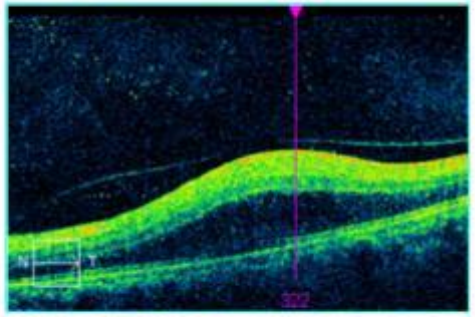


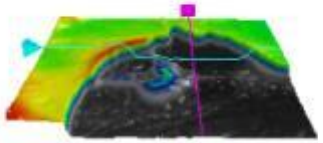


ILM-RPE Thickness (µm)

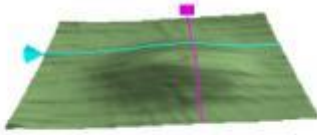


Fovea: 322, 52

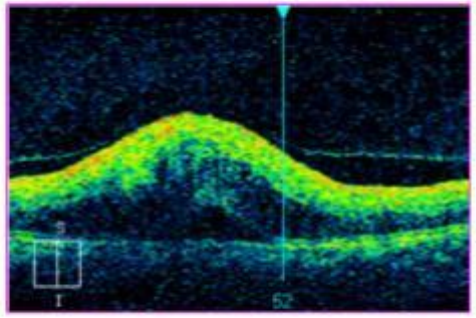


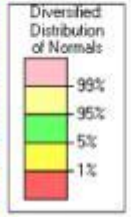


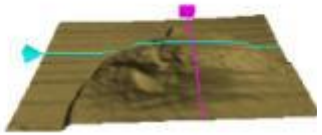
ILM - RPE



ILM

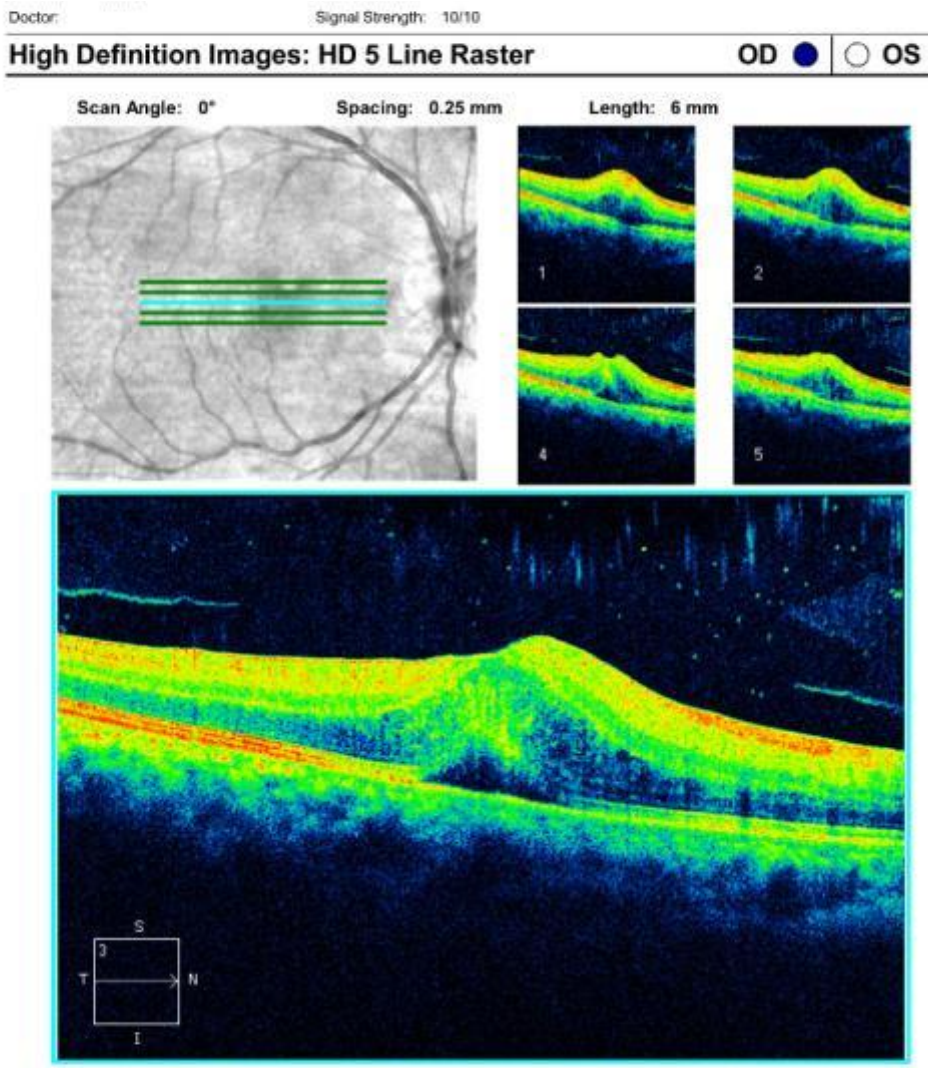


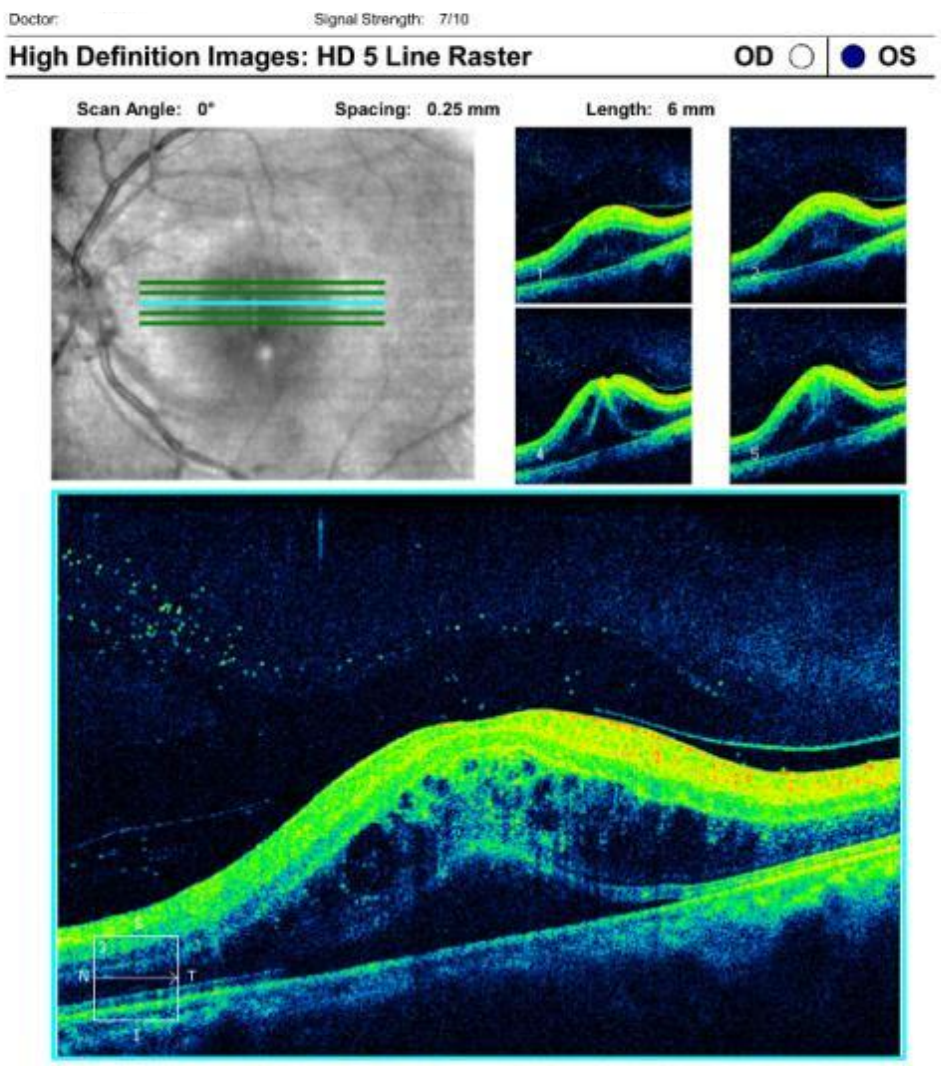




RPE

	Central Subfield Thickness (µm)	Cube Volume (mm³)	Cube Average Thickness (µm)
ILM - RPE	32	7.1	199

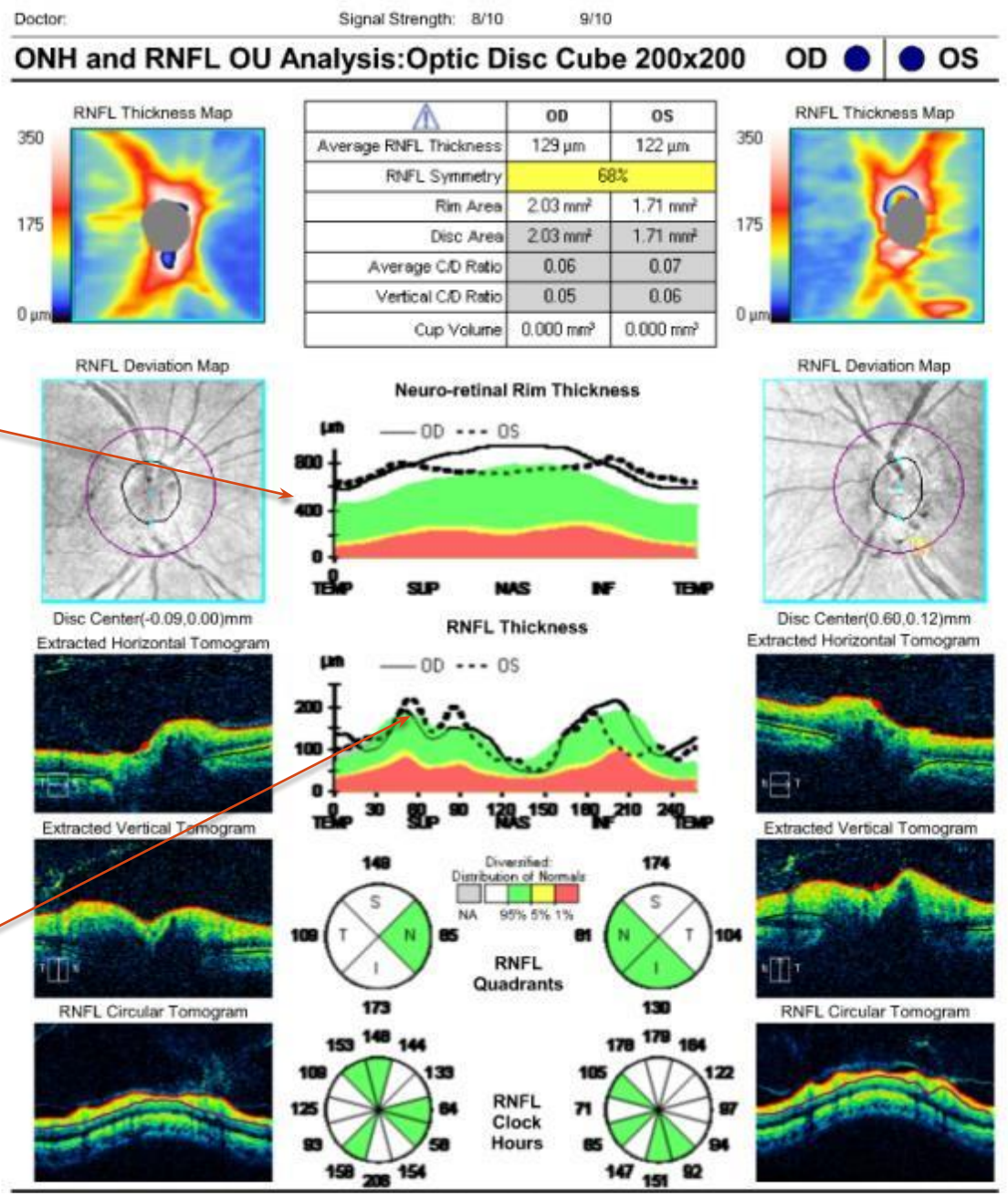




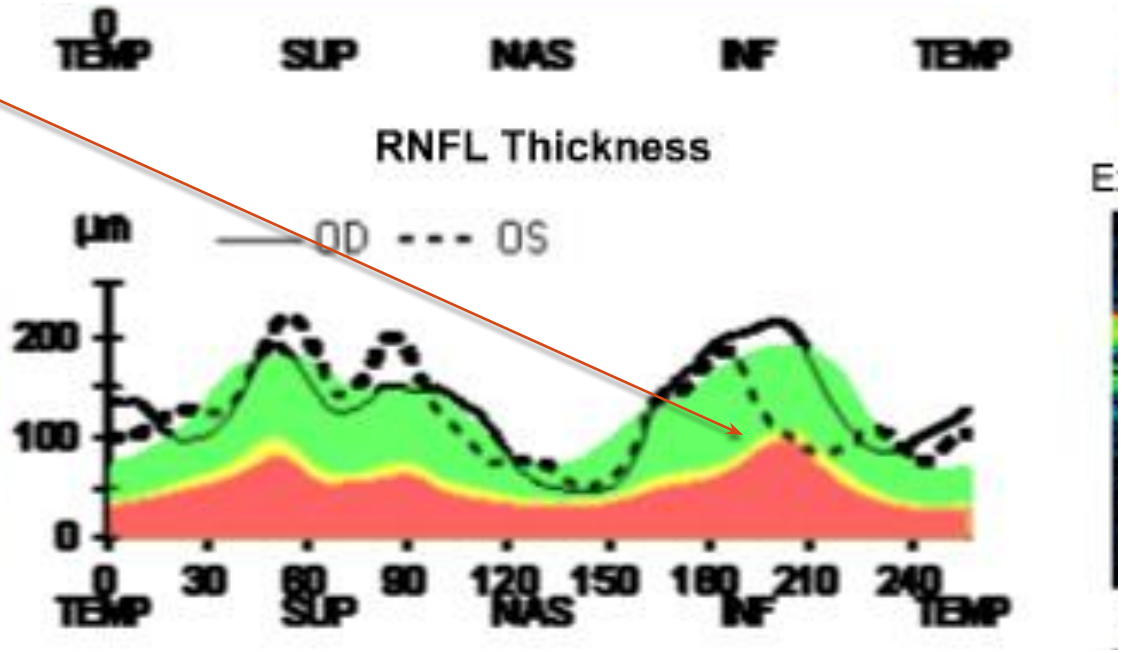
Note serous sub-retinal fluid and cystic macula

Note disc margin elevation

And CWS



And RNFL defect (OS)



46 A M with CSR, HR

- Initiated Nevanac bid
- RTC X 1 wk
- Correspond with PCP

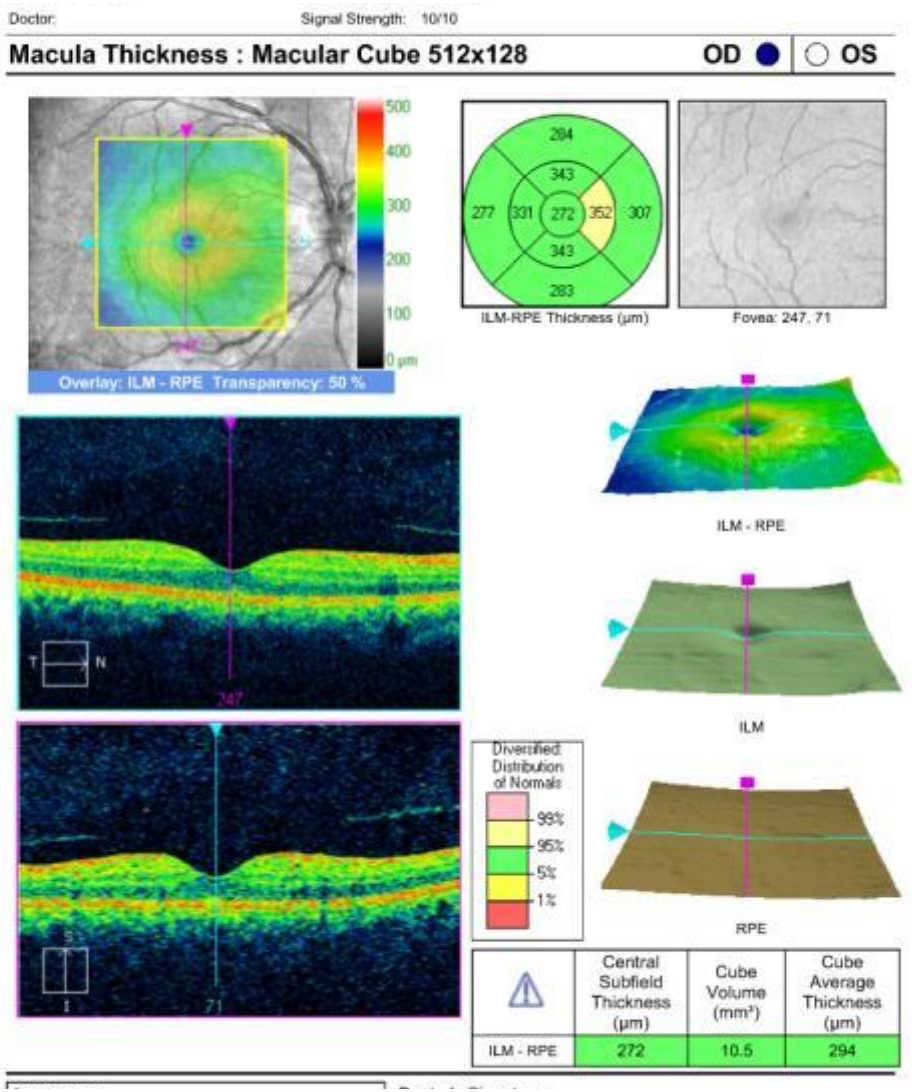
- @ 1- wk F/U
- BP = 138/92
- VA 20/25 , 20/40 !!!
  - (-1.00 / -0.75 – 0.50 X 070)
- Continue Nevanac bid

46 A M with CSR, HR

- Initiated Nevanac bid

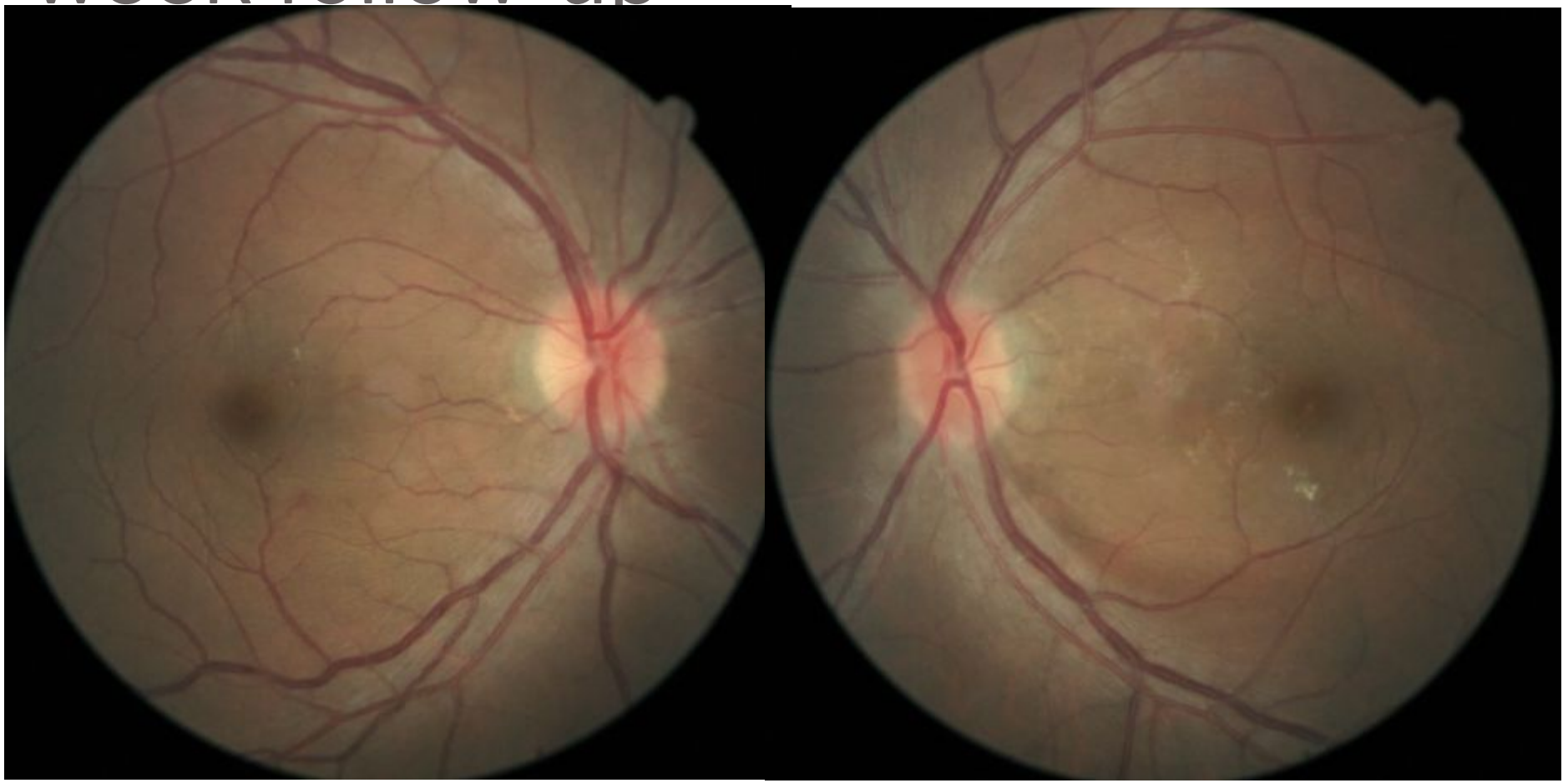
- @ 2- wk F/U
- BP = 140/92
- VA 20/20- , 20/20- !!!
  - (refraction unchanged)
- Continue Nevanac bid
- RTC X 1 Wk

3-week follow up



D/C Nevanac

3-week follow-up

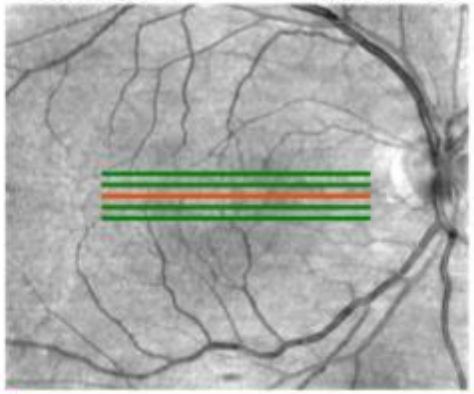


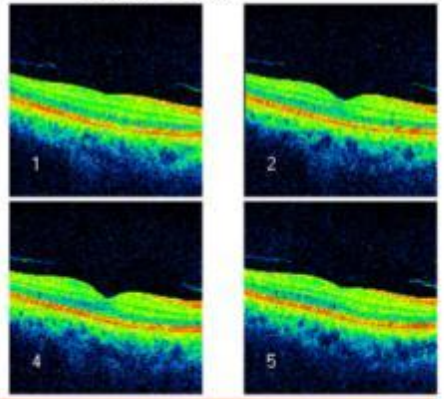
D/C Nevanac

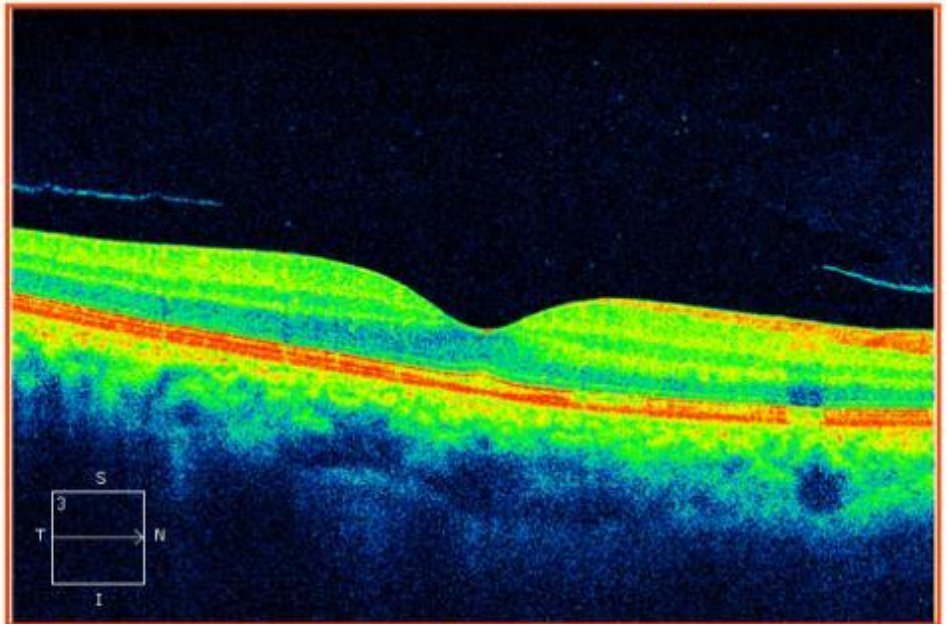
Doctor: Signal Strength: 10/10

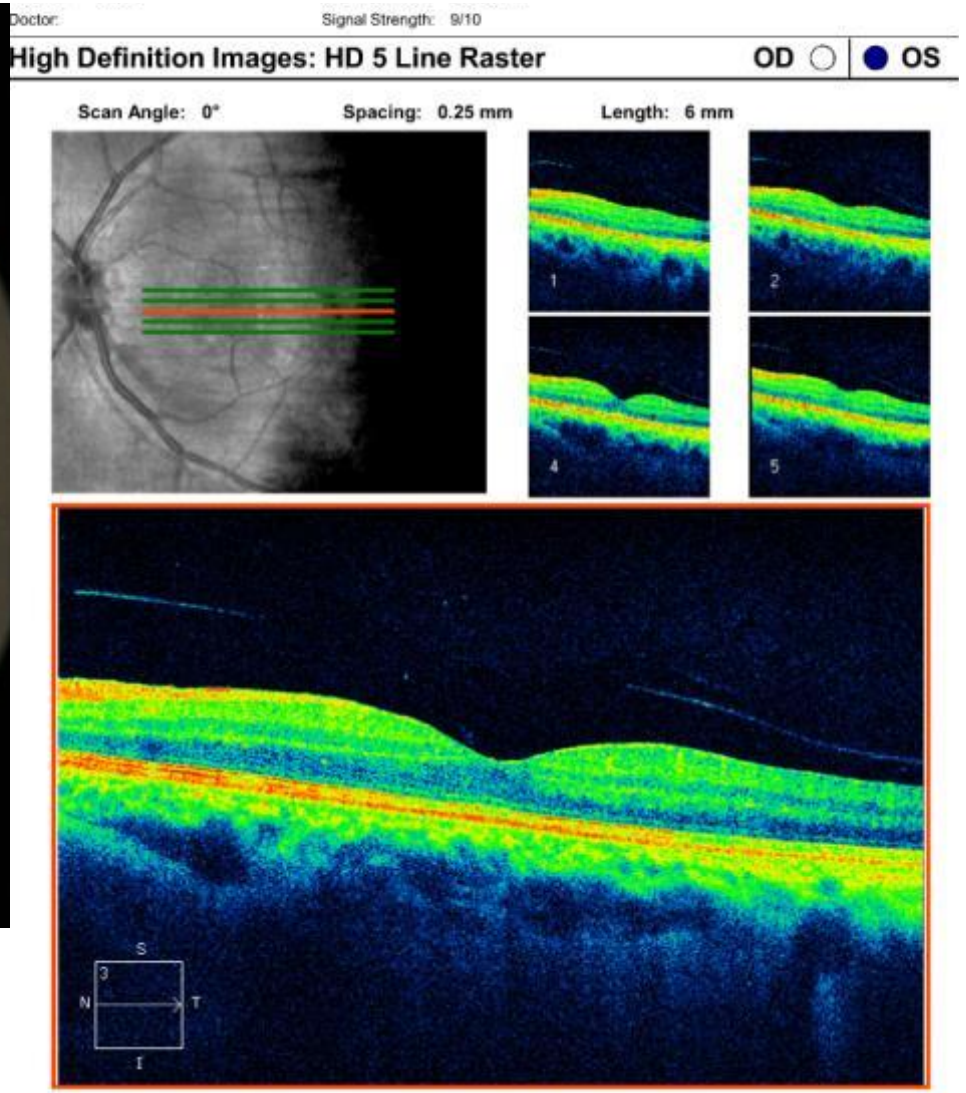
High Definition Images: HD 5 Line Raster OD ● ○ OS

Scan Angle: 0° Spacing: 0.25 mm Length: 6 mm








Doctor:
Signal Strength: 9/10
High Definition Images: HD 5 Line Raster
OD
OS
Scan Angle: 0°
Spacing: 0.25 mm
Length: 6 mm
1
2
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"…central serous is not an innocuous disease."

- R. Morris, MD (c. 1982)

("Retinologist")

JP

Recent developments in CSR

- One potential therapy applicable to primary eyecare is eplerenone, the oral medication that appears to be the most promising.

Fusi-Rubiano W, Saedon H, Patel V, Yang YC. Oral medications for central serous chorioretinopathy: a literature review. Eye (Lond). 2020 May;34(5):809-824. doi: 10.1038/s41433-019-0568-y. Epub 2019 Sep 16. PMID: 31527760; PMCID: PMC7182569.

- Another is half-fluence PDT or High-density subthreshold laser treatment.

van Dijk EHC, Fauser S, Breukink MB, et al. Half-Dose Photodynamic Therapy versus High-Density Subthreshold Micropulse Laser Treatment in Patients with Chronic Central Serous Chorioretinopathy: The PLACE Trial. Ophthalmology. 2018 Oct;125(10):1547-1555. doi: 10.1016/j.ophtha.2018.04.021. Epub 2018 Jun 14. PMID: 29776672.

CSR

- Questions
- Comments

Recent support for topical NSAIDs for acute CSR

Bahadorani S, Maclean K, Wannamaker K, Chu ER, Gresores N, Sohn JH, Diaz-Rohena R, Singer MA. Treatment of central serous chorioretinopathy with topical NSAIDs. Clin Ophthalmol. 2019 Aug 15;13:1543-1548. doi: 10.2147/OPTH.S202047.

CSR

- Questions
- Comments

two-minute drill

Homage to Dr. Kathleen Elliott

- PEPSI (T) mnemonic for Angioid streaks (AS)
- In hemoglobinopathies, iron deposition stiffens Bruch membrane
- CNVM standard treatments apply to NV in AS
- Retinal angiomatous proliferation (RAP) is a mimicker of AMD
- A variation of familial drusen originates from the Leventine valley of Switzerland near the Italian border
- Ocular toxoplasmosis responds to the combo antibiotic (Trimethoporim + sulfmethoxozole)

two-minute drill

Homage to Dr. Kathleen Elliott

- *Bartonella henselae* is the bacterium that causes "cat-scratch" **neuroretinitis**
- "Central serous retinopathy is not a benign disease" and is caused by choroidal hyperperfusion

Thank you

leopsemes@gmail.com