Things I thought I knew… About the retina

Brianne Hobbs, OD, FAAO

No financial disclosures

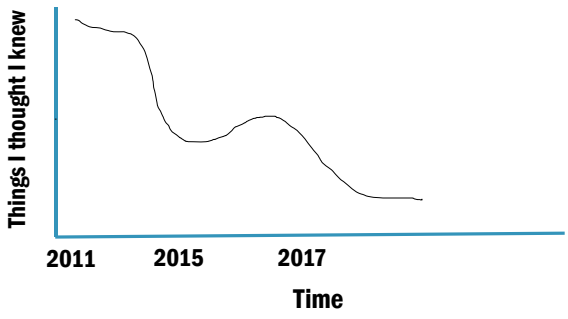
Purpose

Address misconceptions about the retina that may impact diagnosis or management of ocular disease

Motivation for this lecture

Optometry

Life



The dreaded disc diopter

5

Histoplasmosis

Central serous chorioretinopathy

Ocular ischemic syndrome

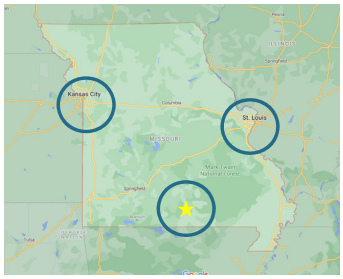
AMD

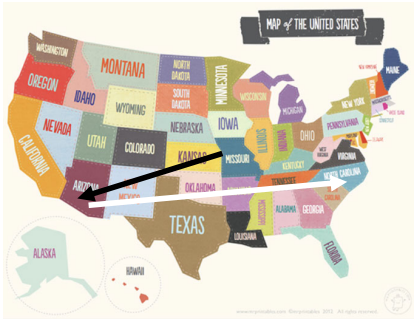
Choroidal neovascularization

Misconception #1

Histoplasmosis only occurs in the OH and MS river valleys

Why I care about this





Histoplasmosis only occurs in the OH and MS river valleys

MYTH

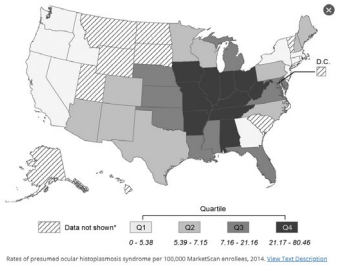
Presumed Ocular Histoplasmosis Syndrome

About

Presumed ocular histoplasmosis syndrome (POHS) is a condition that can cause vision loss. Many scientists believe POHS could be a long-term complication of histoplasmosis. However, no one has completely proven that *Histoplasma* (the fungus that causes histoplasmosis) causes POHS.[1] People can get histoplasmosis after breathing in the microscopic fungal spores from the air, but most people who breathe in the spores don't get sick. Scientists are still not sure exactly how *Histoplasma* spreads from a person's lungs to affect their eyes in POHS. Some people with signs of POHS in their eyes never have any symptoms, but other people can lose their vision because of it.

CDC Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™

Distribution of POHS

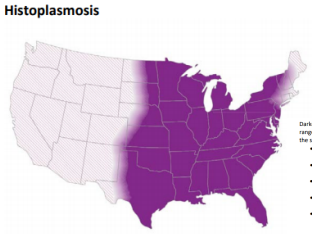


Rates of presumed ocular histoplasmosis syndrome per 100,000 MarketScan enrollees, 2014. View Text Description

Arizona	5.39 to 7.15
Missouri	21.17 to 80.46
Montana	Data not shown
Nebraska	7.16 to 21.16
Nevada	0 to 5.38
New Hampshire	5.39 to 7.15
New Jersey	0 to 5.38
New Mexico	5.39 to 7.15
New York	0 to 5.38
North Carolina	5.39 to 7.15

CDC Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™

Histoplasmosis



Darker= more likely
Diagonal shading=potential

Darker shading shows areas where *Histoplasma* is more likely to live. Diagonal shading shows the potential range of *Histoplasma*. This map might change in the future as more data become available. CDC used data from the sources below to create this map.

- Armstrong PA, Jackson BR, Haselow D, Fields V, Ireland M, Austin C, et al. Multistate epidemiology of histoplasmosis, United States, 2011-2014. Emerg Infect Dis. 2018 Mar;24(3):425-31.
- Benedict K, Mody RK. Epidemiology of histoplasmosis outbreaks, United States, 1938-2013. Emerg Infect Dis. 2016 Mar;22(3).
- Benedict K, Thompson GR, Deresinski S, Chiller T. Mycotic infections acquired outside areas of known endemicity, United States. Emerg Infect Dis. 2015 Nov;21(11):1935-41.
- Edwards LB, Acquaviva FA, Livesay VT, Cross FW, Palmer CE. An atlas of sensitivity to tuberculin, PPD-B, and histoplasmin in the United States. Am Rev Respir Dis. 1969 Apr;99(4):Suppl:1-132.
- Manos NE, Ferebee SH, Kerschbaum WF. Geographic variation in the prevalence of histoplasmin sensitivity. Diseases of the chest. 1956 Jun;29(6):649-68.

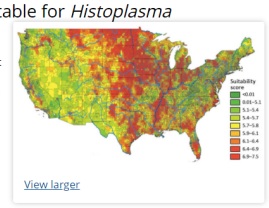
This map shows CDC's current estimate of where the fungus that causes histoplasmosis lives in the environment in the United States. This fungus (*Histoplasma capsulatum*) is more widely distributed than scientists once thought, with cases reported around the world, particularly in humid areas. Histoplasmosis is occasionally

https://www.cdc.gov/fungal/diseases/histoplasmosis/maps.html

Statistical model for Histoplasma

Areas where the environment is likely suitable for *Histoplasma*

Scientists believe that the fungus *Histoplasma* grows best under certain environmental conditions. This map shows the results of statistical modeling that estimates where this fungus might be most likely to live. The model includes land cover, soil acidity, and distance from water, although other environmental factors can also affect where *Histoplasma* can live.[8] On this map, higher suitability scores (in red) indicate areas that are likely to have better conditions for *Histoplasma*. It is important to recognize that this map is based on a statistical model and that all models have assumptions and limitations. More research is needed to better understand where *Histoplasma* most commonly lives.



View larger

Histoplasmosis surveillance

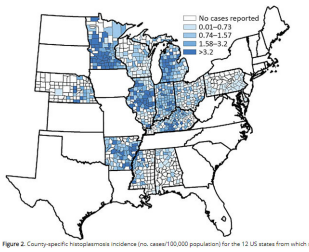
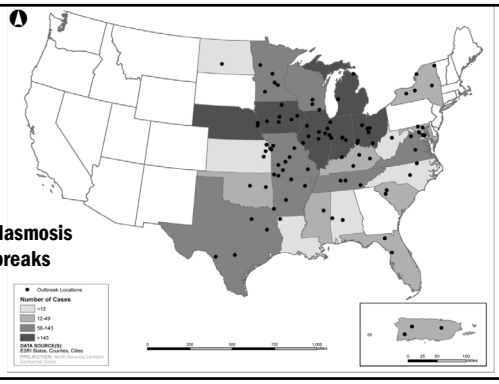


Figure 2. County-specific histoplasmosis incidence (no. cases/100,000 population) for the 12 US states from which surveillance data were available, 2011–2014.

https://wwwnc.cdc.gov/eid/article/24/3/17-1258-f2

Histoplasmosis outbreaks



Why don't we have better data?

PLOS ONE

OPEN ACCESS PEER-REVIEWED

RESEARCH ARTICLE

Presumed ocular histoplasmosis syndrome in a commercially insured population, United States

Kaitlin Benedict, Jessica G. Shantha, Steven Yeh, Karlyn D. Beer, Brendan R. Jackson

Published: March 13, 2020 • https://doi.org/10.1371/journal.pone.0230305

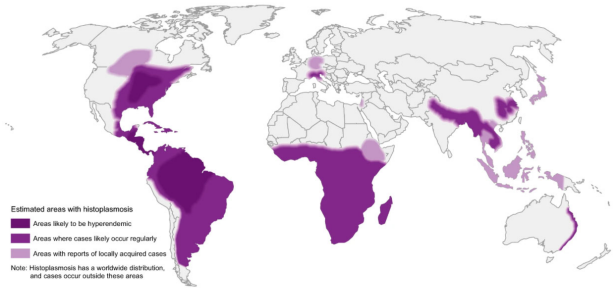
https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0230305

Epidemiology of Histoplasmosis Outbreaks, United States, 1938–2013

Kaitlin Benedict and Rajal K. Mody

EMERGING INFECTIOUS DISEASES

Histoplasmosis has been described as the most common endemic mycosis in the United States. However, histoplasmosis is not nationally notifiable. Its presumed geographic distribution is largely derived from skin test surveys performed during the 1940s, and information about its local features comes primarily from outbreak investigations.



https://link.springer.com/article/10.1007/s11046-020-00431-2

Areas for further research

Overall, more research is needed about:

- *Histoplasma* as a potential cause of POHS
- The prevalence of and risk factors for POHS
- The personal and public health impact of POHS-associated vision loss

https://www.cdc.gov/fungal/diseases/histoplasmosis/POHS.html

References

1. Manos NE, Ferebee SH, Kerschbaum WF. Geographic variation in the prevalence of histoplasmin sensitivity . Dis Chest. 1956 Jun;29(6):649-68.
2. Colombo AL, Tobon A, Restrepo A, Queiroz-Telles F, Nucci M. Epidemiology of endemic systemic fungal infections in Latin America . Med Mycol. 2011 Nov;49(8):785-98.
3. Loulergue P, Bastides F, Baudouin V, Chandenier J, Mariani-Kurkdjian P, Dupont B, et al. Literature review and case histories of Histoplasma capsulatum duboisii infections in HIV-infected patients . Emerg Infect Dis. 2007 Nov;13(11):1647-52.
4. Chakrabarti A, Slavin MA. Endemic fungal infections in the Asia-Pacific region . Med Mycol. 2011 May;49(4):337-44.
5. McLeod DS, Mortimer RH, Perry-Keene DA, Allworth A, Woods ML, Perry-Keene J, et al. Histoplasmosis in Australia: report of 16 cases and literature review . Medicine. 2011 Jan;90(1):61-8.
6. Benedict K, Mody RK. Epidemiology of histoplasmosis outbreaks, United States, 1938-2013 . Emerg Infect Dis 2016;22.
7. Armstrong PA, Jackson BR, Haselow D, Fields V, Ireland M, Austin C, et al. Multistate epidemiology of histoplasmosis, United States, 2011-2014 . Emerg Infect Dis 2018;24:425-31.
8. Maiga AW, Deppen S, Scaffidi BK, et al. Mapping *Histoplasma capsulatum* exposure, United States . Emerg Infect Dis 2018;24:1835-9.

Why this matters

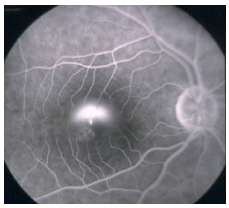
Failure to diagnose is a possibilistic failure

The importance of counting

Misconception #2

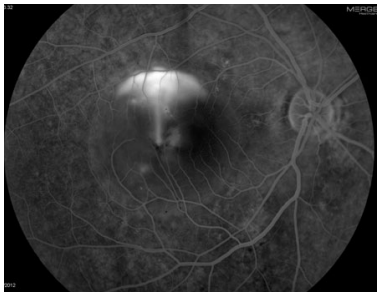
The smokestack appearance on fluorescein angiography is the best indicator of central serous chorioretinopathy.

Smokestack appearance

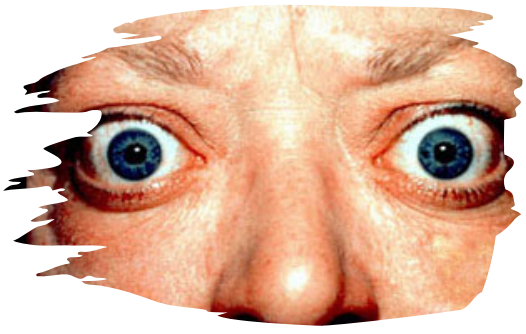


Central Serous Chorioretinopathy Cedar Rapids | CSC Iowa City (iowaretina.com)

Smokestack Sign



Retina Louisville | Central Serous Retinopathy KY | Bennett & Bloom (eyecenters.com)



Background

Smokestack leak is the "classic" appearance of CSR

1961 – Novotny and Alvis	Perfected use of FA to evaluate retinal circulation
1971 – Shimizu and Tobari	*upward diffusion*
1972 – Burton	***smokestack*** *or vertical appearing leak*
1973 – Wessing	*fluorescein flag*

How often does it happen?

7% to 29%

95% of cases have leaks in RPE as demonstrated by FA

Graefes Arch Clin Exp Ophthalmol (2010) 248:339–351
DOI 10.1007/s00417-009-1212-5

RETINAL DISORDERS

Smokestack leak in central serous chorioretinopathy

Dhiren Bujarborua · Pran N. Nagpal · Manab Deka

84% male

479 cases of CSR

Median age 34

Median duration of symptoms 15 days

Graefes Arch Clin Exp Ophthalmol (2010) 248:339–351
DOI 10.1007/s00417-009-1212-5
RETINAL DISORDERS
Smokestack leak in central serous chorioretinopathy
Dhiren Bujarborua · Pran N. Nagpal · Manab Deka

Most common in parafovea
Superior nasal
(31%)

69 of 479 cases had smokestack appearance

14.4%

70% in first acute episode

Why does it happen?

Point leak from RPE in AV or early venous phase in FA
.....which increases in size
.....and moves up
.....extends nasal and temporally

Rayleigh-Benard convection
Material in subretinal fluid
Convection currents
Temperature gradient of choroid

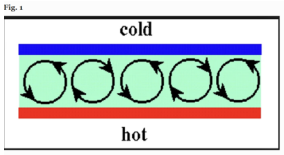
Rayleigh-Benard Convection

Buoyancy-driven flow of a fluid heated from below and cooled from above

Choroid is warmer than the retina

Forms convection rolls

Fig. 1



https://msd-journal.springeropen.com/articles/10.1186/s40486-020-00123-y

20% of unbound protein dye is the portion which fluoresces

This portion rises as it is less dense

Creating a thin upward trail

It's complicated.

Prevalence

1. Age-related macular degeneration
2. Diabetic retinopathy
3. Retinal vein occlusions
4. Central serous chorioretinopathy

Incidence

9.9 cases/100,000 men

1.7 cases/100,000 women

20/20

20/200

Central scotoma

Acute and Chronic forms

3-4 months

Longer than 4-6 months

American Journal of Ophthalmology
Volume 223, March 2021, Pages 129-139

Original Article

OCT Risk Factors for 3-Year Development of Macular Complications in Eyes With "Resolved" Chronic Central Serous Chorioretinopathy

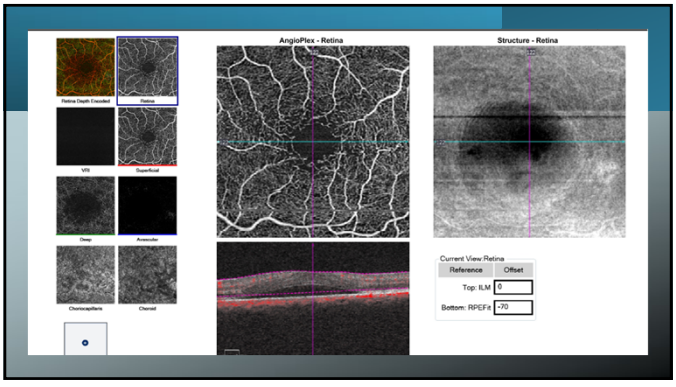
Results

At month 36, 20 eyes (28.2%) developed macular complications. Nine eyes (12.7%) displayed CNV, 9 eyes (12.7%) had large areas of RPE atrophy, and 2 eyes (2.8%) developed cystoid macular degeneration. The following factors were associated with an increased risk of development of CNV: intraretinal hyper-reflective foci had an HR of 11.58 (95% confidence interval [CI]: 1.10-37.24; $P = .040$); inner choroidal attenuation had an HR of 9.66 (95% CI: 1.07-22.34; $P = .043$); and the presence of macular complications in the fellow eye had an HR of 20.17 (95% CI: 1.34-39.41; $P = .030$). Factors associated with the development of RPE atrophy were also identified: ONL thinning had an HR of 13.47 (95% CI: 1.10-39.86; $P = .042$); dome-shaped PED had an HR of 21.40 (95% CI: 1.50-41.10; $P = .031$); and inner choroidal attenuation had an HR of 13.20 (95% CI: 1.07-39.32; $P = .044$).

28.2% had macular complications

12.7% CNV

12.7% RPE atrophy



Why this matters

Failure to diagnose is a possibilistic failure

We aren't doing FAs…but the physiology matters

Misconception #3

Ocular ischemic syndrome always needs treatment

Ocular Ischemic Syndrome

Decreased vision

Orbital pain

Afterimages

Transient monocular vision loss

Signs

Retinal hemorrhages

Iris neovascularization

Posterior segment neovascularization

Often unilateral

Men>women

7.5 per million

Average age = 65

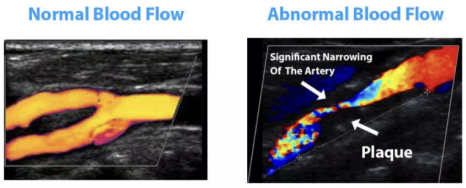
50% with OIS have total ipsilateral occlusion of carotid

5-year mortality rate is 40%

Additional testing

1. MRA/CTA
2. FA
3. ESR/CRP
4. Carotid doppler

Utility of Carotid Doppler



https://ultrasound.ie/the-carotid-doppler-ultrasound/

89-year-old male

- **Red spot** in vision, unsure which eye x 8 months
- Previous CRVO OS in 2016
- Non-exudative AMD OU
- Central geographic atrophy OS
- Count fingers @ 3 ft vision OS
- IOP 16/20 mmHg

Initial Presentation

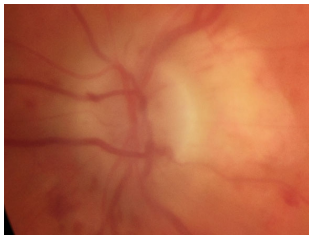
DFE

0.15 OD/0.2 OS

Disc collaterals versus early neo OS

Mid-peripheral to peripheral dot hemes OS

RPE clumping OD, central atrophy OS



Return to clinic one month

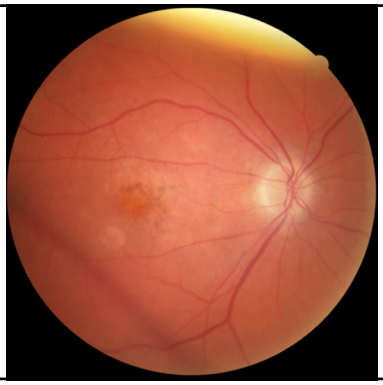
3 months later...

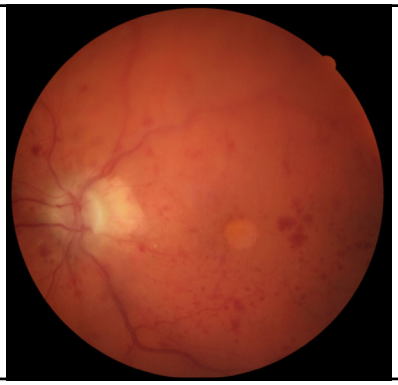
Anterior Segment

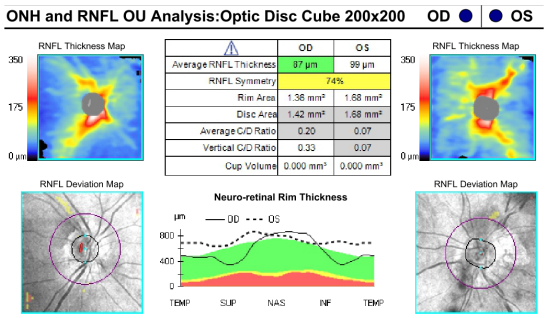
- Iris
  - No NVI
- Gonioscopy
  - No NVA

Posterior Segment

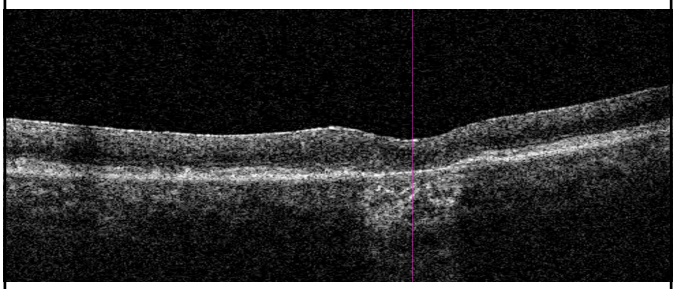
- Hemes – MUCH worse
- No pain
- IOP 18/16 mmHg







	OD	OS
Average RNFL Thickness	87 µm	99 µm
RNFL Symmetry	74%	
Rim Area	1.38 mm²	1.68 mm²
Disc Area	1.42 mm²	1.68 mm²
Average C/D Ratio	0.20	0.07
Vertical C/D Ratio	0.33	0.07
Cup Volume	0.000 mm³	0.000 mm³



Thoughts

Initial sense of urgency

Vision is already very poor

What is happening here?

Re-bleed?

Neovascularization?

OIS?

Does it matter?

Keep the eye from becoming a blind, PAINFUL, eye

Review of medical history

Carotid doppler

2016

No hemodynamically significant stenosis

2020

Less than 50% stenosis

Plan

Consult with retina within 2 weeks

No additional imaging ordered

*Appointment scheduled for 2 months later

Case 2

78-year old male

Jabbing pain over right eye

Case 2

Type 2 Diabetes
Hypertension
Hyperlipidemia

Medical History

History of BRVO or CRVO OD

Ocular History

Case 2

20/400 OD

IOP 16/18 mmHg
1+ NSC OU
Posterior cortical spoking OS

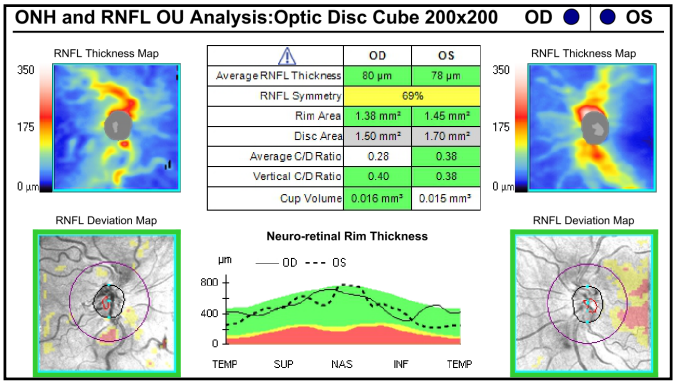
Timolol BID OU

Brimonidine BID OU

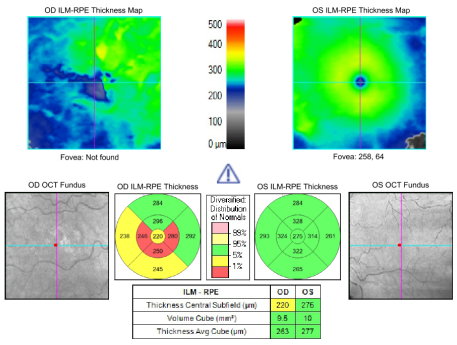
OD	OS
0.2 with collaterals **ERM** Central atrophy with RPE hyperplasia	**0.3**
Blot hemes inferior-temp	Unremarkable

OD	OS
0.2 with collaterals	**0.3**
ERM Central atrophy with RPE hyperplasia	
Blot hemes inferior-temp	Unremarkable

Fine NVE???



	OD	OS
Average RNFL Thickness	80 µm	78 µm
RNFL Symmetry	69%	
Rim Area	1.38 mm²	1.45 mm²
Disc Area	1.50 mm²	1.70 mm²
Average C/D Ratio	0.28	0.38
Vertical C/D Ratio	0.40	0.38
Cup Volume	0.016 mm³	0.015 mm³



ILM - RPE	OD	OS
Thickness Central Subfield (µm)	220	276
Volume Cube (mm³)	9.5	10
Thickness Avg Cube (µm)	263	277

Thoughts

BRVO?

Neovascularization?

OIS?

Likely not OIS

Plan

Refer to retina within 2 weeks

8/2021

Ophthalmology

- Collaterals OD
- Vessels attenuated and tortuous OD
- Hemes inf and inf-temp OD

- OCT disruption of IS/OS junction with retinal disorganization and atrophy, no SRF
- Recommend IVFA OD—*pending results*

Case 3

75-year-old AA male

Blurred vision at distance OU

20/50 OD,OS

Case 3

Medical history

3 different blood thinners

Left superior quadranopsia from old cerebral bleed

Type 2 diabetes

Ocular history

Mild/moderate DM retinopathy

Physiological cupping

Scattered dot /blot hemes in midperiphery OU

20/30 2+ NSC 1+ CS	Previous DFE (10/2020)	20/40 2+ NSC 1+ CS
OD		OS
0.35		0.5
One dot heme inf-nasal		2 blot hemes sup-temp 1 sup nasal 1 temp 2 bigger blot hemes inf-nasal

Carotid Doppler 2013 and 2014

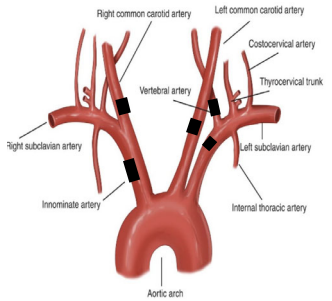
No hemodynamically significant stenosis

Unchanged reversed flow in left vertebral artery (subclavian steal)

Carotid Doppler 2020

Impression

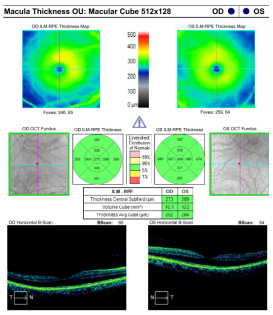
1. Diffusely occluded right internal carotid artery
2. Diffusely occluded left internal carotid artery
3. Diffusely occluded left vertebral artery. Moderate to severe proximal left subclavian artery stenosis
4. Right vertebral artery patient with kinked appearance in V1. Approximately 50% stenosis of the proximal innominate artery
5. These findings are known and the patient was evaluated by vascular surgery at an outside facility



http://healthtechindia.org.in/supra-aortic-therapies/

DFE 2021

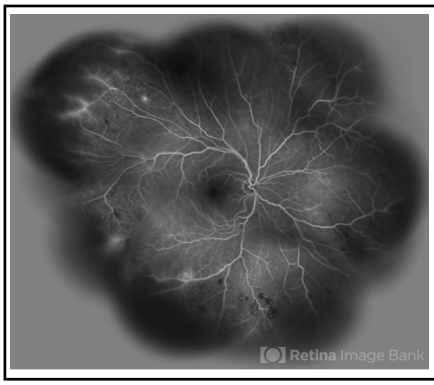
OD	OS
0.4	0.4
Scattered dot hemes mid-peripheral	Scattered dot hemes mid-peripheral



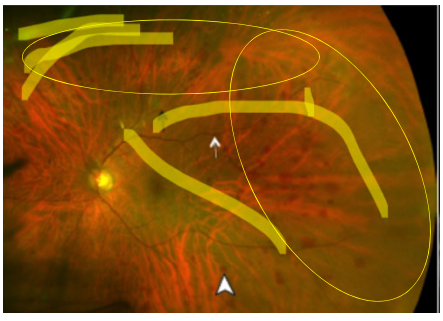
Plan

Return to clinic 6 months

Continue care with cardiologist and vascular surgeon



Why the mid-periphery?



Unilateral retinopathy in a patient with diabetes and coronary heart disease

BMJ 2018 ; 361 doi: https://doi.org/10.1136/bmj.k1703 (Published 14 June 2018)
Cite this as: BMJ 2018;361:k1703

Venous Stasis Retinopathy

Carotid artery stenosis or occlusion → **Reduced perfusion pressure** → **Microaneurysms form in capillary beds** → **Microaneurysms leak forming small, non-confluent hemorrhages in mid-periphery**

Ocular ischemic syndrome always needs treatment

- ☐ Pain
- ☐ Neovascularization of anterior segment
- ☐ Neovascularization of posterior segment
- ☒ Retinal hemes

Misconception #3

Ocular ischemic syndrome always needs treatment

1. Cardiac disease
2. Stroke
3. Cancer

Why this matters

OIS doesn't always present with mid-peripheral hemorrhages

Failure to diagnose OIS has a major systemic impact

Misconception #4

CNV always occurs near fovea and is linked to an etiology

October 2020

CC: Blurred vision when looking at TV OU

Ocular history: Non-exudative macular degeneration OU
Cataracts OS>OD
Med Hx: HTN

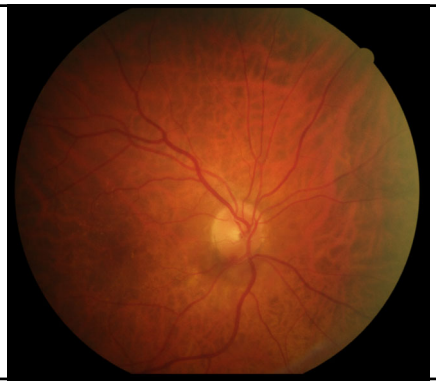
BCVA
20/20 OD
20/40 OS

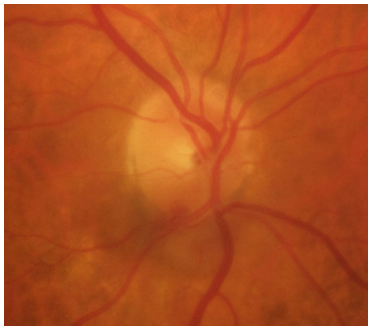
IOP 12/11 mmHG

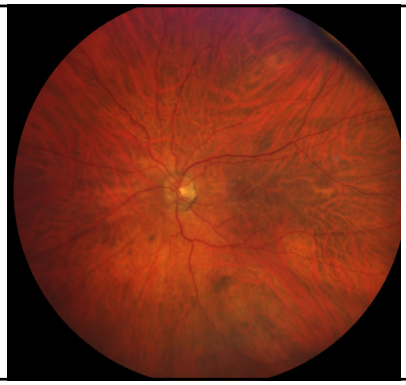
DFE

- 0.3 OD/0.35 OS
- Peripapillary hemorrhage OD inferiorly
- RPE mottling OU

- Tried to take OCT RNFL—difficulty fixating
- Macula OCT
  - Drusenoid pigment epithelial detachment OD,OS



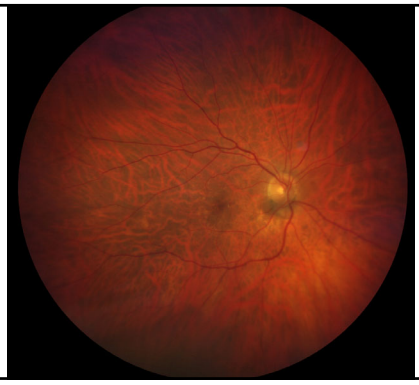


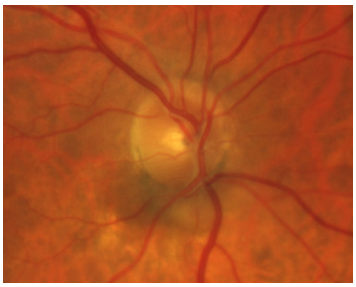


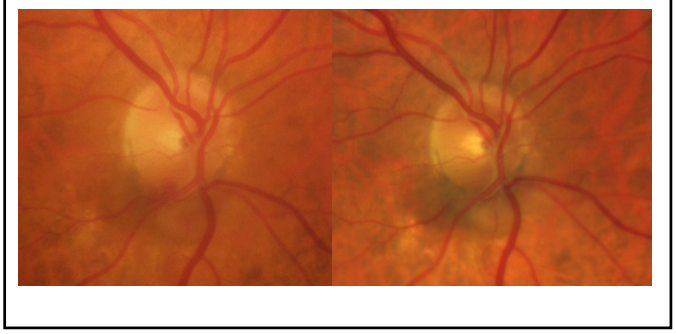
Plan

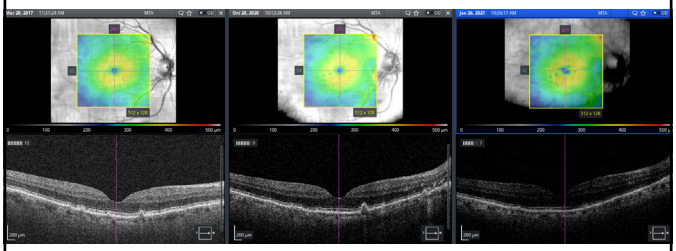
Return to clinic 3 months

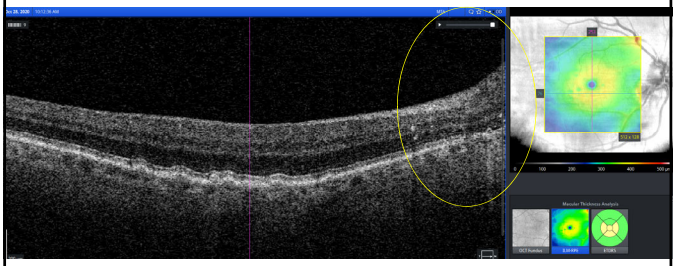
COVID-19…patient wasn't seen until 1/26/21











1/2021

- Follow-up
- Refill on artificial tears requested
- Glasses are working well
- BCVA 20/25 OD, 20/70 OS (PH 20/40)
- IOP 9/10 mmHg

DFE

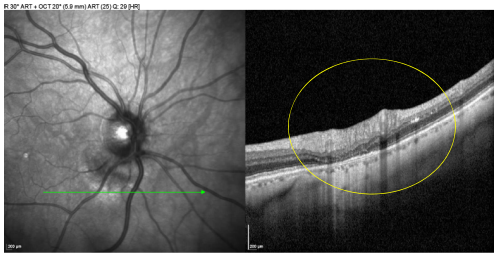
- C/D 0.3 OD/0.3 OS
  - Elevated pigmented region from 6-9 o'clock OD
  - Previous disc hemorrhage resolved
- **Suspect optic nerve melanoma OD**

Plan

Refer to ophthalmology for consult

3 days later (Ophthalmology)

- Vision is still stable (20/25, 20/40 PH)
- OCT ONH
  - Flat choroid with hypperreflective elevation inferior temporal optic nerve, trace sub-retinal fluid
- **Diagnosis: Peripapillary CNV**
  - No treatment due to good visual acuity OD

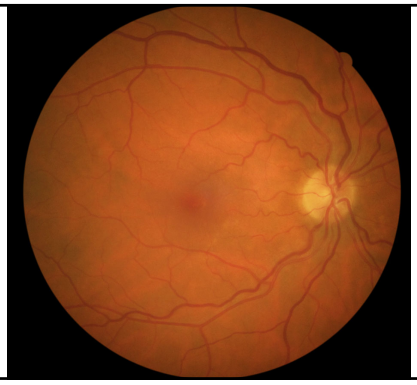


Normal nerve	Disc heme inf	Suspected ONH melanoma	Peri-papillary CNV	Stable appearance
20/25	20/25	20/25	20/20-1	20/25

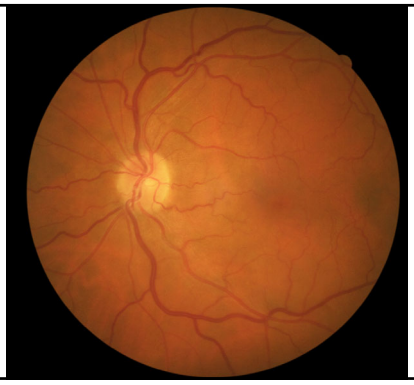
72 year old white male

Just here for a check-up

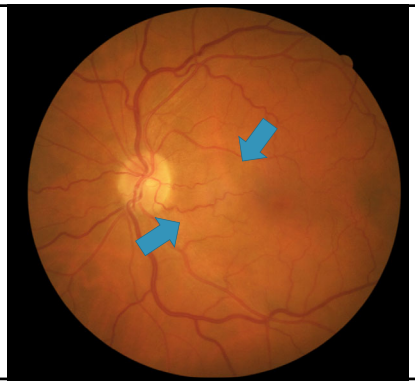
2019



2019

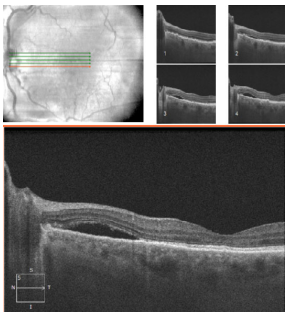


2019



Why is this happening ?

20/60



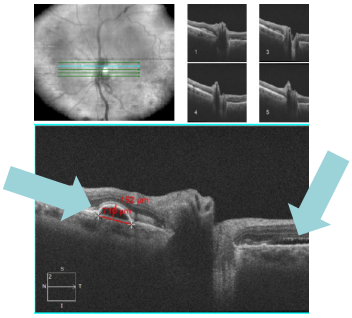
Shallow neurosensory detachment between nerve and fovea

Central serous chorioretinopathy?

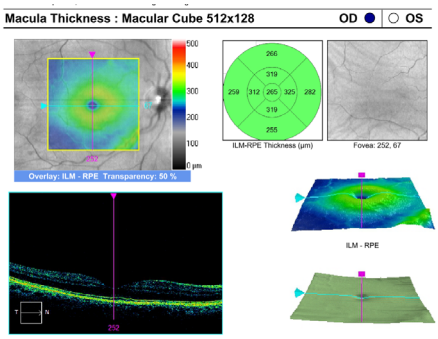
CNV?

AMD?

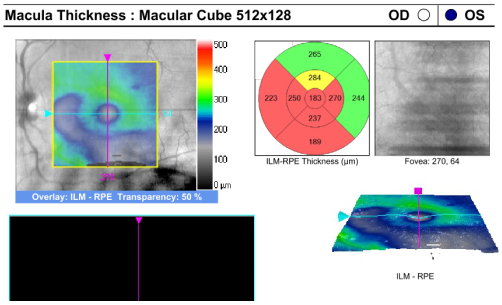
10/2020
New PED



20/30

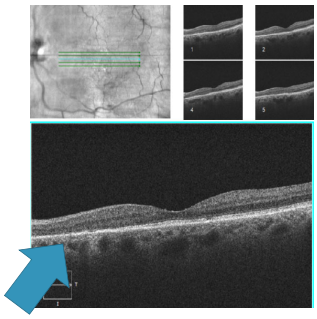


20/40



Loss of outer retina

Irregular IS/OS



Plan

No treatment due to good acuity (and there is nothing to treat)

Ophthalmology opted to not perform a FA

Choroidal neovascularization

- Can occur in 30 different diseases
- Over age 50, most commonly caused by AMD
- Under age 50
  - Pathologic myopic
  - Angioid streaks
  - Trauma
  - Idiopathic → **Inflammation**

American Journal of Ophthalmology
Available online 21 July 2021
In Press, Journal Pre-proof

Original Articles

Long-term analysis of clinical features and treatment outcomes of inflammatory choroidal neovascularization

Mirinae Kim [1,2], Junhyuck Lee [1], Young-Gun Park [1,2], Young-Hoon Park [1,2,*]

n=65

5-year study

43.1% had recurrence

Peripapillary CNV

European Journal of Ophthalmology / Vol. 19 no. 1, 2009 / pp. 163-165

SHORT COMMUNICATIONS & CASE REPORTS

Treatment of peripapillary choroidal neovascularization with intravitreal bevacizumab

A.E. HOEH, K.B. SCHAAL, T. ACH, S. DITHMAR
Department of Ophthalmology, University of Heidelberg, Heidelberg - Germany

1500 microns

Uncommon

Problematic because of foveal spread

Typically idiopathic or due to AMD

Anti-VEGF is first-line treatment

10% of extrafoveal CNV occurs at ONH

ARVO Annual Meeting Abstract | May 2004

Peripapillary Choroidal Neovascularization

H.S. Reddy; J.I. Loewenstein; A. Lane; L. Shen
+ Author Affiliations & Notes

Investigative Ophthalmology & Visual Science May 2004, Vol.45, 3077. doi:

Conservative management

75% were occult
38% extended into fovea
Visual acuity preserved >750 microns

Misconception #4

CNV always occurs near fovea and is linked to an etiology

MYTH

Why this matters

CNV that occurs outside the macula doesn't always have a specific etiology

Often this type of CNV isn't treated

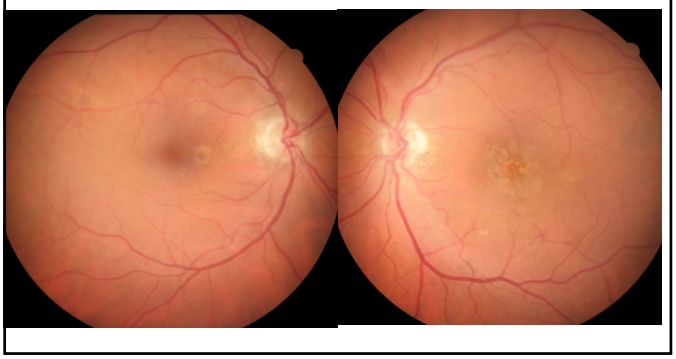
Misconception #5

In macular degeneration a dramatic drop in acuity is always due to development of CNV

Case 1: Non-exudative AMD OS>OD

- 71 year old white male
- VA slowly decreasing OS, OD stable
- IVFA 9/2020—FAZ distortion OS, no fluid
- Last appointment: 20/50, 20/70
- Would like new glasses

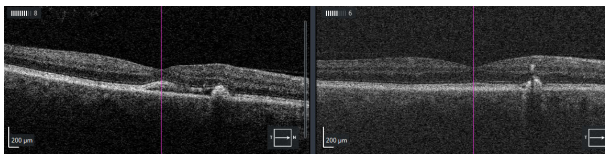
20/50 20/150



0.25 OD/0.25 OS

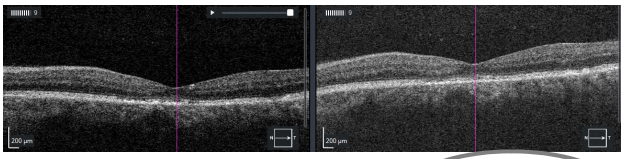
- OD- PED with RPE changes
- OS - PEDs with RPE changes OS
  - Soft drusen with areas of atrophy

20/50 20/50



OD

2019 2021



20/50

OS

20/150

Progression of visual acuity decline with stable OCT

No CNV

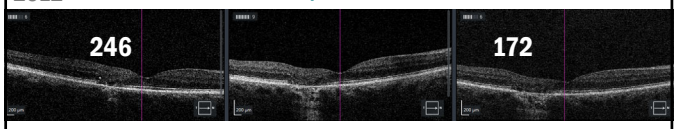
Progression on OCT with stable visual acuity

74-year-old white male

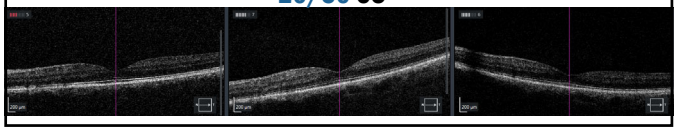
- No visual changes
- Wants new glasses
- Non-exudative AMD OD>OS
- Nuclear cataracts
- Physiological nerve cupping
- HLD, HTN,CAD, DM

Non-neovascular AMD OD>OS

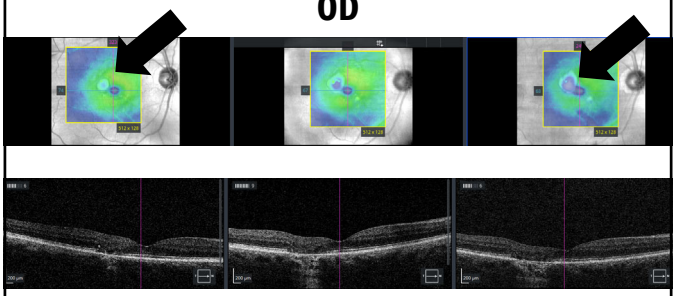
2012 20/40 OD 2021

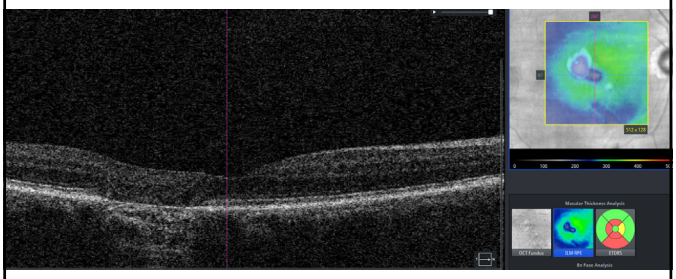


20/30 OS



OD





Case 3

- Non-exudative AMD OD 20/30
- Exudative AMD OS 20/60
  - History of Eylea
    - TREX (14 weeks)

CC: Here for a refraction

- 20/40 OD
- Worse than 20/800
  - Patient couldn't see the bottom half of the screen
  - Tech measured him as 20/150

Thoughts

New CNV activity

Retinal detachment

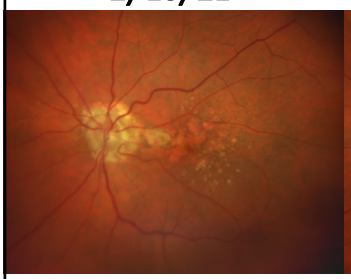
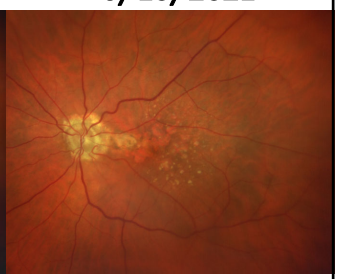
NAION/AION

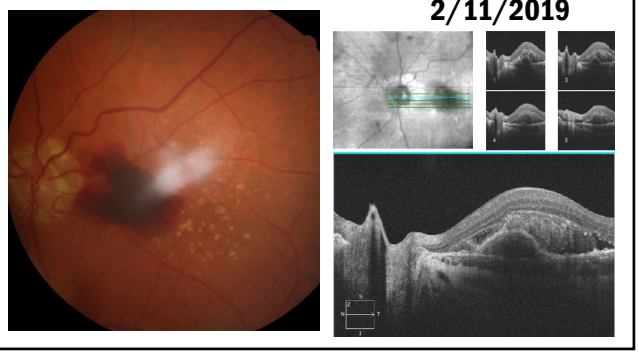
Vascular occlusion

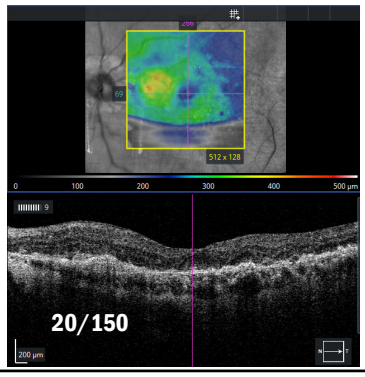
Refraction → DFE

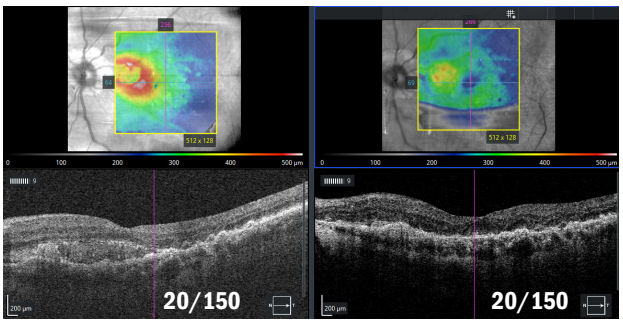
0.35 OD/0.35 OS

Optic nerves were normal—no swelling or pallor

2/18/21	5/10/2021
	
20/60	20/150







Plan

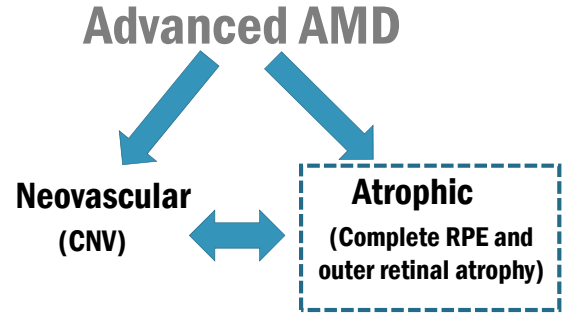
Instant messaged ophthalmologist

-Looked at OCT together

Already had an appointment scheduled the following week

90% **of AMD is dry**

80% **of vision loss is caused by exudative AMD**



CATT 2 year results

59% increase of new atrophy

IVAN

OR 1.47 new atrophy

PLOS ONE

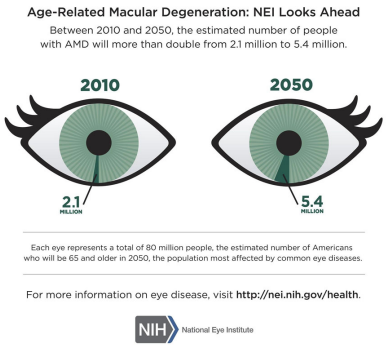
PLoS One. 2020; 15(5): e0232353.
Published online 2020 May 5. doi: 10.1371/journal.pone.0232353
PMCID: PMC7200004
PMID: 32369500

Complete RPE and outer retinal atrophy in patients receiving anti-VEGF treatment for neovascular age-related macular degeneration

No difference in anti-VEGF agents

Aflibercept
Ranibizumab
Bevacizumab

Leading cause of blindness in industrialized countries



Misconception #5

In macular degeneration a dramatic drop in acuity is always due to development of CNV

MYTH

5 Misconceptions

Histoplasmosis only occurs in the OH and MS river valleys

Histoplasmosis only occurs in the OH and MS river valleys

The smokestack appearance on fluorescein angiography is the best indicator of central serous chorioretinopathy.

Histoplasmosis only occurs in the OH and MS river valleys

The smokestack appearance on fluorescein angiography is the best indicator of central serous chorioretinopathy.

Ocular ischemic syndrome always needs treatment

Histoplasmosis only occurs in the OH and MS river valleys

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Ocular ischemic syndrome always needs treatment

CNV always occurs near fovea and is linked to an etiology

Histoplasmosis only occurs in the OH and MS river valleys

The smokestack appearance on fluorescein angiography is the best indicator of central serous chorioretinopathy.

Ocular ischemic syndrome always needs treatment

CNV always occurs near fovea and is linked to an etiology

In macular degeneration a dramatic drop in acuity is always due to development of CNV

Why this matters

Thinking we know is not the same as knowing

Thinking we know is dangerous

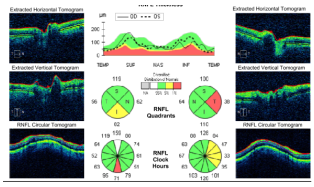
Thinking we know prevents us from knowing

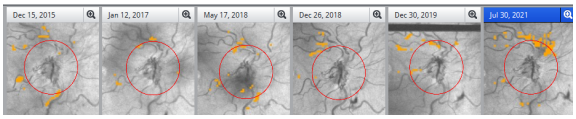
Thank you!

Central serous chorioretinopathy: An update on risk factors, pathophysiology and imaging modalities

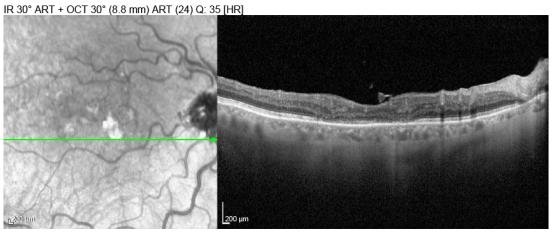
Progress in Retinal and Eye Research
Volume 79, November 2020, 100865

OIS S

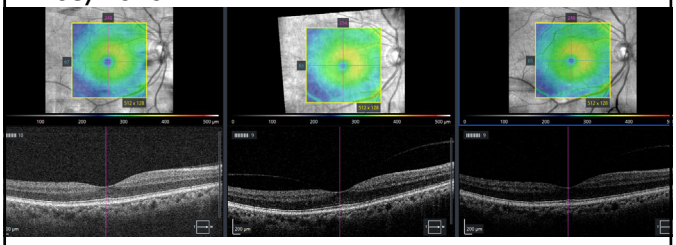


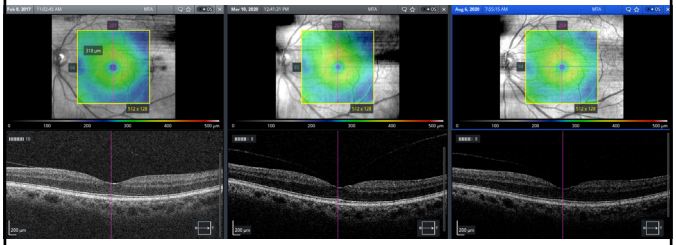


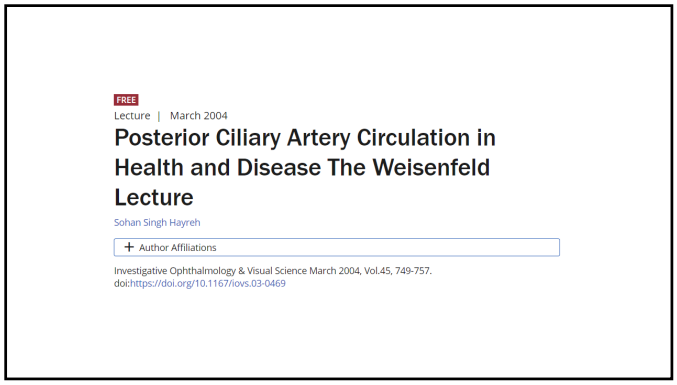
OD Mac scan August 21 -oph



08/2020







FREE

Lecture | March 2004

Posterior Ciliary Artery Circulation in Health and Disease The Weisenfeld Lecture

Sohan Singh Hayreh

+ Author Affiliations

Investigative Ophthalmology & Visual Science March 2004, Vol.45, 749-757.
doi:https://doi.org/10.1167/iovs.03-0469