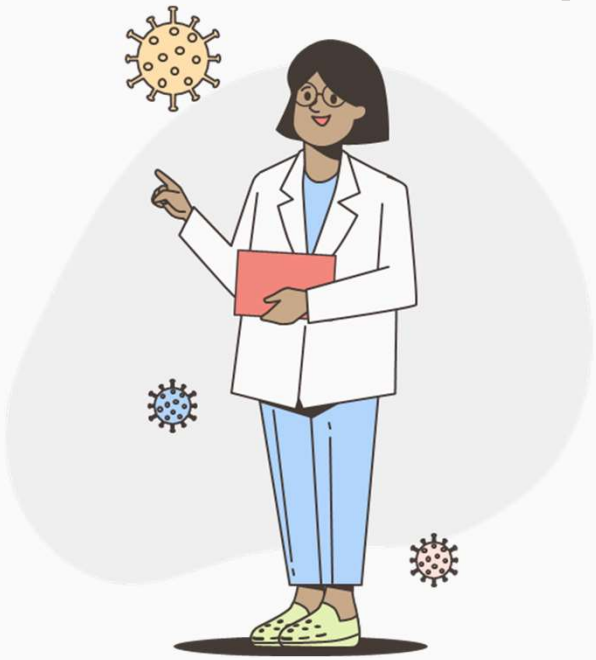


From Diagnosis to Dialogue: Identifying AMD Mimics to Improve Patient Education & Management

Casey Peterson, OD, FAAO & Emily Gum, OD

No financial disclosures

Course Objectives

Upon completion of this course, the clinician will be able to:

01 *Identify the diagnostic criteria for Age-Related Macular Degeneration (AMD)* including recent updates on detection, clinical features, and treatment and management.

02 *Differentiate AMD from its clinical mimickers* using key history, examination findings, and multimodal imaging to improve diagnostic accuracy.

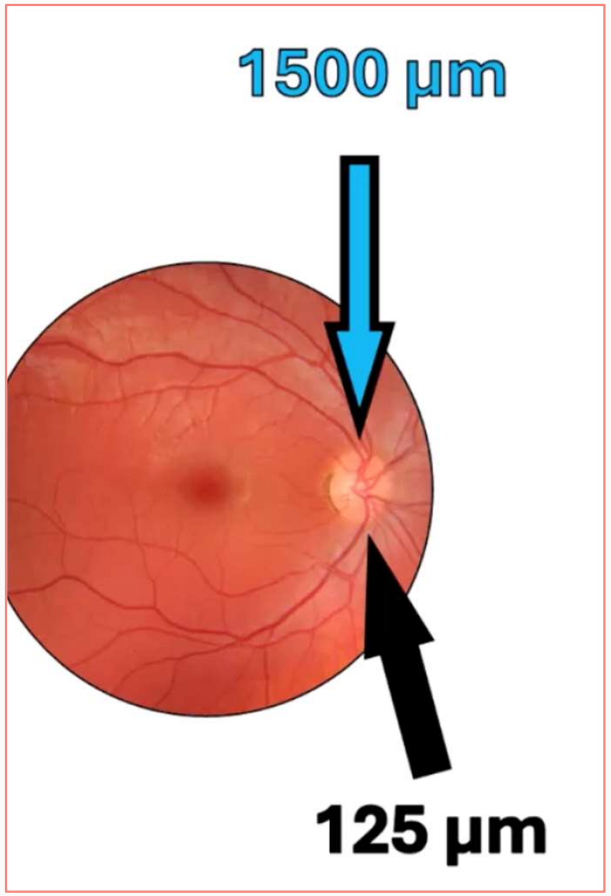
03 *Understand the psychological and behavioral impacts of AMD misdiagnosis*, including the associated depression, reduced quality of life, and changes in health-related behaviors that can arise when patients are incorrectly told they have AMD.

04 *Communicate diagnoses effectively* by providing clear, empathetic patient education—especially when navigating diagnostic uncertainty or clarifying conditions that resemble AMD.

First thing's first...

AMD Defined

American Academy of Ophthalmology Preferred Practice Pattern



A macular disorder characterized by ANY of the following:

RPE Changes

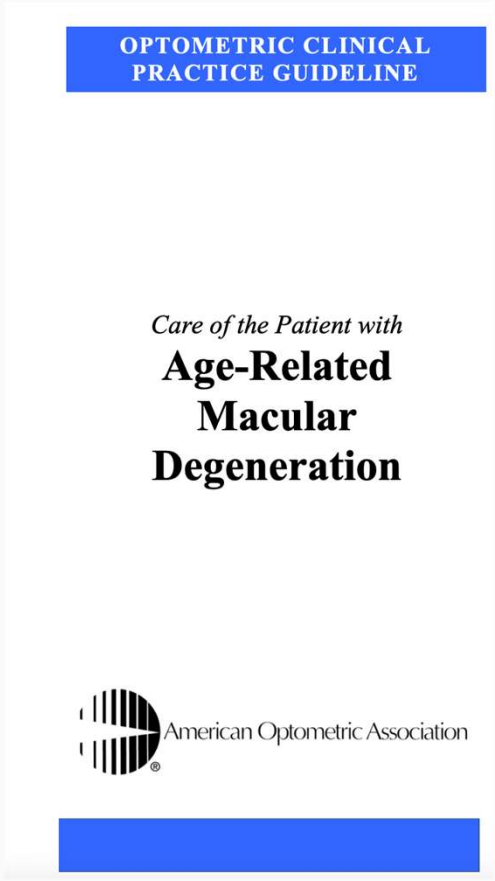
Drusen

Presence of intermediate-sized drusen (>63 µm)

Other Findings

Presence of GA, CNV, polypoidal choroidal vasculopathy, reticular pseudodrusen, or retinal angiomatous proliferation (RAP)

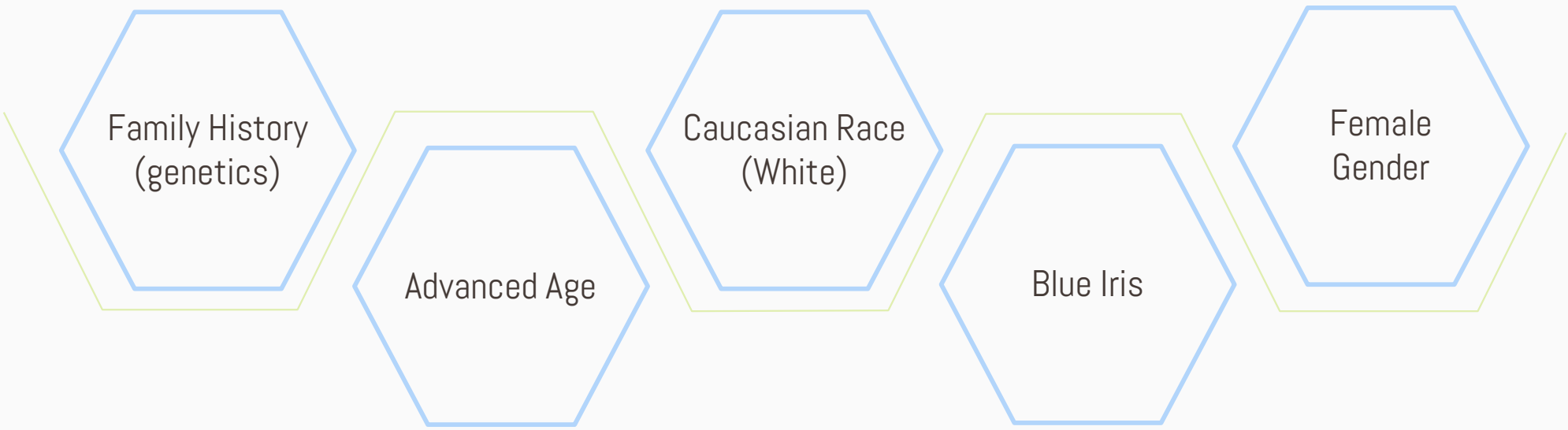
AOA Optometric Clinical Practice Guideline



An acquired retinal disorder characterized by ANY of these findings:

- **Pigmentary degeneration (atrophy)**
- **Drusen/lipofuscin deposits**
- **Exudative elevation of the outer retina within the macula**

AMD: Demographics and Associations



- Modifiable risk factors: Smoking, systemic arterial hypertension, diet

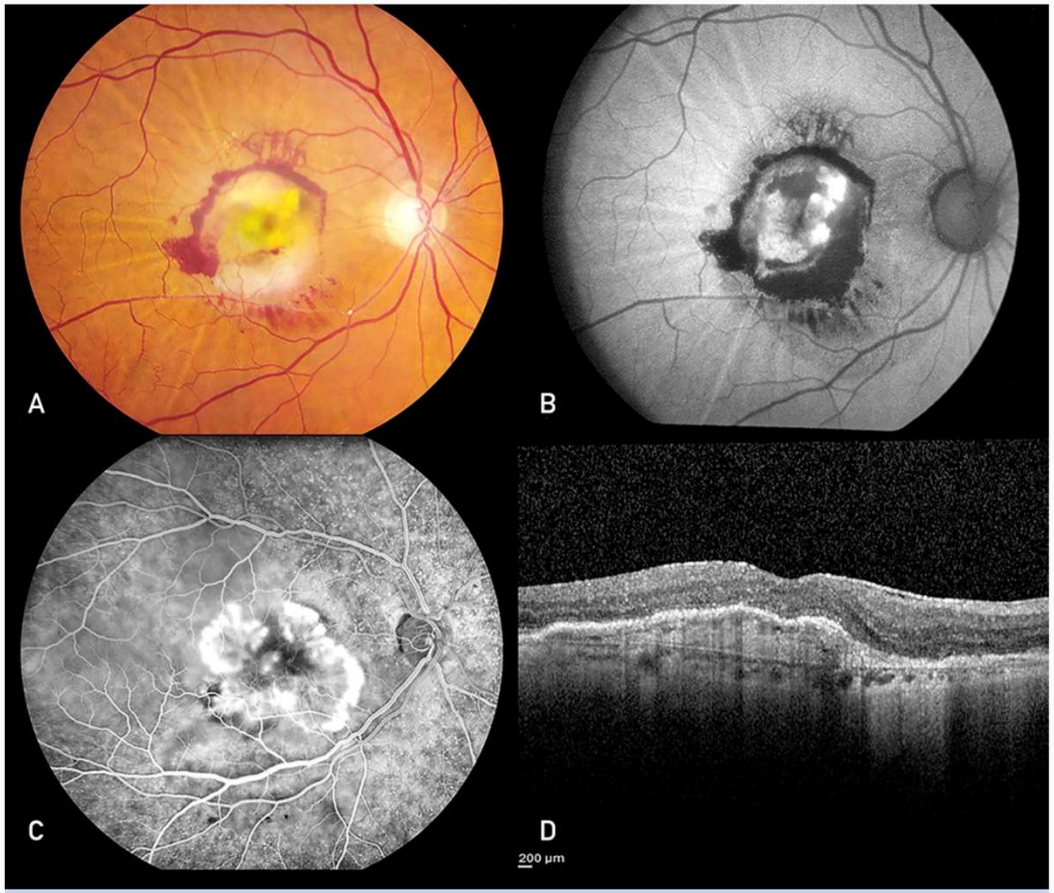


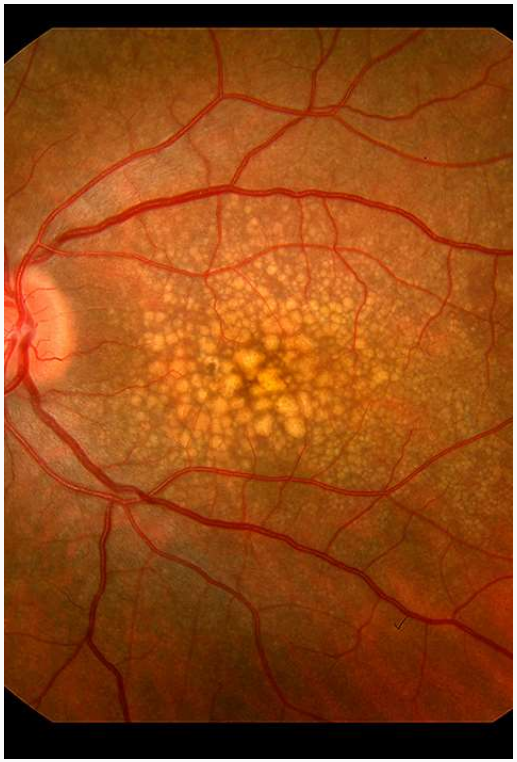
FIGURE 1. Multimodal imaging in neovascular age-related macular degeneration. (A) Color fundus photograph of macular neovascularization (MNV) seen as yellow gray lesion with surrounding hemorrhage. (B) Corresponding fundus autofluorescence showing mixed hyperautofluorescence and hypoautofluorescence. (C) Fluorescein angiography showing leakage from the MNV seen as lacy hyperfluorescence. (D) Optical coherence tomography showing subretinal pigment epithelial hyperreflective lesion suggestive of MNV.

AMD: Diagnosis & Management

- Gold standard for diagnosis
  - DFE and OCT
- Early/intermediate disease
  - Minimize progression risk
  - Watch for treatable change
- Advanced disease
  - MNV --> anti-VEGF injections
  - GA --> complement cascade inhibitors (?); clinical trials

So…what do we EXPECT to see in AMD?

- AGE 50 and older (with increased prevalence over 75)
- Most common (and most studied) in Caucasian populations
  - Late-stage AMD most common among Whites
- Bilateral disease
- Fundus findings of MACULAR drusen, RPE pigment changes, and/or advanced findings



https://www.vrmny.com/conditions/age-related-macular-degeneration/

AMD Prognosis

10–15%

Neovascularization

About 10-15% of patients with AMD develop MNV

10%

Geographic Atrophy

About 10% of patients with AMD suffer significant vision loss due to GA

Psychological implications of AMD diagnosis

Will I go blind from macular degeneration?

AI Overview

Macular degeneration (AMD) generally does not cause total blindness, but it severe loss of central vision, which may be classified as legal blindness. It af central retina, meaning peripheral (side) vision usually remains intact, allowir people to continue to take care of themselves.

Fear of blindness

- Ranked third by Americans as biggest fear (A Prevent Blindness American survey)
- Nearly 50% of Americans said **losing sight** would have the most negative impact on their daily life (Alliance for Eye and Vision Research poll)
- 79% of Americans stated blindness as "the worst possible thing that could happen to me" (aside from their own death/death of a loved one; Eye on Eyesight survey)
- Primary concerns
  - Loss of independence
  - Reduced quality of life

Psychological Implications of AMD: Defining Characteristics

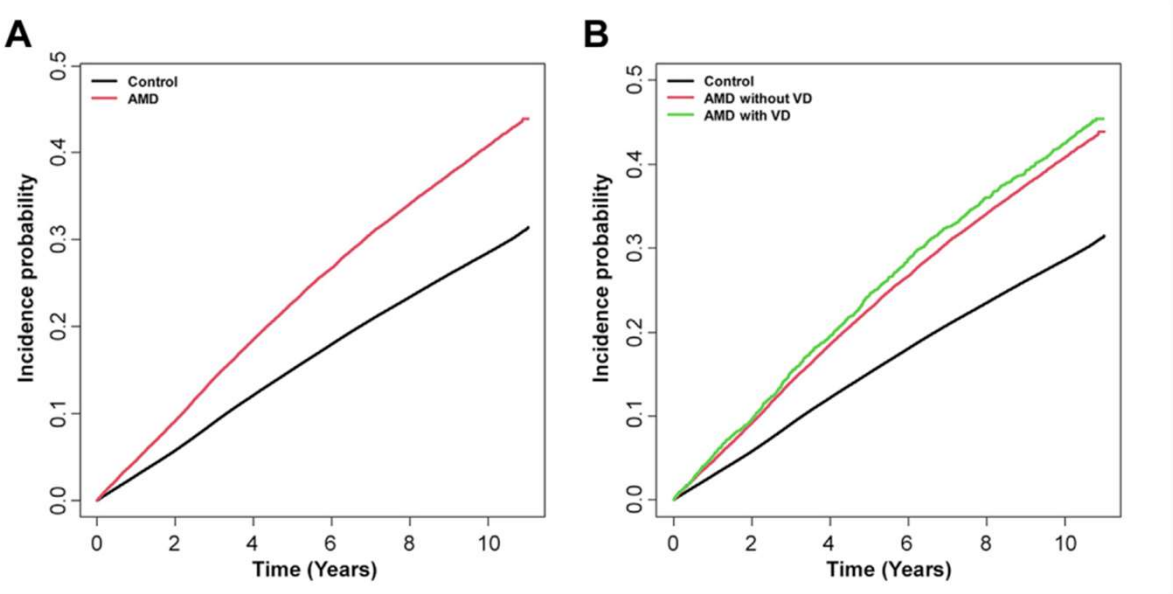
- Leading cause of vision loss and blindness in elderly Americans
    - Fourth leading cause of blindness worldwide
    - Expected to increase from 196 million (2020) to 288 million (2040)
- "Patients with early AMD, therefore, should be educated about the **progressive nature of the disease and the likelihood of vision decline.**"

Normal Vision

The same scene affected by age-related macular degeneration

https://medivizor.com/blog/2016/02/16/age-related-macular-degeneration-awareness-month/

Psychological implications of AMD: Living with a potentially blinding disease



- "Individuals who receive a diagnosis of AMD have been reported to have a greater prevalence of depression." (Hwang 2023)

AMERICAN ACADEMY OF OPHTHALMOLOGY®

Impact of Age-Related Macular Degeneration and Related Visual Disability on the Risk of Depression

A Nationwide Cohort Study

Sungsoon Hwang, MD,[1,2] Se Woong Kang, MD, PhD,[1] Sang Jin Kim, MD, PhD,[1] Kyungdo Han, PhD,[3] Bong Sung Kim, MS,[3] Wonyoung Jung, MD,[4] Dong Hui Lim, MD, PhD,[1,2,*] Dong Wook Shin, MD, PhD[2,4,*]

Screening tool:
Patient health questionnaire-2

Over the **last 2 weeks**, how often have you been bothered by the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	+1	+2	+3
2. Feeling down, depressed or hopeless	0	+1	+2	+3

PHQ-2 score obtained by adding score for each question (total points)

Psychological implications of AMD: Living with a potentially blinding disease

https://www.myamdteam.com/resources/wet-amd-and-your-mental-health)

- Study identified psychosocial effects of AMD
  - Associated with **high rates of depression, anxiety, emotional distress,** and increased mortality (Berman and Brodaty, 2006)
  - Not just the vision loss but *anticipation* of visual impairment
- 2025 study: Depression prevalence 56% (wet), 27% (dry), 20% (controls); Gouliopoulos et al.

Psychosocial implications of AMD: Anti-VEGF treatment

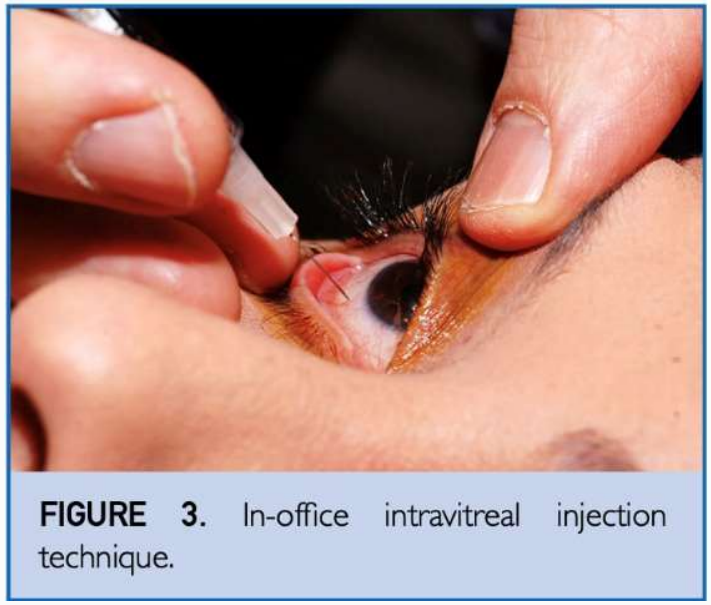


FIGURE 3. In-office intravitreal injection technique.

https://pmc.ncbi.nlm.nih.gov/articles/PMC11259890/pdf/main.pdf

- Studies find patient experiences of discomfort and fear common
  - Waiting for injection, fear of losing sight, fear for the unknown
  - Fears strongly tied to the first injection (patient's expectations of treatment)
- General improvement in patient Quality of Life found once treatments are initiated
  - Strong link between VA and QOL/mental health

Drusen in the retina—is it ever okay?

- Yes!
  - Normal aging change
  - Even found in young adults (in a study of patients <36yo, 22% of patients had small hard drusen)
- Hard drusen: small, round, well-defined
- Number and retinal location
- Absence of pigmentary change

Conclusions **The frequency of drusen in a younger Caucasian population aged 18–54 years is high, with 91.48% of all gradable eyes having drusen. The most frequent drusen subtype was hard distinct drusen <31.5 μm. No druse greater or equal in size to 125 μm was seen. Pigmentary changes are rare.**

Eye (2012) **26,** 1357–1362; doi:10.1038/eye.2012.165; published online 17 August 2012

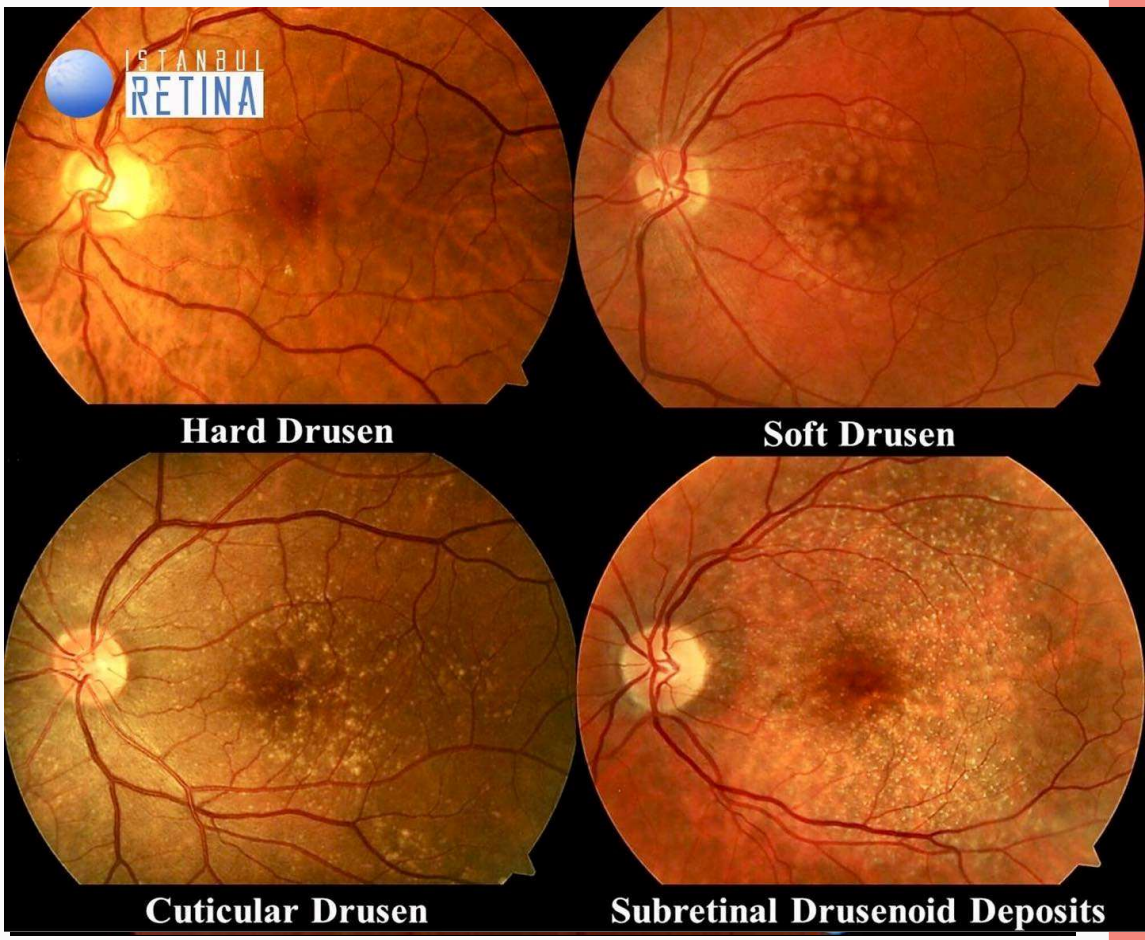
Table 2 Drusen prevalence by size and age

Age-group	*Number in group with gradable images*	*Any drusen present*	*Percent with any drusen present*	*Drusen size (<31.5 μm)*	*Percent*	*Drusen size (32<x<63 μm)*	*Percent*	*Drusen size (63<x<125 μm)*	
18–24	142	132	92.96	130	91.55	48	36.36	3	2.11
25–31	172	155	90.12	152	88.37	71	45.81	0	0.00
32–38	212	192	90.57	183	86.32	103	53.65	6	2.83
39–45	245	229	93.47	226	92.24	142	62.01	4	1.63
46–52	165	148	89.70	146	88.48	67	45.27	3	1.82
53–59	15	12	80.00	12	80.00	7	58.33	0	0.00

Drusen prevalence and pigmentary changes in Caucasians aged 18–54 years - PMC

Differentiating Drusen Types

- Hard
  - Small, yellowish with defined margins
- Soft
  - Larger, yellow/white, indistinct borders and elevated
- Cuticular (basal laminar drusen)
  - Triangular elevations of the RPE/BL --> "clumps" of tiny drusen often sparing macula
- Reticular pseudodrusen (subretinal drusenoid deposits)
  - Yellowish-white netlike pattern



Drusen and the AMD "Spectrum": How does it all relate?

- Hard drusen
  - Benign (normal/aging change)
  - *May* be associated with AMD
- Soft drusen
  - AMD association
    - Conveys an INCREASED RISK of advanced disease
- Cuticular drusen
  - Relatively benign (early stages/young patient)
  - *May* be associated with AMD (large size, macula involvement, patients over 60) --> these patients may develop GA/MNV
- Reticular drusen
  - AMD association
    - Conveys an INCREASED RISK of advanced disease

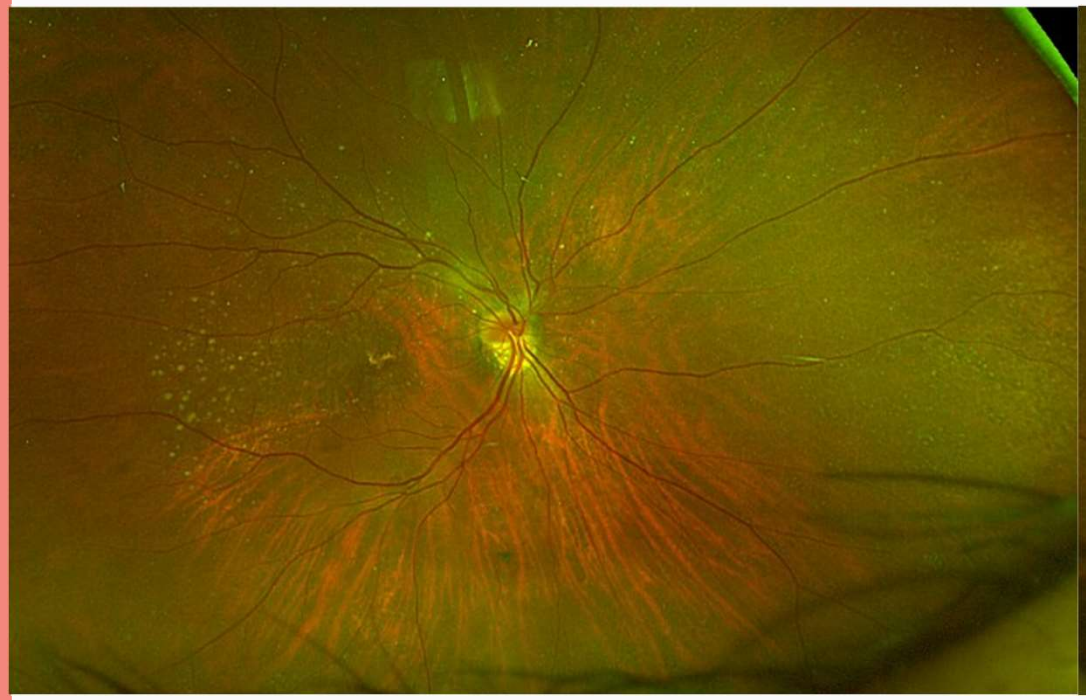
A Case

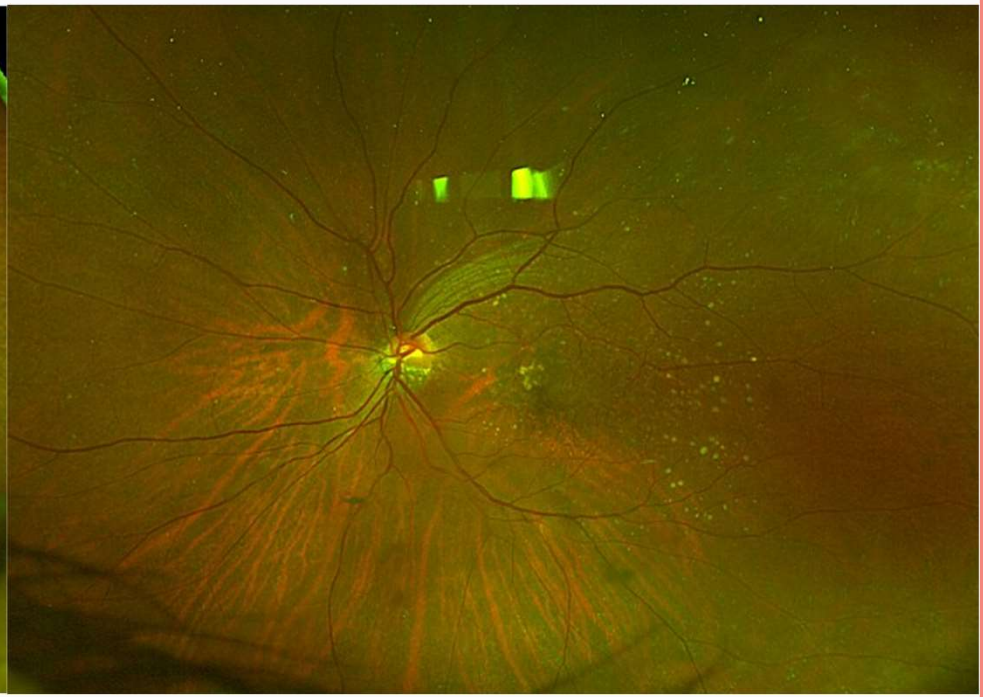
- Demographics
  - 67yo Black female
- Chief complaint
  - Blurry vision at night OU
- Entrance testing
  - All normal except distortion noted on Amsler grid
  - BCVA 20/25- OD, 20/25 OS
- IOP 19/19 mmHg OD, OS
- Medical History
  - (+) cataracts since 2023
  - Social drinker; everyday smoker
  - Hypertension (taking unknown htn medication and unknown statin), Sciatica
  - Family history positive for cataracts

A Case continued

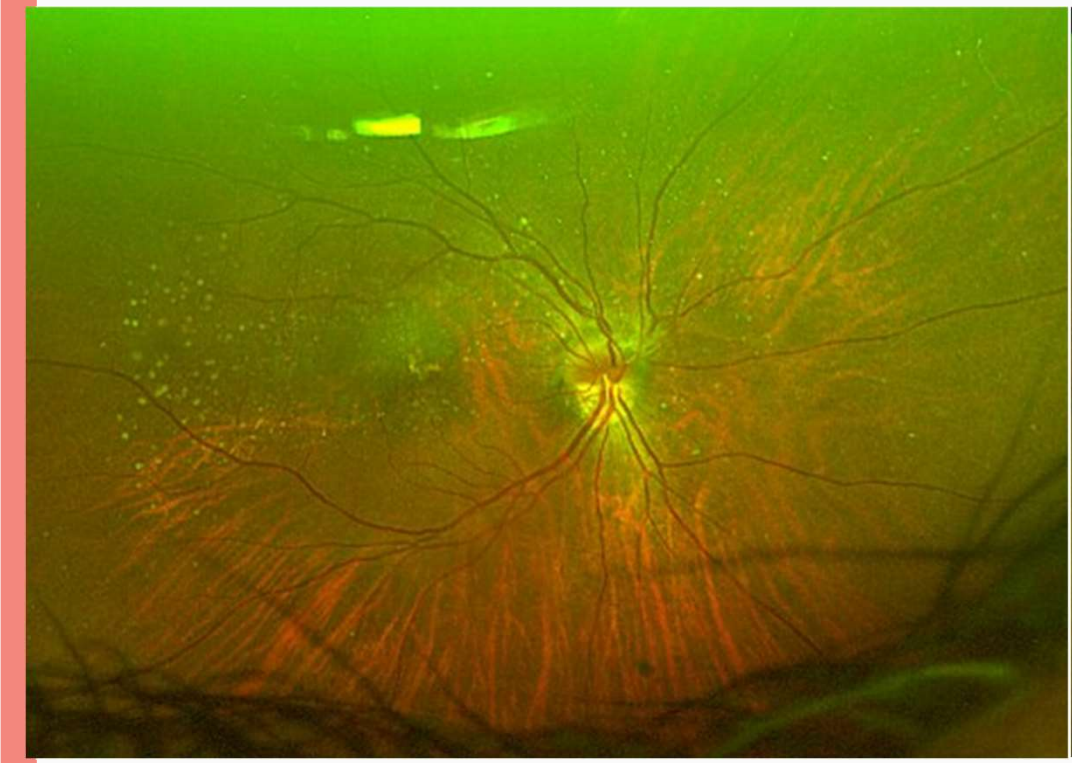
- Anterior segment: 1+ Nuclear sclerotic cataract OU
- DFE reveals tilted optic nerves OU
  - C/D 0.1 OD, OS
- **Drusen throughout posterior pole OU**
- Macula
  - Scattered hard drusen with some large coalesced drusen OU
  - Small areas RPE dropout OD
  - (-) CNV OU
- Vessels normal OU
- Peripheral retinal exam reveals cobblestone degeneration inferiorly OU
  - (-) htd 360 OU
  - (+) PVD OU

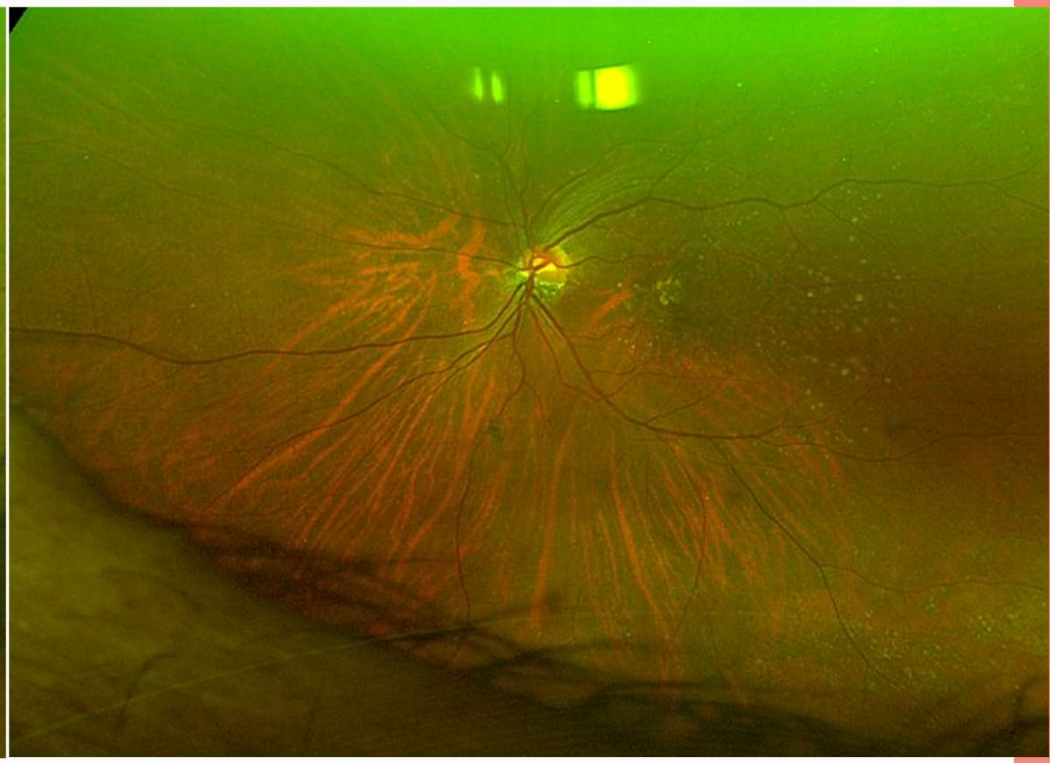
Optos (UWF) Images



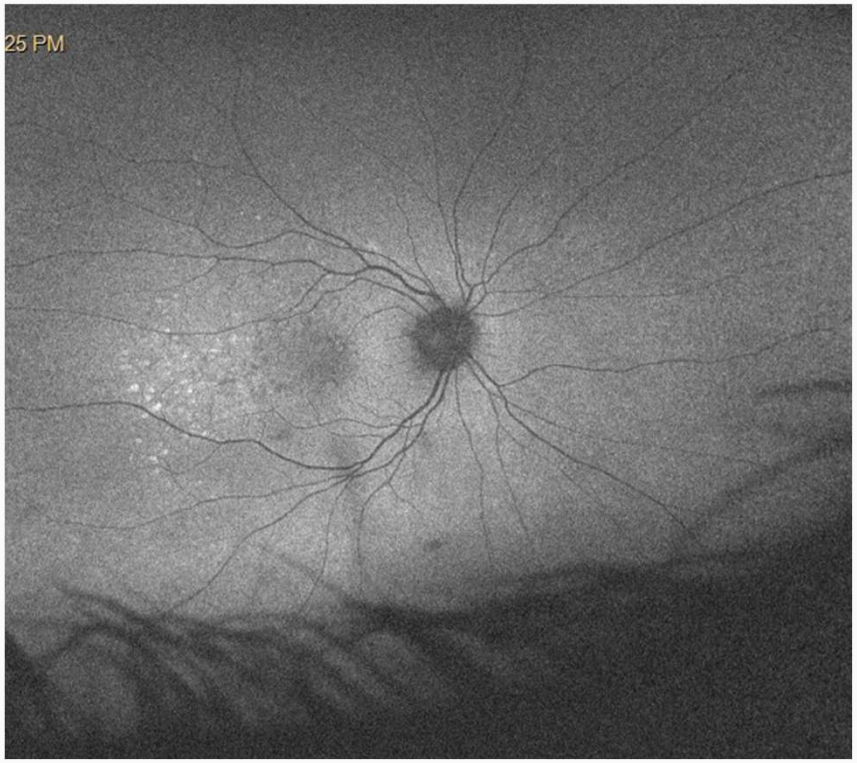


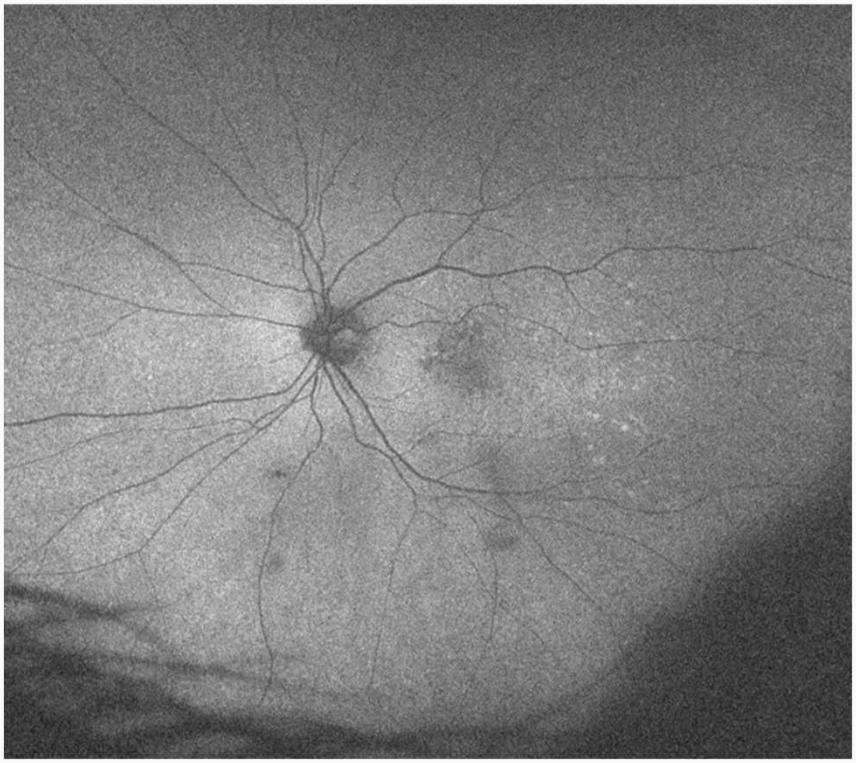
Optos (UWF) Images *2.5 years prior*



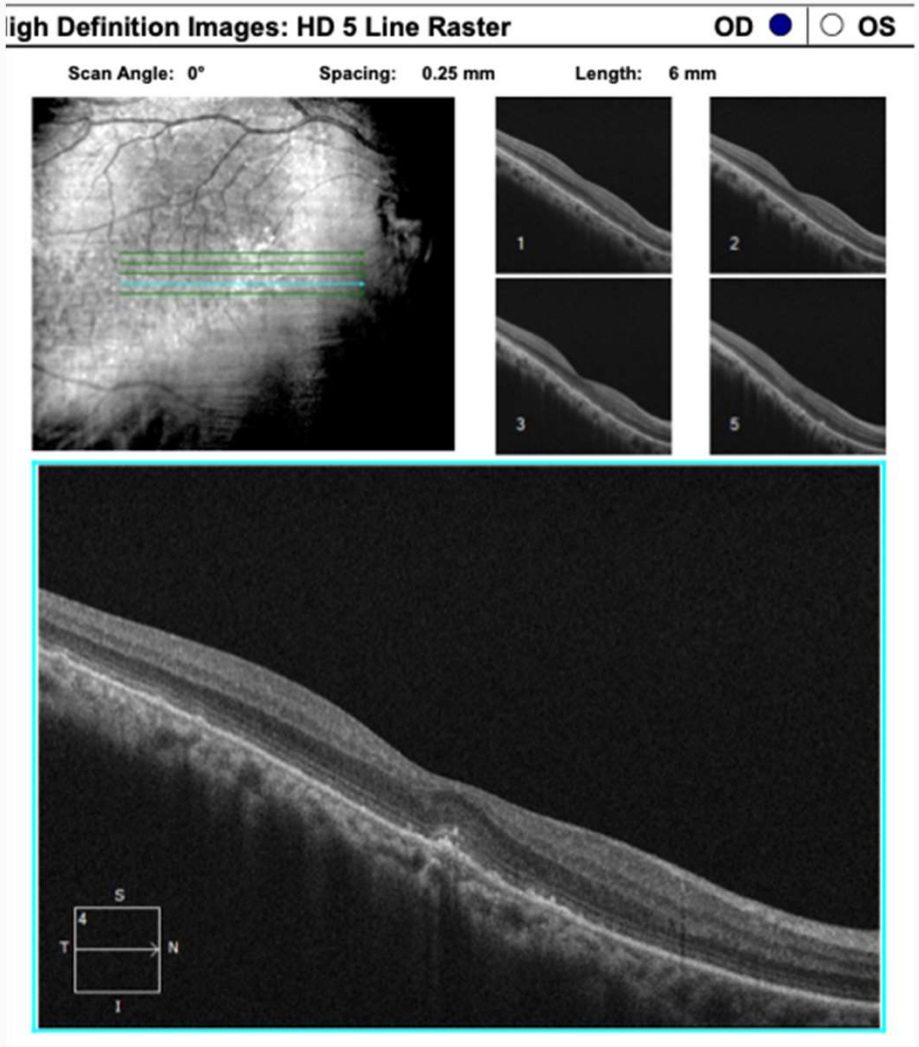


OPTOS FAF Images





OCT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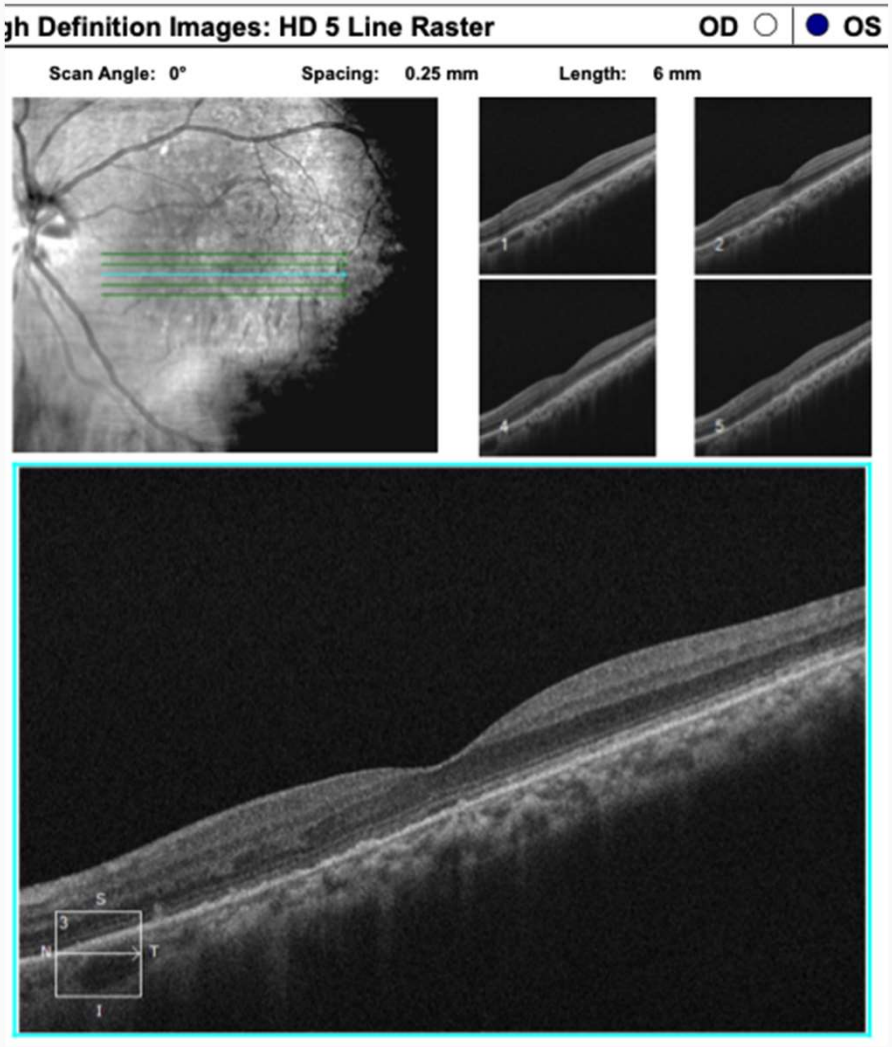
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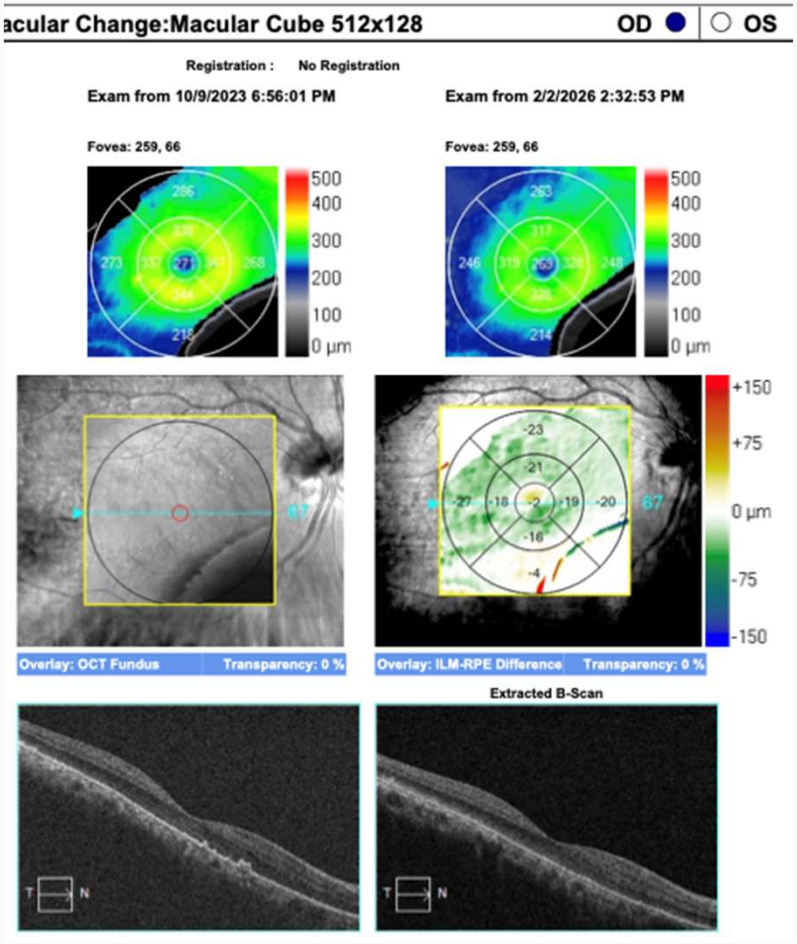
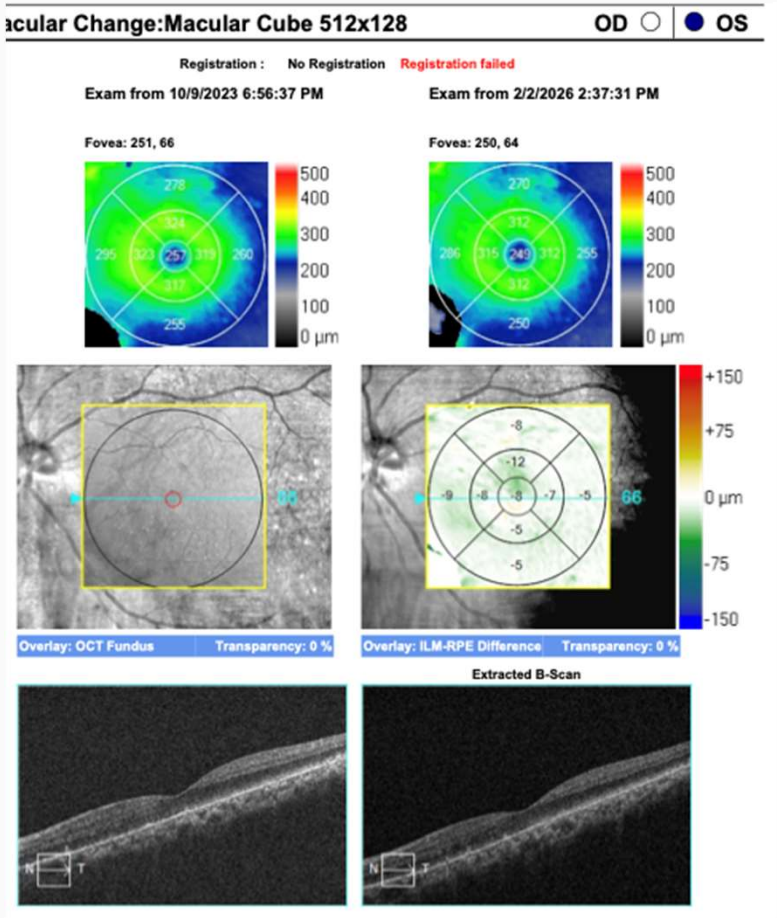
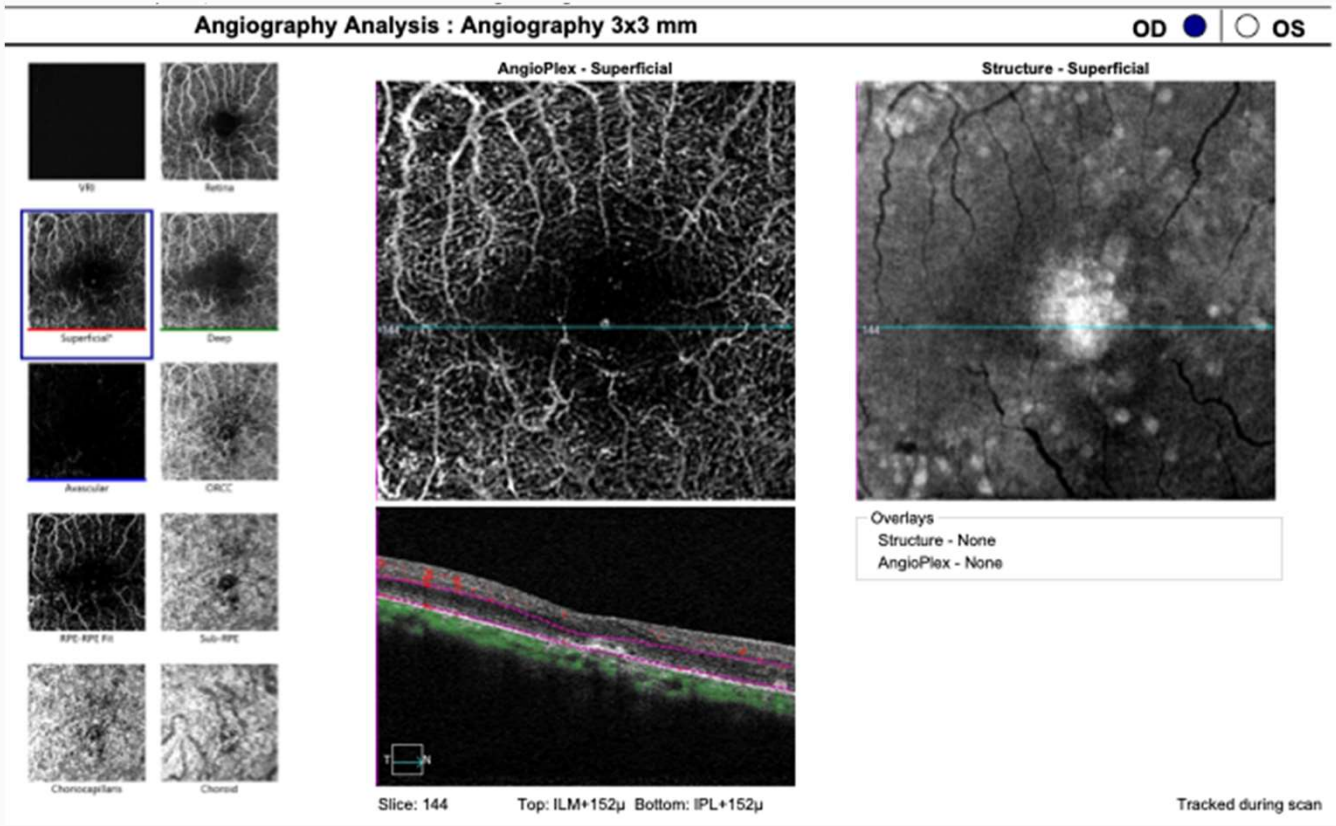
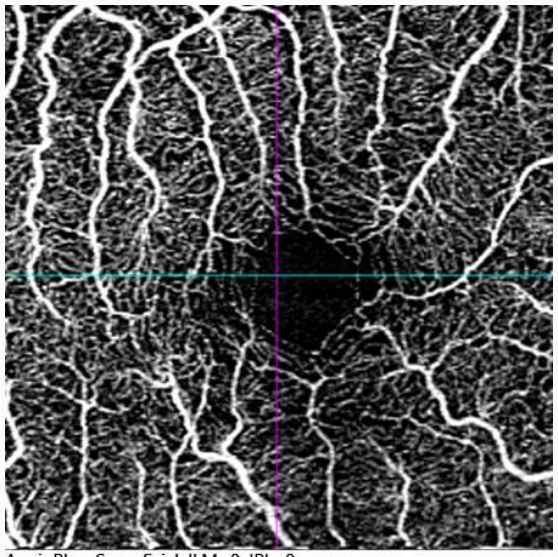
Macular change analysis

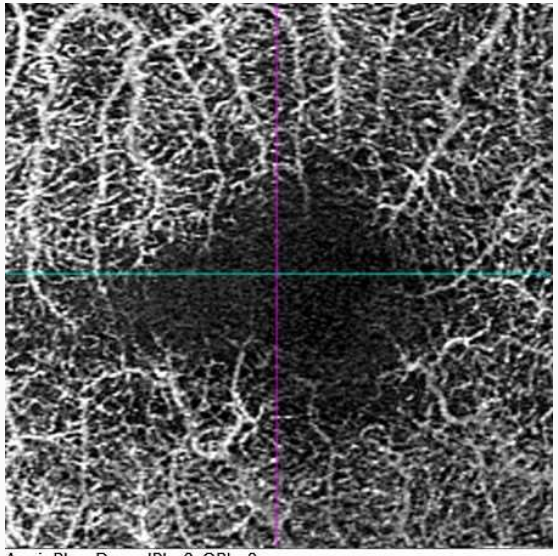






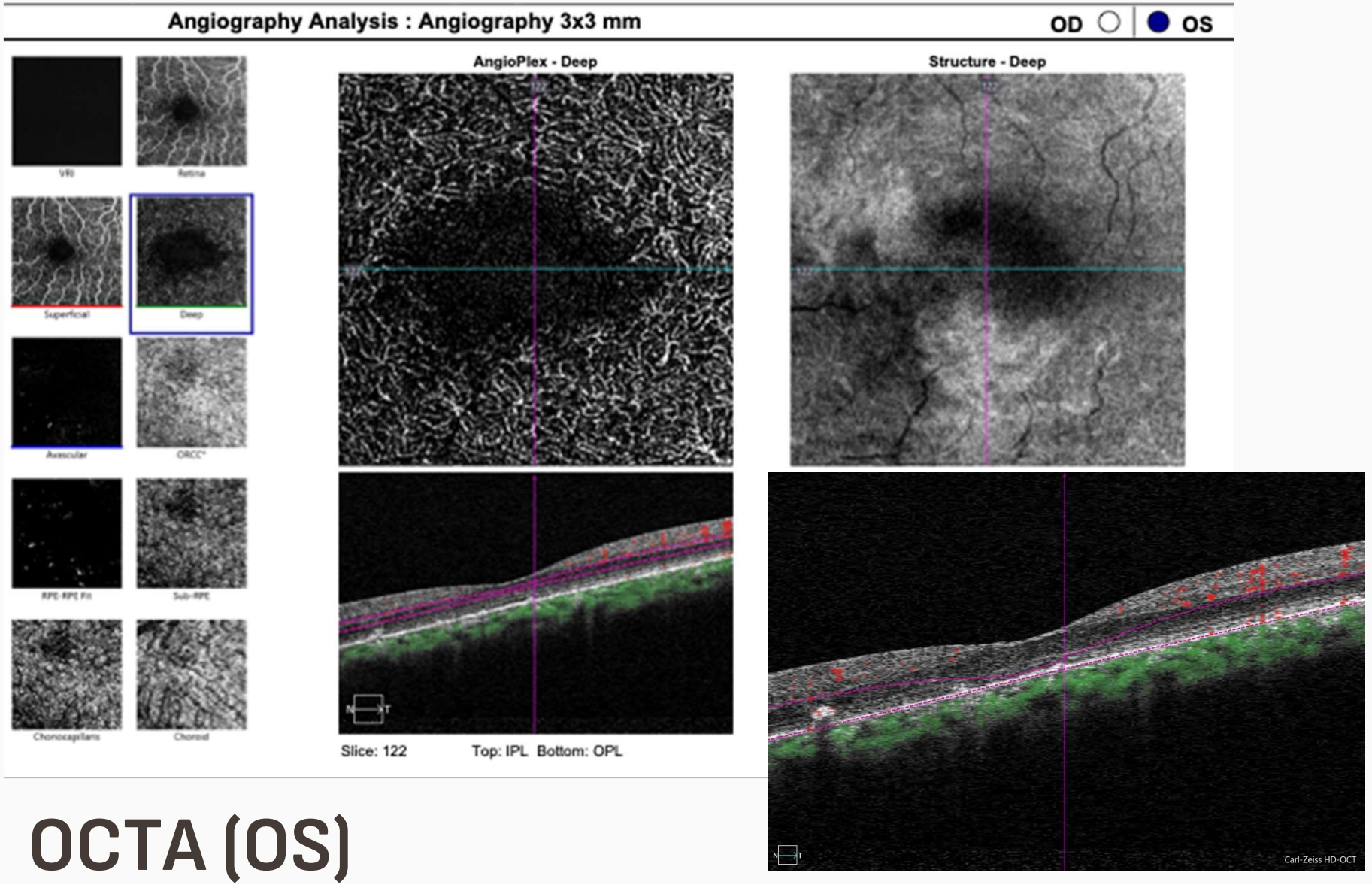


AngioPlex Superficial: ILM+0, IPL+0



AngioPlex Deep: IPL+0, OPL+0

OCTA (OD)


Angiography Analysis : Angiography 3x3 mm
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OS
AngioPlex - Deep
Structure - Deep
VRI
Retina
Superficial
Deep
Avascular
ORCC*
RPE-RPE Fit
Sub-RPE
Choriocapillaris
Choroid
Slice: 122
Top: IPL Bottom: OPL
Carl-Zeiss HD-OCT

OCTA (OS)

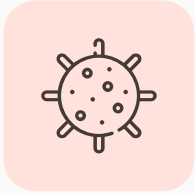
Assessment and Plan

- DDx: Polypoidal, ARMD, coalesced familial drusen

- **Diagnosis: Drusen (Degenerative) of macula, bilateral.**
  - Scattered peripheral and central drusen; familial in appearance

- Pt educated that although unlikely "true" AMD, important to monitor for any macular disruption
  - Smoking cessation
  - Option to begin AREDS II vitamins
  - Continue home Amsler grid weekly

AMD Mimics



What is it?

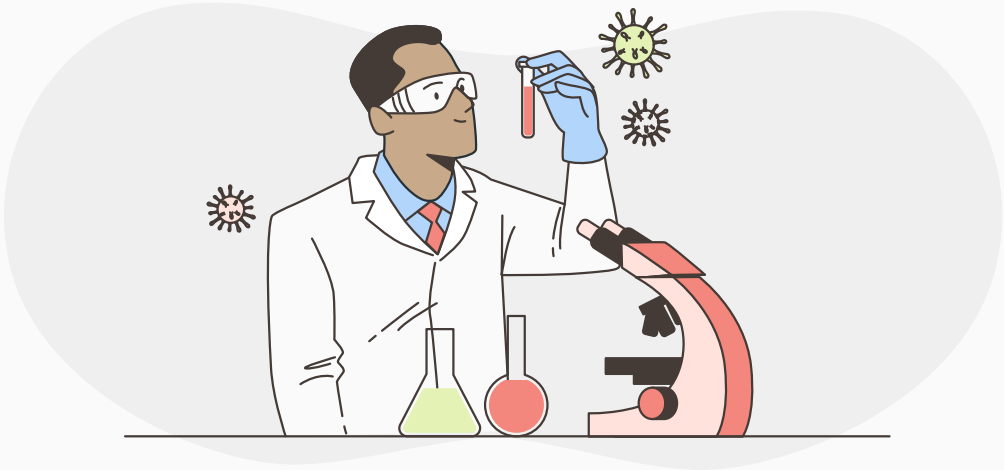
- Pathophysiology
- Retinal findings

Who gets it?

- Patient demographics

Can we treat it?

- Educating the patient



Retinal Dystrophies

PATIENT POPULATION

Refers to AMD

Patients are typically aged 50 years or older, with or without visual symptoms. Clinicians should consider the possibility of hereditary macular dystrophies in patients under 50 years of age who have clinical features that resemble AMD.

Retinal Dystrophies

Drusen

- Doyne Honeycomb Retinal Dystrophy
- Autosomal Dominant Drusen
- Malattia Leventinese

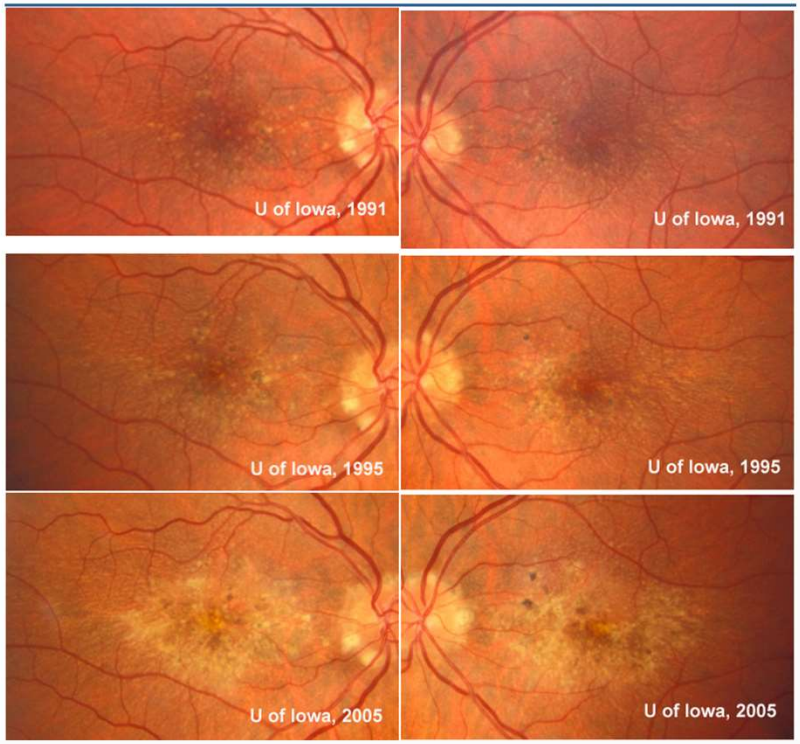
Pattern Dystrophies

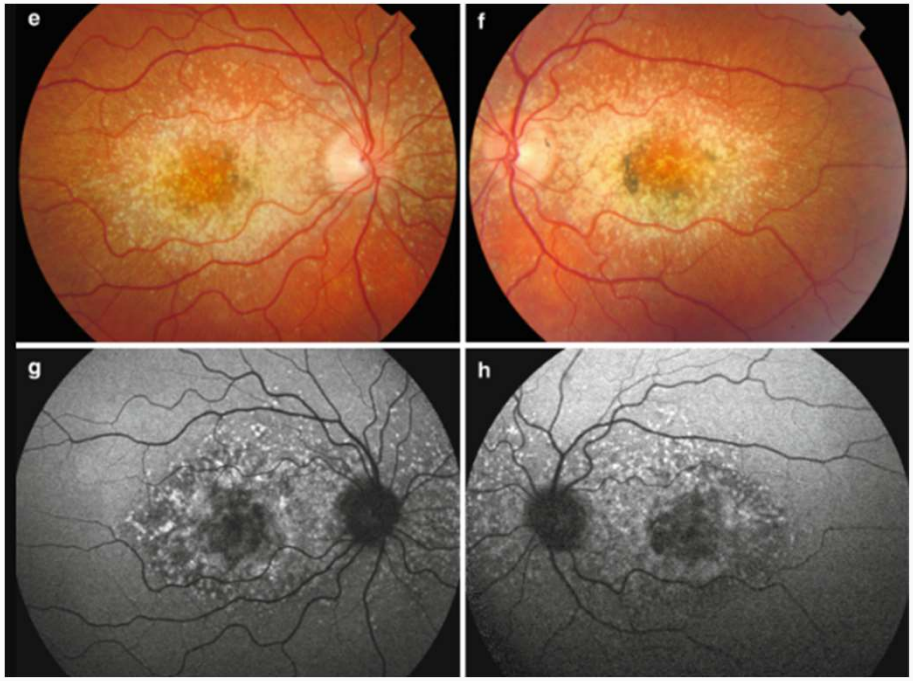
- Adult-Onset Foveomacular Vitelliform Dystrophy
- Fundus Pulverulentus
- Multifocal Pattern Dystrophy Simulating Stargardt Disease
- Reticular Dystrophy
- Butterfly-shaped pattern dystrophy

What is it?

- Malattia Leventinese aka Doyne Honeycomb Retinal Dystrophy aka Autosomal Dominant Drusen
  - Pathophysiology: Autosomal dominant mutation in the EFEMP1 gene

Fundus Findings	OCT Findings
• Large central and peripapillary drusen with smaller radially-oriented drusen temporal to the macula • Notably found with drusen *nasal to the ONH • Can have associated macular atrophy and CNVM	• Hyperreflective thickening of the retinal pigment epithelium–Bruch membrane complex • Dome-shaped or saw-toothed hyperreflective elevations at the level of RPE/Bruch's





Hereditary Chorioretinal Disorders

Who gets it?

- Autosomal Dominant Drusen: Symptomatic at age 30-50

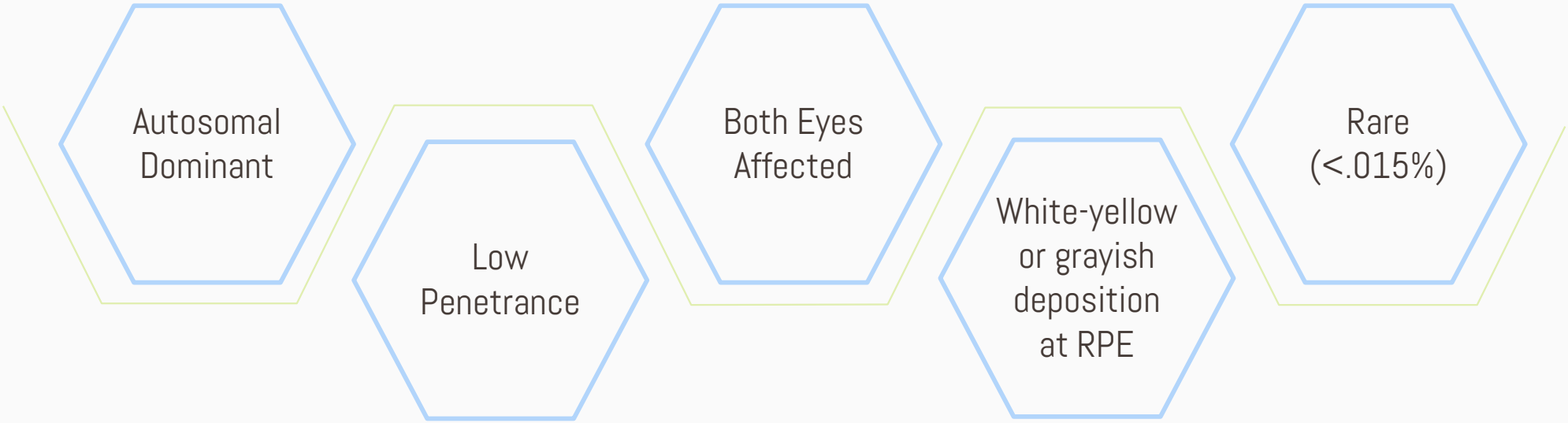
Numbers and Sex Distributions for Inherited Macular Dystrophies With Results of Two-tailed Binomial Testing

Genetic Subgroup	No. of Patients			Female % (95% CI)	*P* Value for Imbalance
	Total	Males	Females		
BEST1					
Dominant	115	71	44	38.3 (29.4–47.8)	0.015
Recessive	37	20	17	46.0 (29.5–63.1)	0.743
EFEMP1	35	8	27	77.1 (59.9–89.6)	0.0019*
PROM1 (Dominant)	30	16	14	46.7 (28.3–65.7)	0.467
PRPH2 (172 142)	50	25	25	50.0 (35.5–64.5)	1
RP1L1 (Dominant)	14	7	7	50.0 (23.0–77.0)	1
TIMP3	44	17	27	61.4 (45.5–76.5)	0.174

Sex Distributions in Non-ABCA4 Autosomal Macular Dystrophies - PMC

What is it?

- **Pattern dystrophies:** Likely result from mutations in the PRPH2 or BEST1 gene
  - Many different phenotypes, each with its own retinal presentation



Who gets it?

- Pattern dystrophies
  - Adult-onset foveomacular vitelliform dystrophy: onset **age 30-50**
  - Butterfly-shaped pattern dystrophy: **late 20s – early 30s**
  - Multifocal Pattern Dystrophy Simulating Stargardt Disease: presentation **30s-50s**
  - Reticular dystrophy: symptomatic in **40s**
  - Fundus Pulverulentus: **age 30-50**

Most common

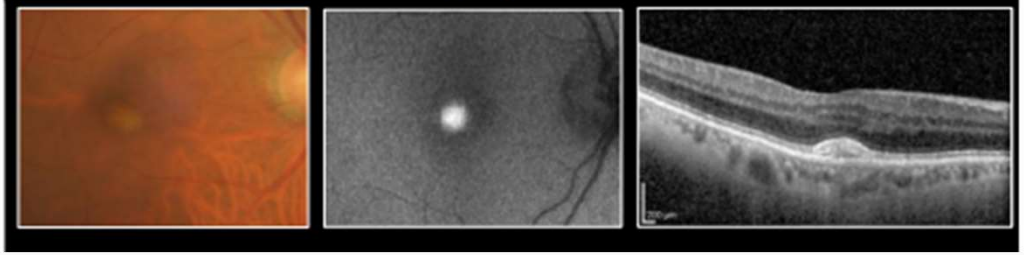
Least common

Genetic Subgroup	No. of Patients			Female % (95% CI)
	Total	Males	Females	
PRPH2 (172 142)	50	25	25	50.0 (35.5–64.5)

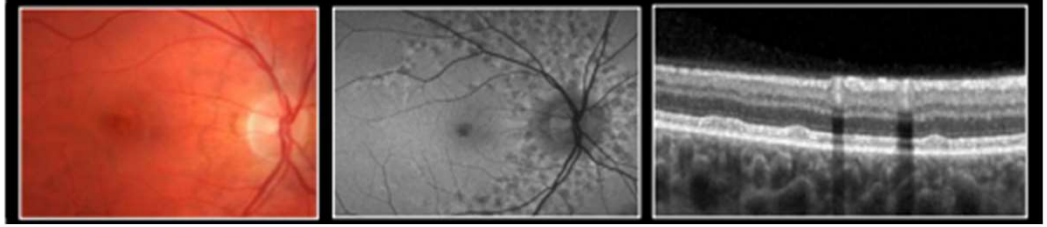
Dystrophy	Typical Age of Onset	Typical VA
Adult-Onset Vitelliform Dystrophy	4th-6th decades	20/30-20/60
Butterfly-Shaped Pigment Dystrophy	2nd-5th decades	20/20-20/25
Reticular Dystrophy	5th decade	20/30-20/70
Multifocal Pattern Dystrophy	4th-6th decades	20/20-20/40
Fundus Pulverulentus	4th-5th decades	20/20-20/40

Pattern Recognition: How to Identify and Confirm Multifocal Pattern Dystrophy

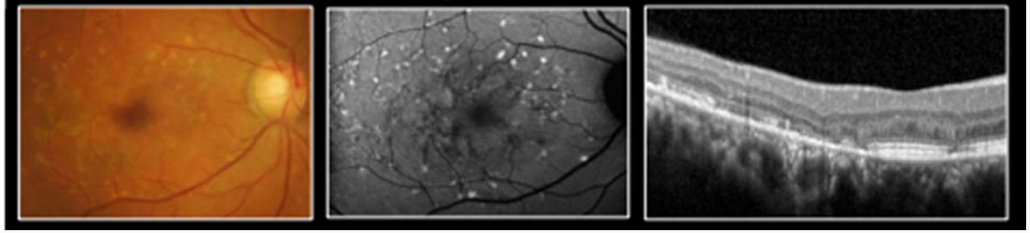
Adult-Onset Foveomacular Vitelliform Dystrophy



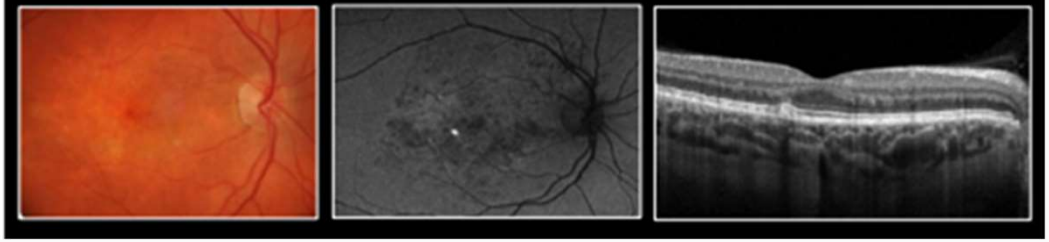
Reticular Dystrophy of the RPE



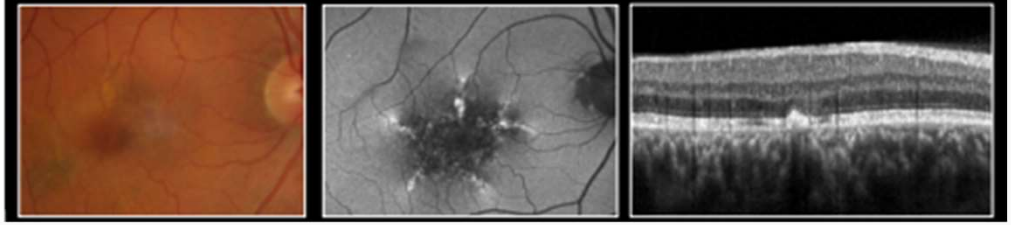
Multifocal Pattern Dystrophy simulating Stargardt



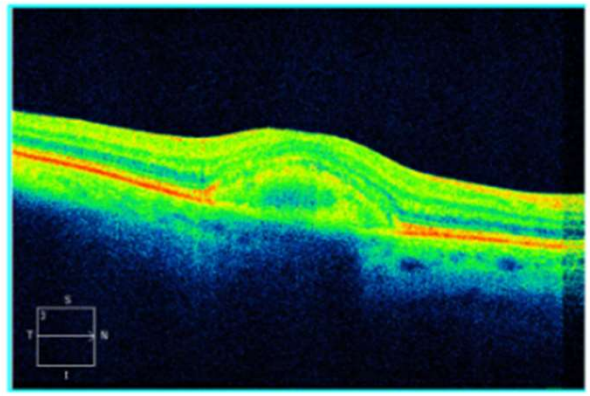
Fundus Pulverulentus



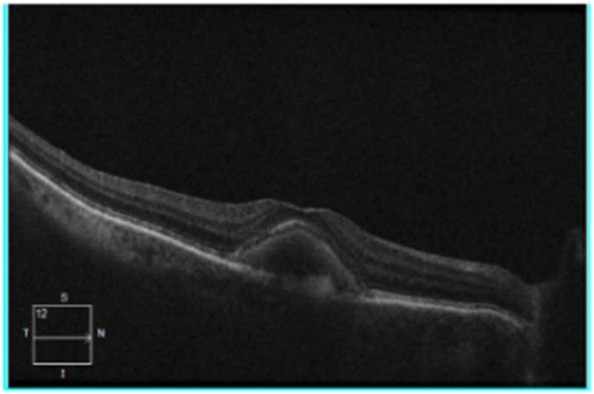
Butterfly Pattern Dystrophy



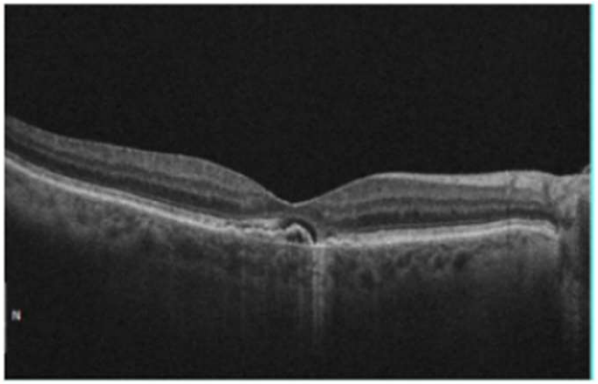
	Adult-onset Foveomacular Vitelliform Dystrophy	Butterfly-shaped Retinal Dystrophy	Multifocal Pattern Dystrophy Simulating Stargardt Disease	Reticular Dystrophy	Fundus Pulverulentus
Fundus Findings	• "egg yolk" appearance • Pigment clump with hyperpigmented haloes	• "butterfly shaped" yellow deposits central macula with spokes • Yellow flecks in posterior pole	• scattered yellow/white macular deposits (resembling flecks)	• "network of hyperpigmentation resembling a fishing net"	• Multiple irregular pigment clumps • Granular macular appearance • No drusen
OCT Findings	• dome-shaped subretinal hyperreflective material	• Subfoveal hyperreflective deposits at the level of the RPE • Retinal thickness remains intact	• Disruption of EZ and RPE	• Multiple small hyperreflective areas at the RPE • EZ loss or disruption	• Hyperreflective lesions at the RPE (associated with pigment clumping)
FAF Findings	• Lesion is hyper-autofluorescent	• Pigmentation shows increased autofluorescence, surrounded by hypoautofluorescence	• Deposits are hyper-autofluorescent with surrounding hypo-autofluorescence	• ?mixture of hyper and hypo-autofluorescence in the reticular pattern	• Mottled appearance with hyper- and hypo-autofluorescence



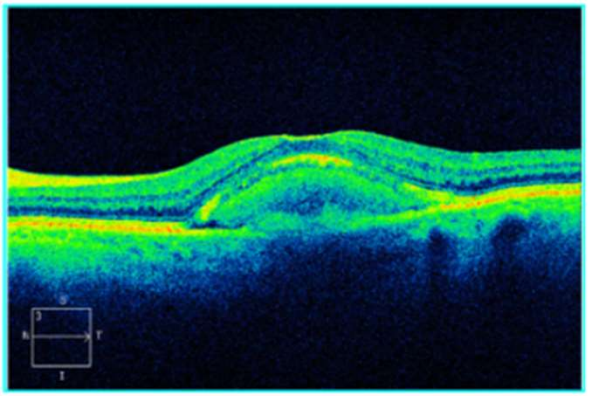
2/21/2017

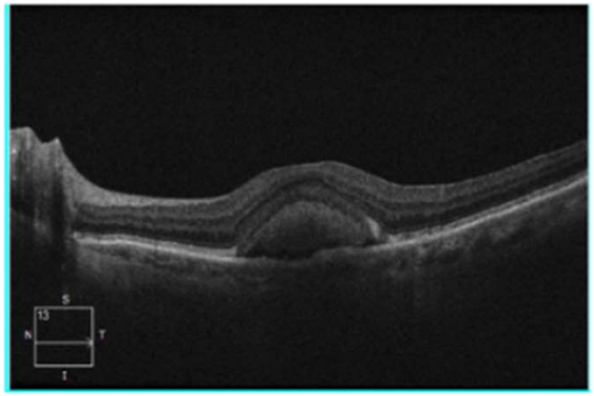


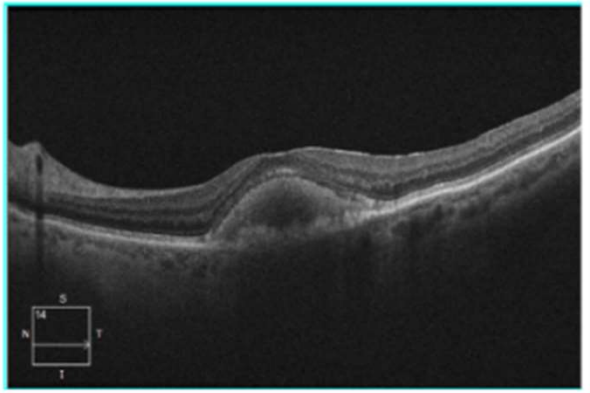
3/30/2017



8/23/2018







What is the visual prognosis?

Autosomal Dominant Drusen

Visual prognosis is *variable*

14/44
asymptomatic

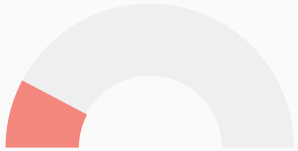
15/44
with CNVM

Visual acuity progression (over 8 years):

~20/30 – 20/40 → ~20/60 – 20/80

Pattern Dystrophies

Visual prognosis generally good
Dystrophy-dependent



18%
CNVM

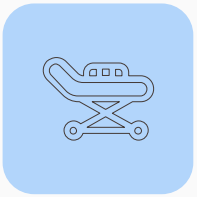
26%
GA

Can we treat it?

CNVM

- Autosomal Dominant Drusen
- Any Pattern Dystrophy

- Treated with anti-VEGF

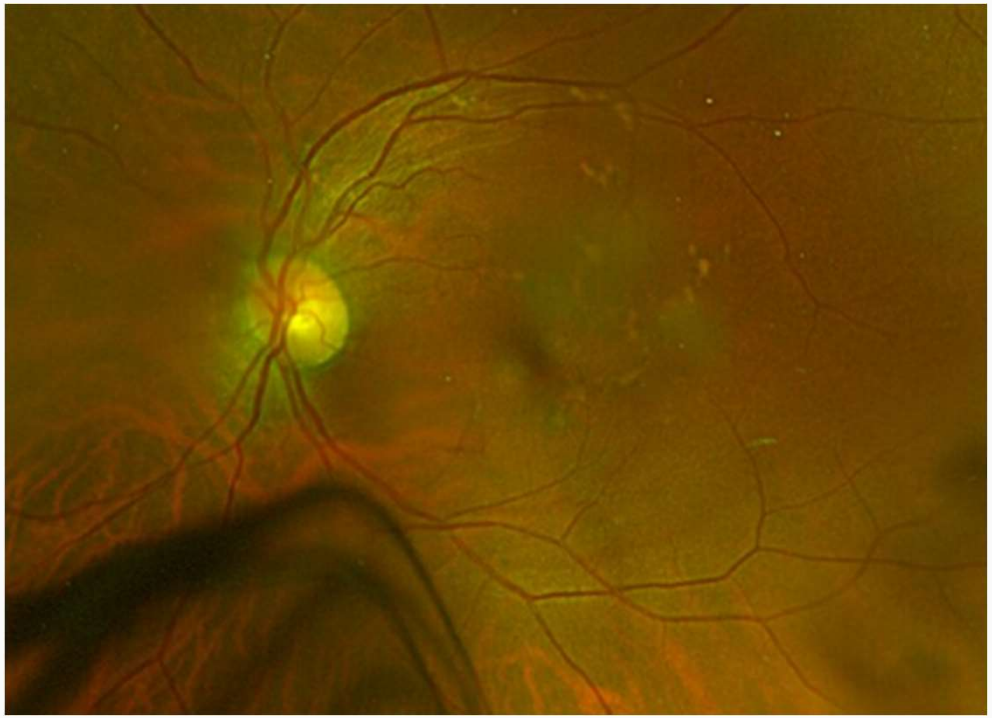


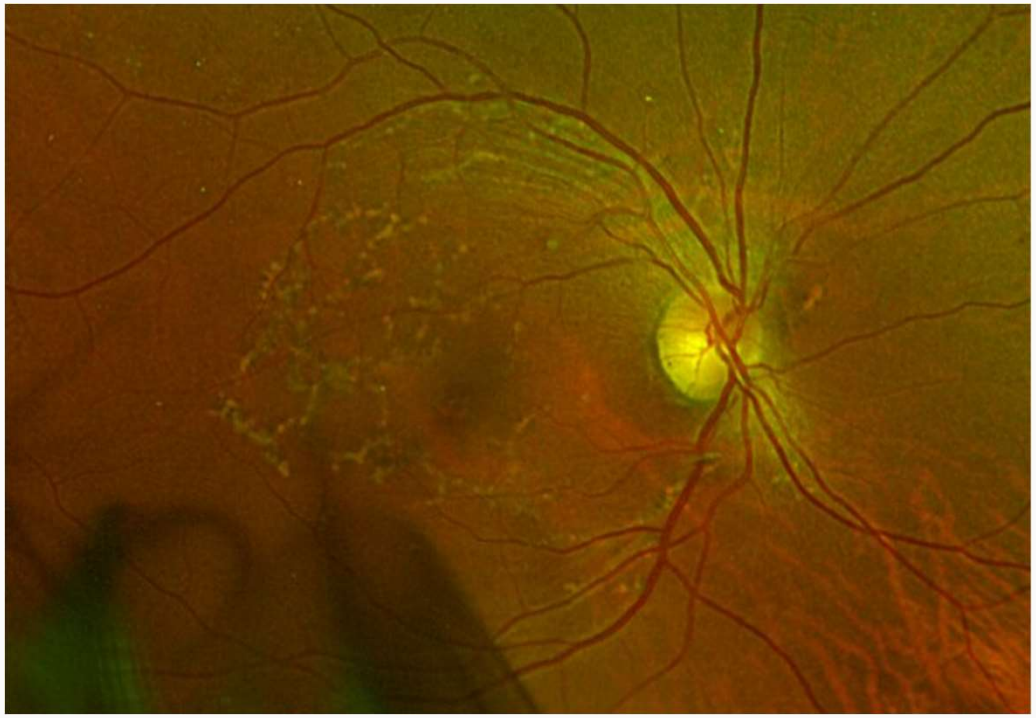
Macular Holes

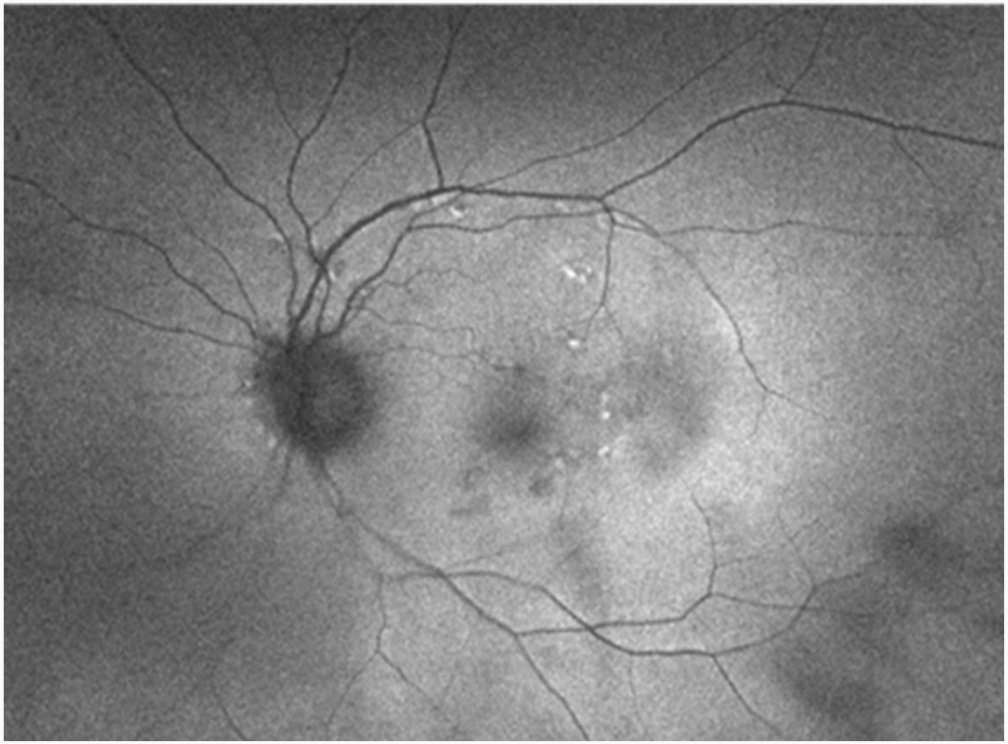
- Can occur in AOFVD
- Limited response to surgical intervention

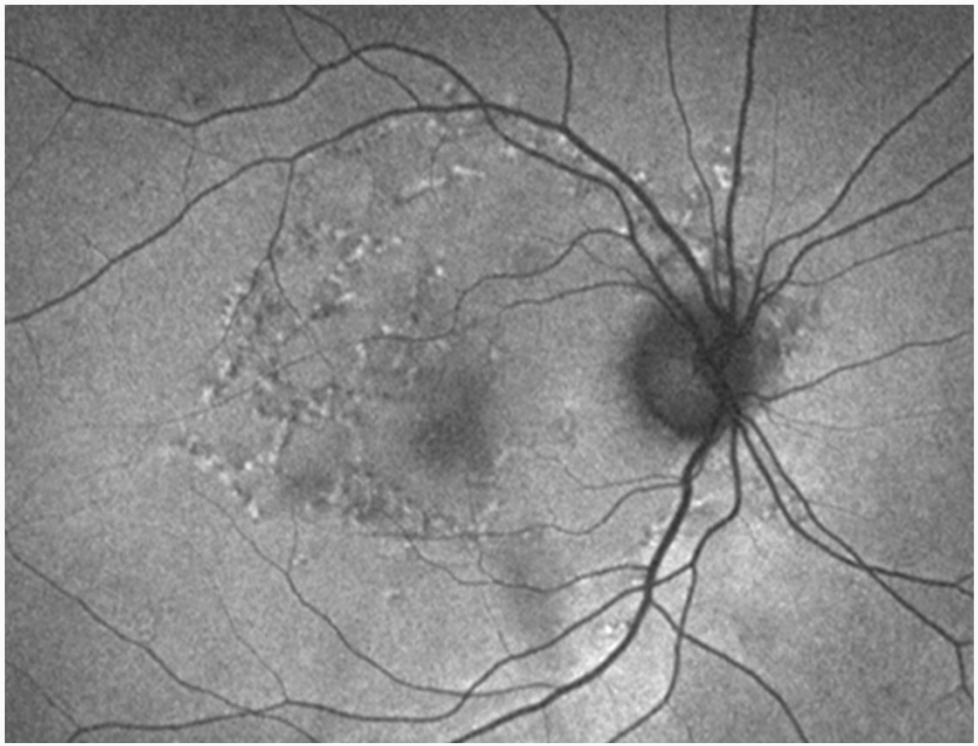
Another case

- 70-year-old Black female
- CC: “Noticing a break in pictures and images appear distorted”; OD>OS; other symptoms: floaters, wavy lines in vision
- BCVA: 20/25+ OD, 20/20-3 OS
- IOP: 9/9
- Anterior segment: mild NS and cortical cataracts OU
- Posterior segment….

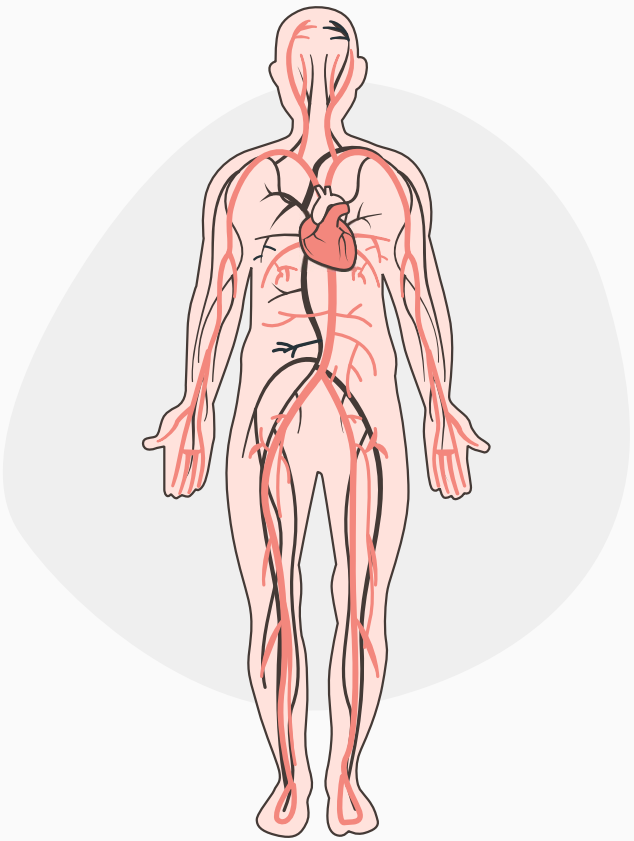








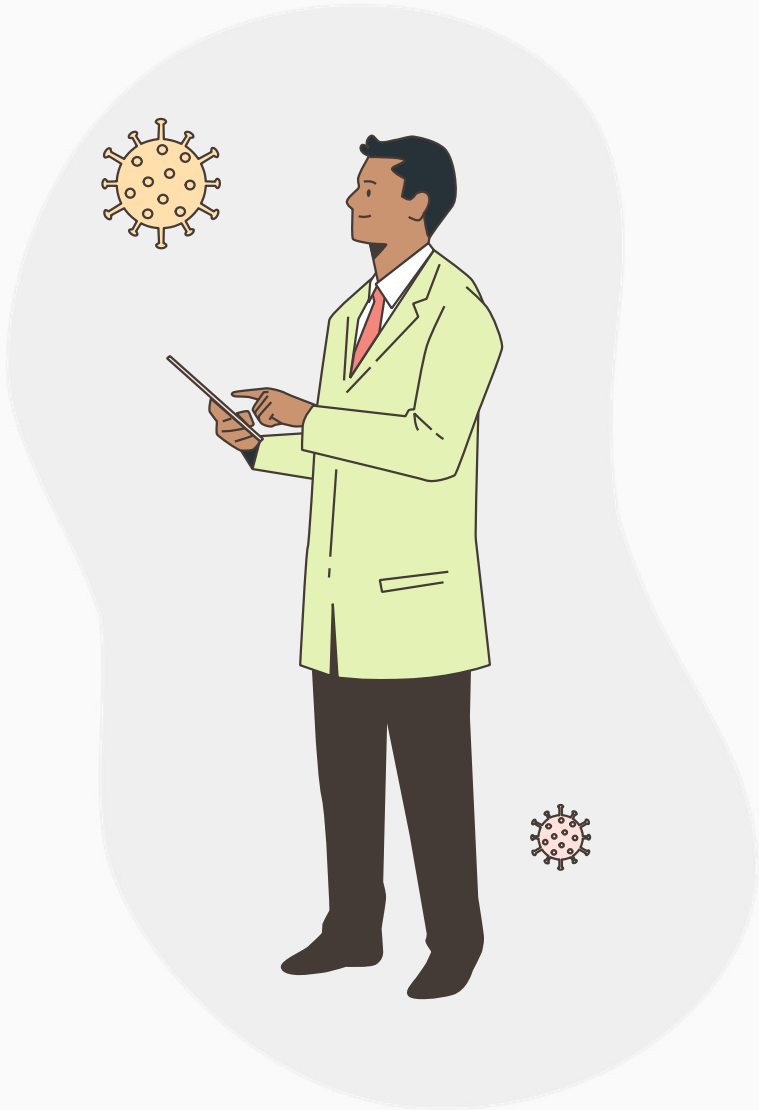
Vascular Anomalies



Pachychoroid Spectrum

- Pachychoroid Pigment Epitheliopathy
- Polypoidal Choroidal Vasculopathy
- Pachychoroid Neovasculopathy

Macular Telangiectasia Type II



Pachychoroid Spectrum

What is it?

- Pachychoroid Spectrum: **"thickened choroid"**
  - Thickened Haller layer, thinned Sattler layer and choriocapillaris
  - **Pachychoroid pigment epitheliopathy**
    - RPE changes + pachychoroid, *no soft drusen* or subretinal fluid
    - Usually asymptomatic
  - **Pachychoroid neovasculopathy**
    - Type 1 neovascularization + pachychoroid; *minimal to no drusen*
  - **Polypoidal choroidal vasculopathy**
    - Type 1 neovascularization + polypoidal lesions + pachychoroid

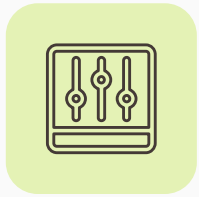
Who gets it?

Pachychoroid pigment epitheliopathy

- Age: average 55yo

Pachychoroid neovasculopathy

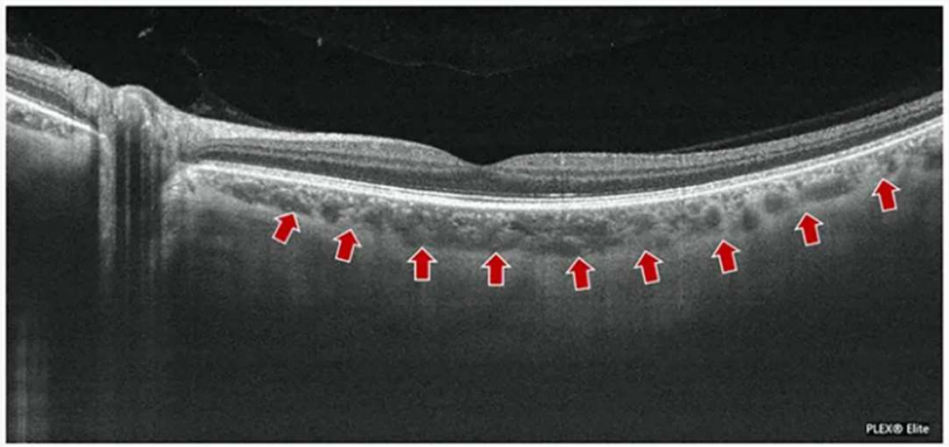
- Age: mid 50s–mid 60s
- Males > Females



Polypoidal choroidal vasculopathy

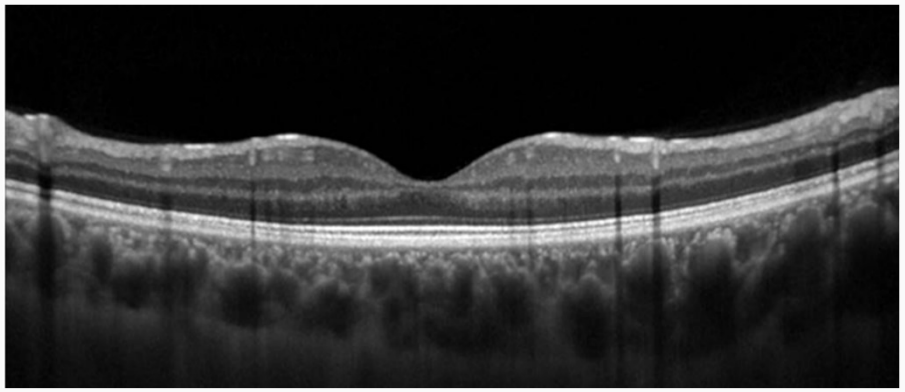
- Age: average 69yo
- Males > females
- Higher prevalence in patients of Asian and African ethnicity

How do we diagnose it?

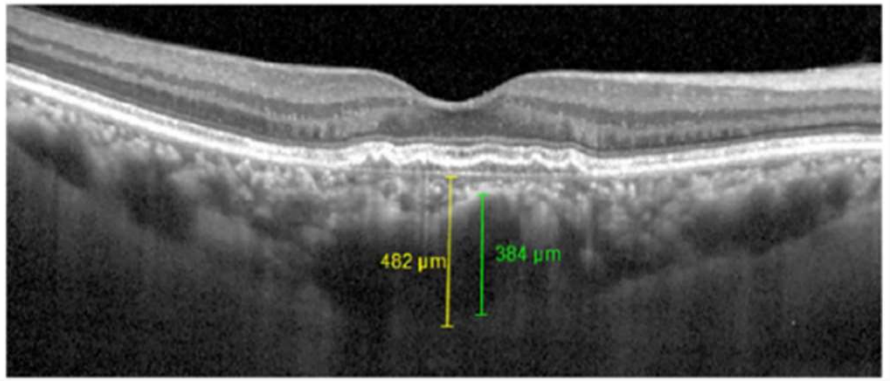


Frontiers | Evaluation of Choroidal Thickness Using Optical Coherent Tomography: A Review

Enhanced Depth Imaging (EDI)



Pachychoroid Spectrum Diseases | OCT Club



(PDF) Short-term effect of anti-VEGF for chronic central serous chorioretinopathy according to the presence of choroidal neovascularization using optical coherence tomography angiography

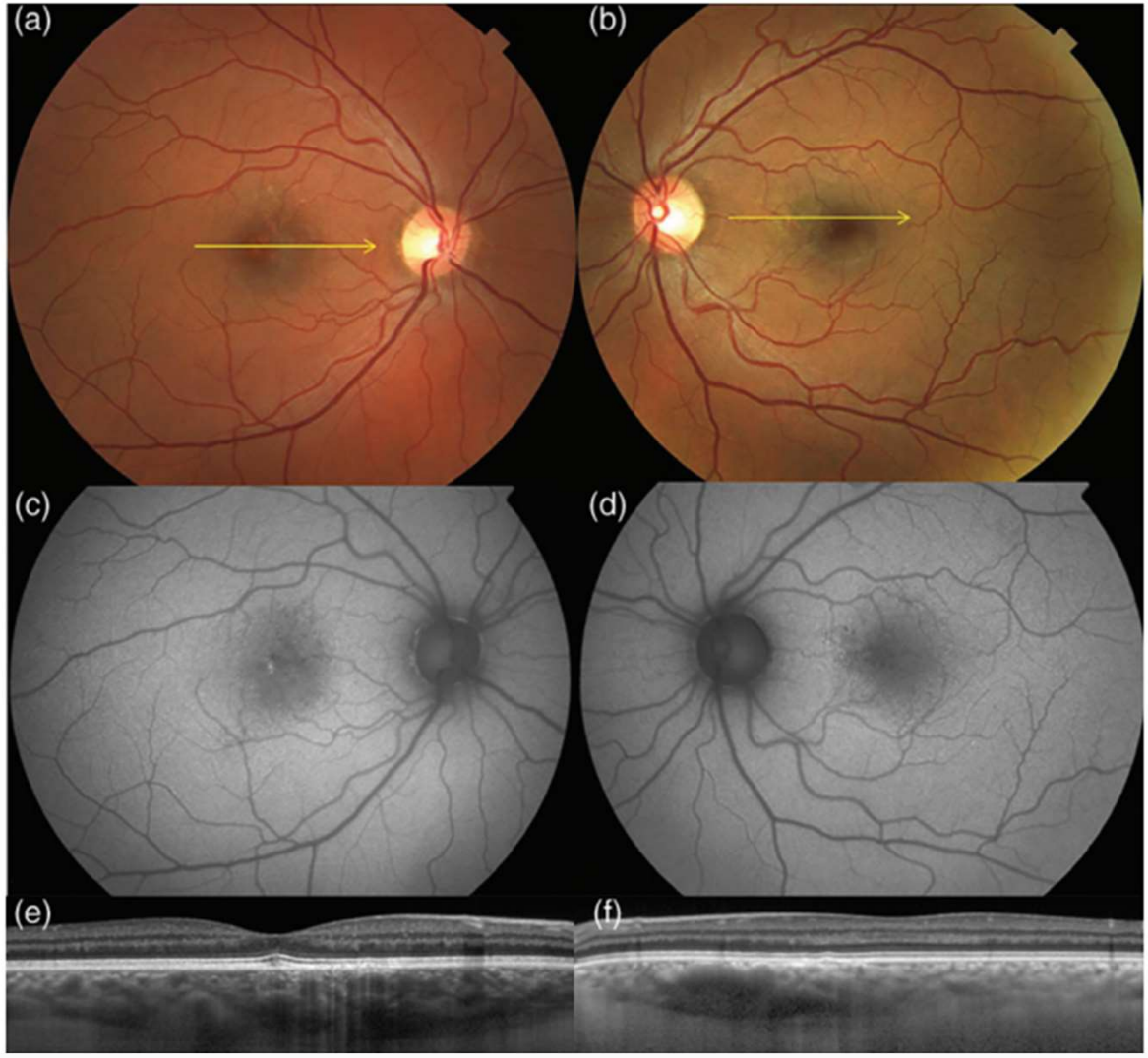
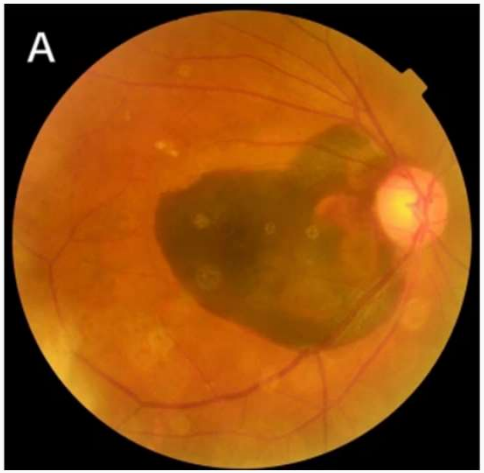
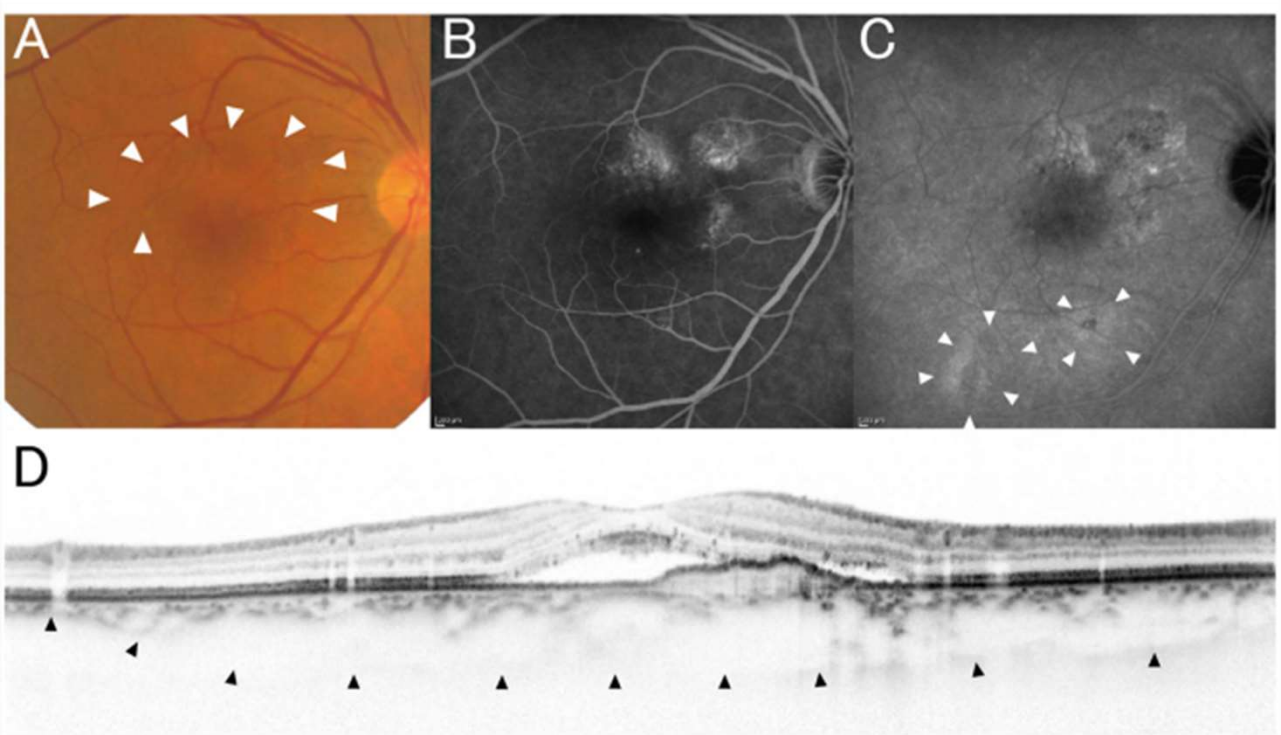


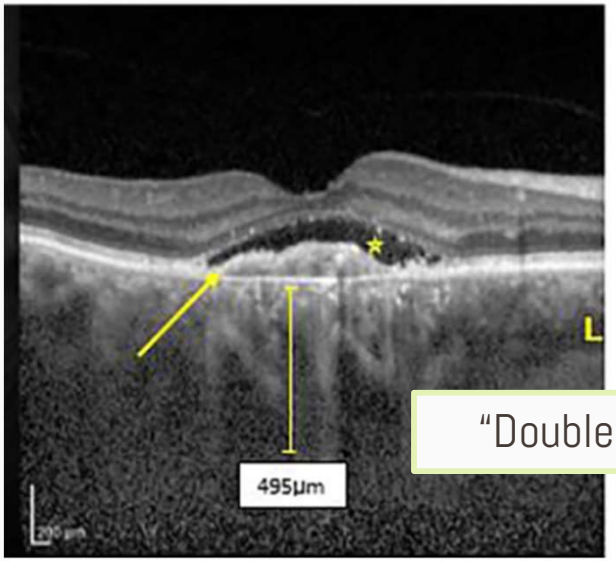
Figure 5. Pachychoroid pigment epitheliopathy in a 37-year-old white male. Colour photographs of the right (a) and left (b) eyes show absent drusen and reduced fundus tessellation. Fundus autofluorescence of the right (c) and left (d) eyes show non-specific pigment epithelial changes, including a small focus of hyperautofluorescence at the right fovea (c). Spectral domain optical coherence tomography of the right (e) and left (f) eyes shows thick choroids in both eyes. In the left eye (f), a region of extrafoveal maximal choroidal thickness is attributable to Haller's layer vessel dilatation, anterior to which the inner choroid is attenuated.



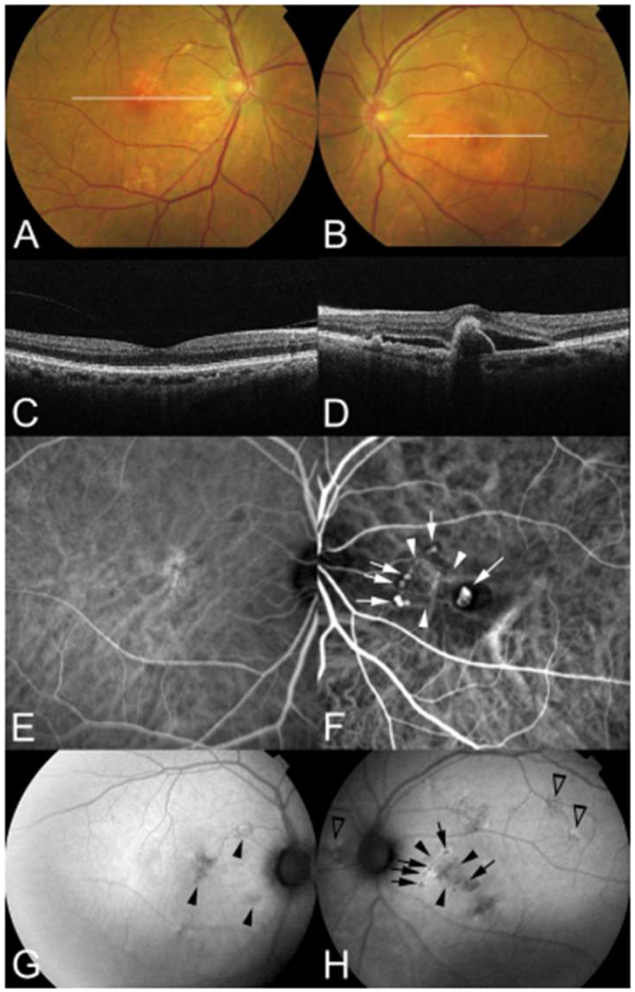
(PDF) Pachychoroid neovasculopathy can mimic wet type age-related macular degeneration



Pachychoroid neovasculopathy and age-related macular degeneration - PMC



"Double-Layer Sign"



Fundus Autofluorescence in Polypoidal Choroidal Vasculopathy - Ophthalmology

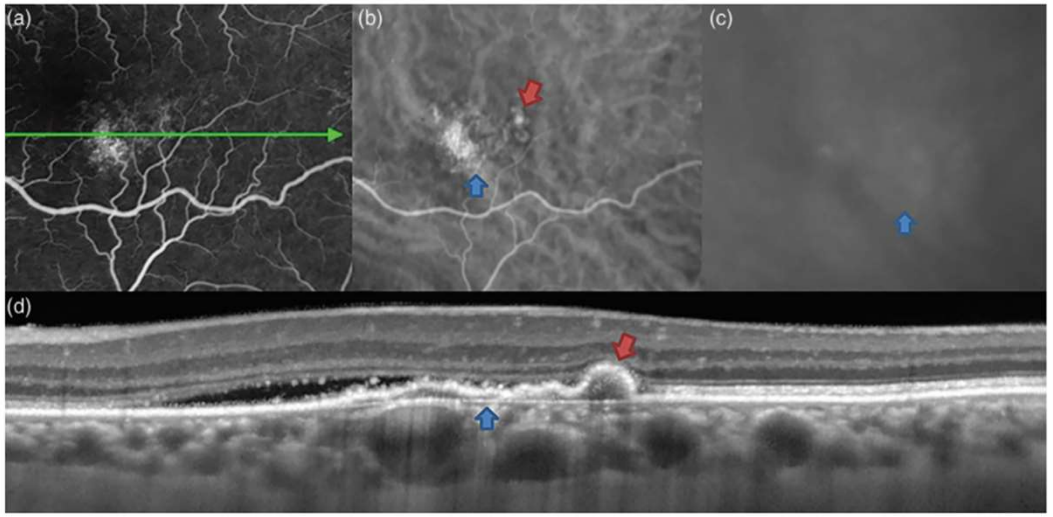
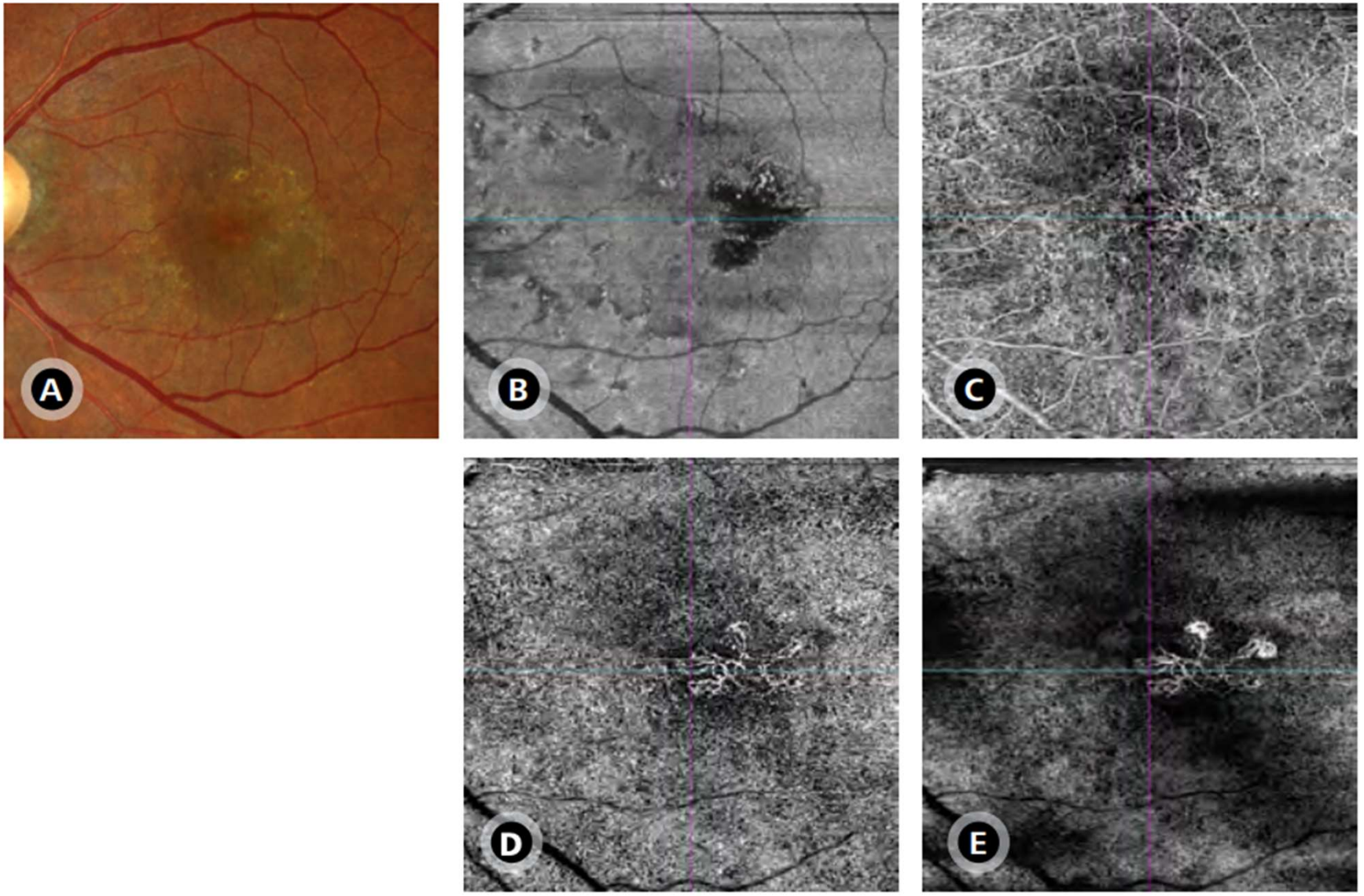


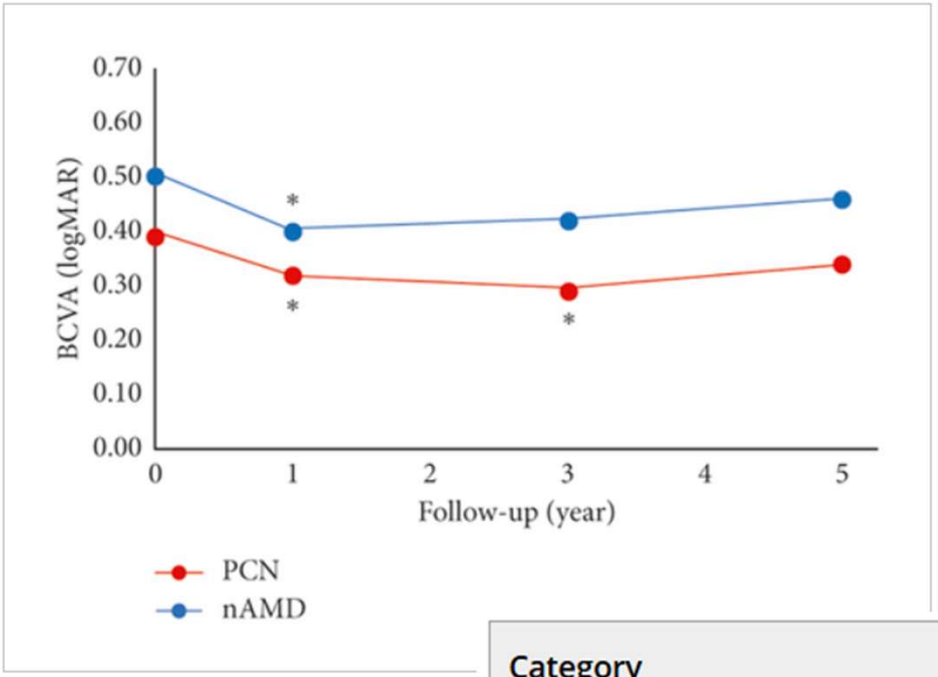
Figure 2. Aneurysmal type 1 neovascularization (polypoidal choroidal vasculopathy) in a 60-year-old white female. Mid-phase fluorescein angiography (a) shows non-specific hyperfluorescence inferotemporal to the fovea, consistent with 'occult' choroidal neovascularization. Mid- and late-phase indocyanine green angiography findings (b, c) show a hyperfluorescent plaque (blue arrow) at this focus. In the transit phase, an aneurysmal (polypoidal) lesion is also noted (red arrow). Spectral domain optical coherence tomography (d) shows subretinal fluid adjacent to pigment epithelial detachment (PED). The PED has a shallow irregular component (blue arrow), corresponding to type 1 neovascularization, and a peaked component (red arrow) at the site of the aneurysmal (polypoidal) lesion. The choroid is relatively thick due to dilated Haller's layer vessels, anterior to which the inner choroid is attenuated.


A
B
C
D
E

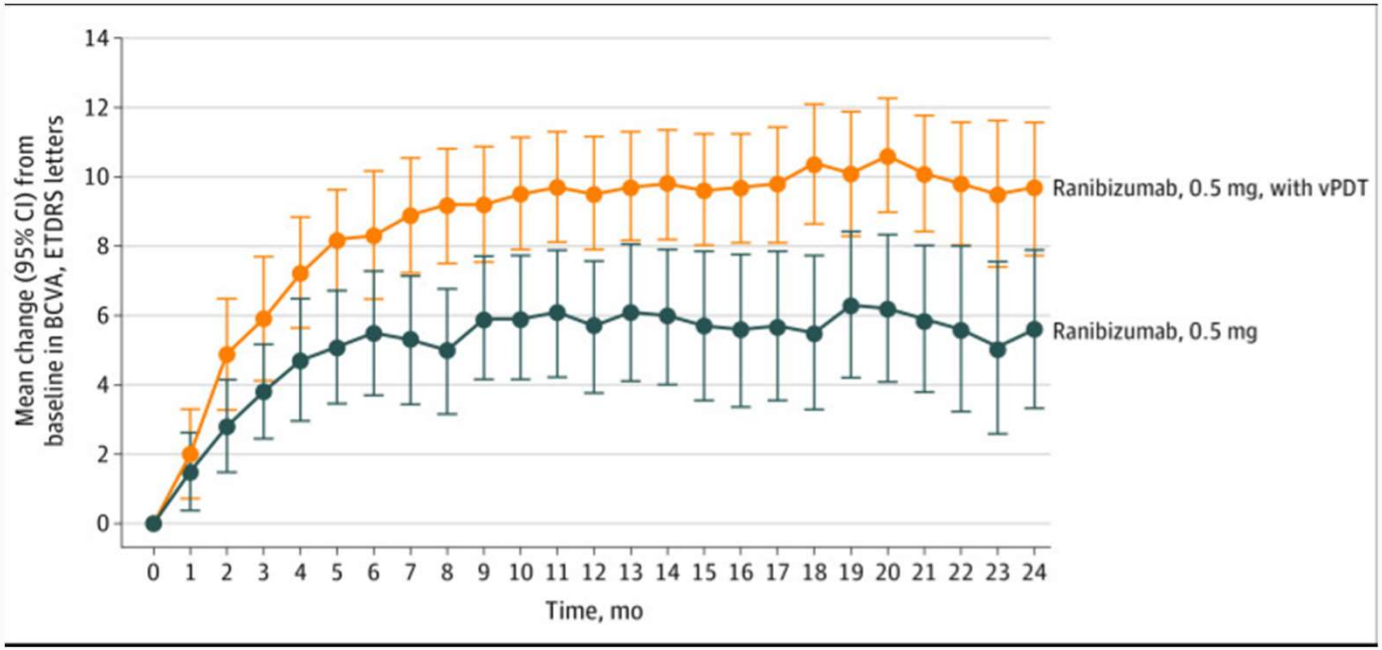
Can we treat it?

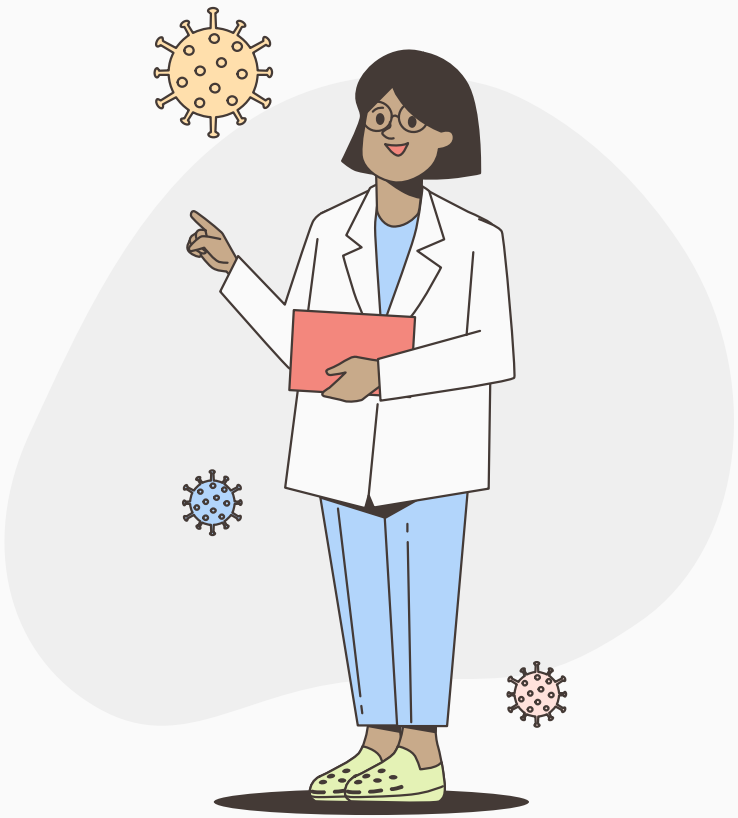
- Pachychoroid pigment epitheliopathy
  - Prognosis is good!
  - 95% of patients kept 20/30 or better acuity at 7 years after diagnosis
- Pachychoroid neovasculopathy
  - Anti-VEGF (effective)
  - PDT (+/- anti-VEGF)
- Polypoidal choroidal vasculopathy
  - Anti-VEGF monotherapy (noted resistance)
  - Anti-VEGF + PDT

If we treat this the same as neovascular AMD, why does the diagnosis matter?



Category	PCN group	nAMD group	*p* value
Number of intravitreal injection for 1 year†	4.1 ± 0.9	4.7 ± 1.1	0.545
Number of intravitreal injection for 3 years†	6.0 ± 1.1	7.1 ± 1.3	0.081
Number of intravitreal injection for 5 years†	7.2 ± 1.2	9.5 ± 1.3	0.028*
Treatment of PDT‡ (eyes, (%))	15 (50%)	20 (33.3%)	0.114


Mean change (95% CI) from baseline in BCVA, ETDRS letters
14
12
10
8
6
4
2
0
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
Time, mo
Ranibizumab, 0.5 mg, with vPDT
Ranibizumab, 0.5 mg



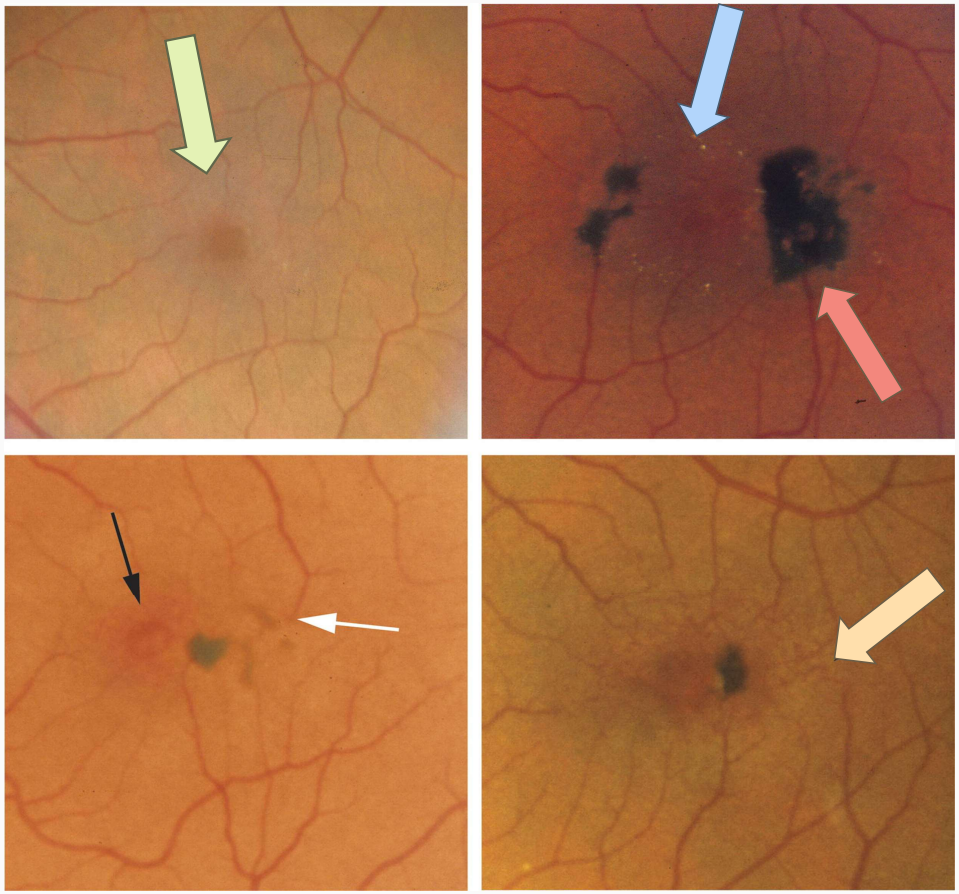
Macular Telangiectasia Type II

What is it?

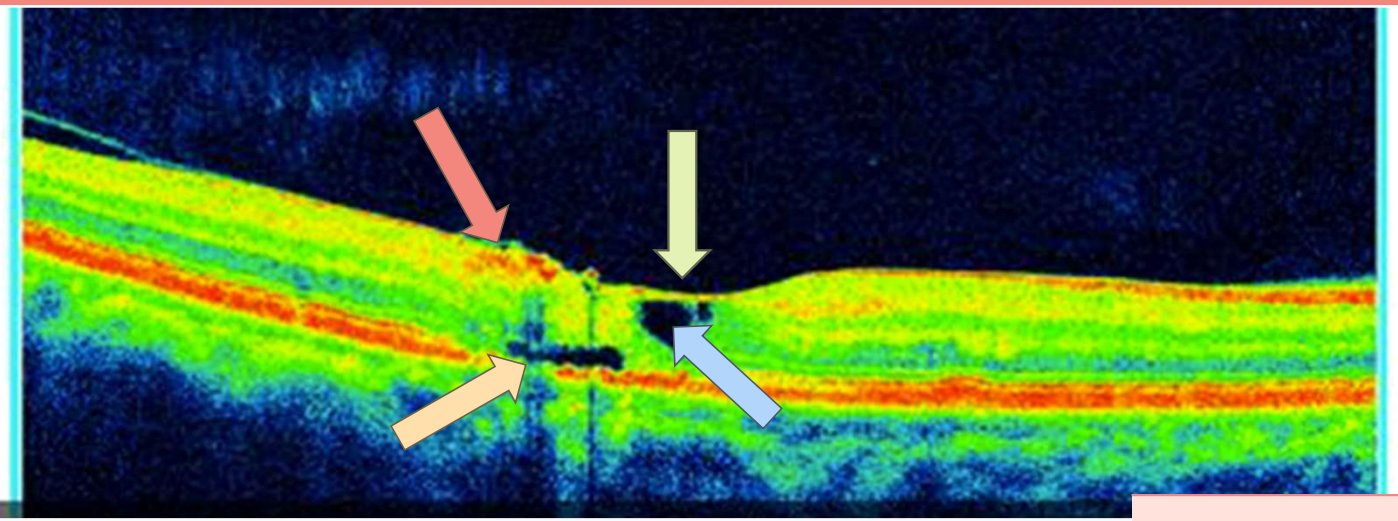
- Macular Telangiectasia Type II (aka MacTel)
  - Pathophysiology:
    - vascular vs neurodegenerative

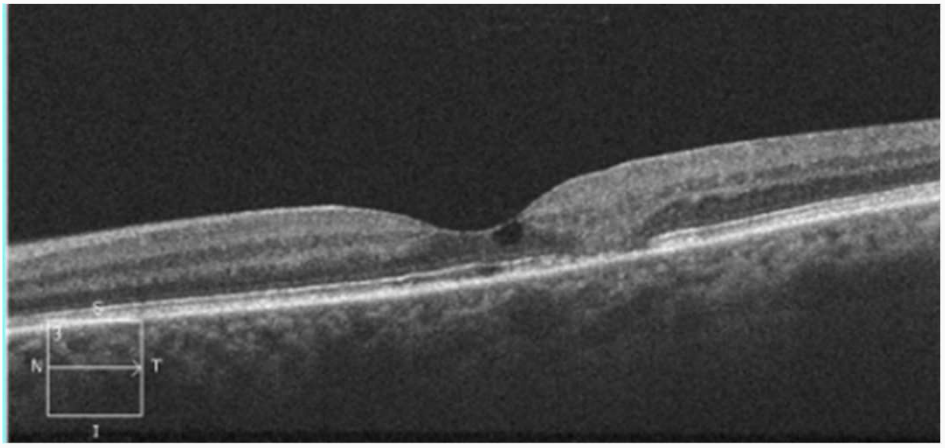
Fundus Findings

- Right-angle venules
- Tiny yellow crystalline deposits
- Loss of retinal transparency
- Telangiectatic vessels
- Hyperpigmentation



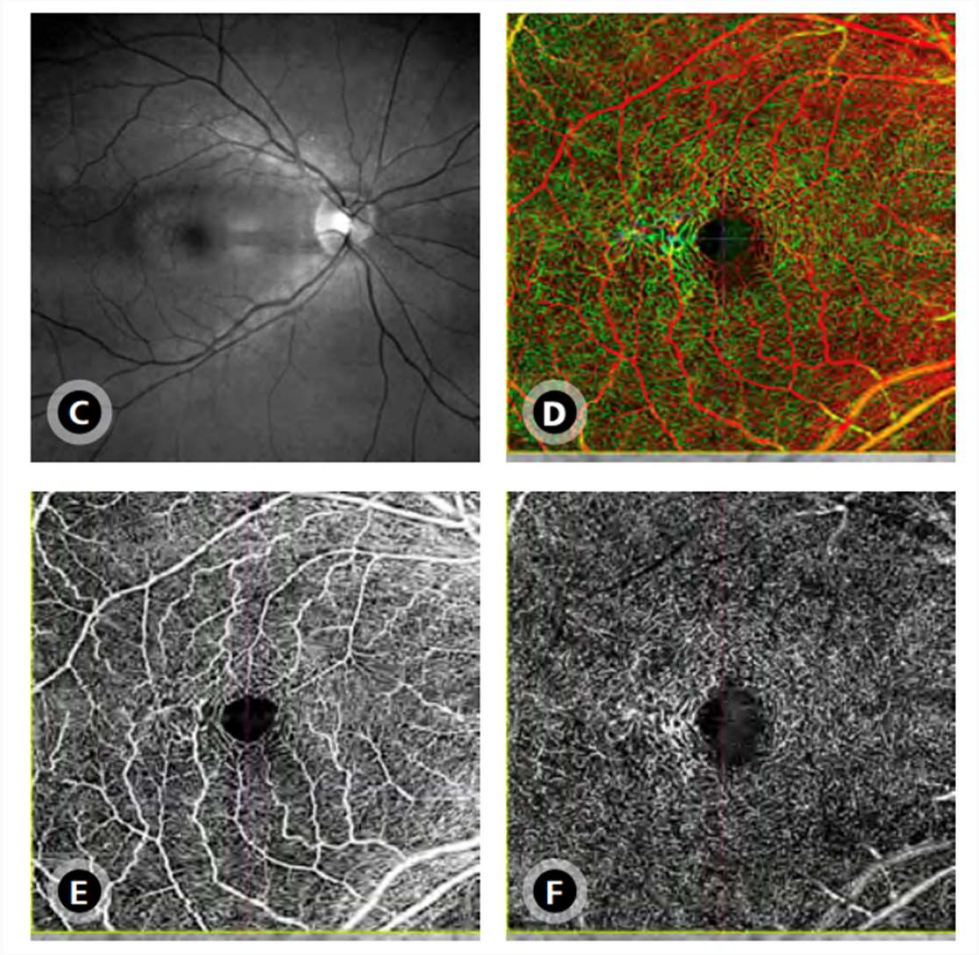
The Prevalence of Macular Telangiectasia Type 2 in the Beaver Dam Eye Study - ScienceDirect

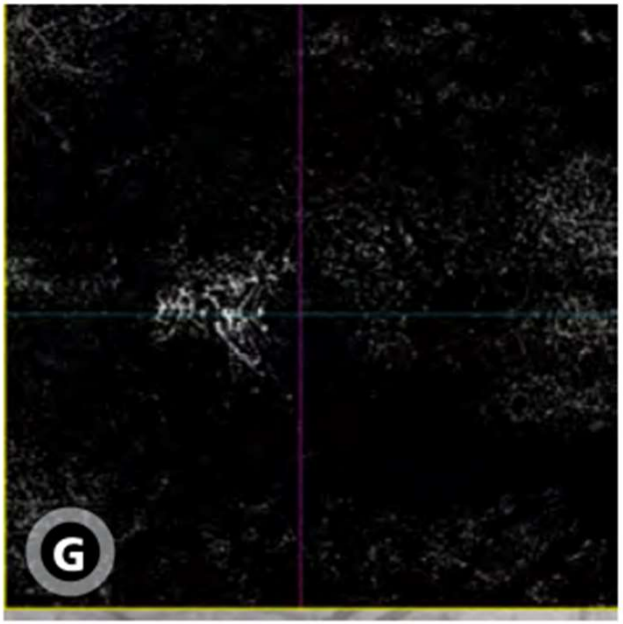




OCT Findings

- Widening of the foveal pit
- ILM drape
- Hyporeflective cavities
- ELM/EZ disruption
- Hyperreflective spots on the ILM
- Subretinal neovascular membrane



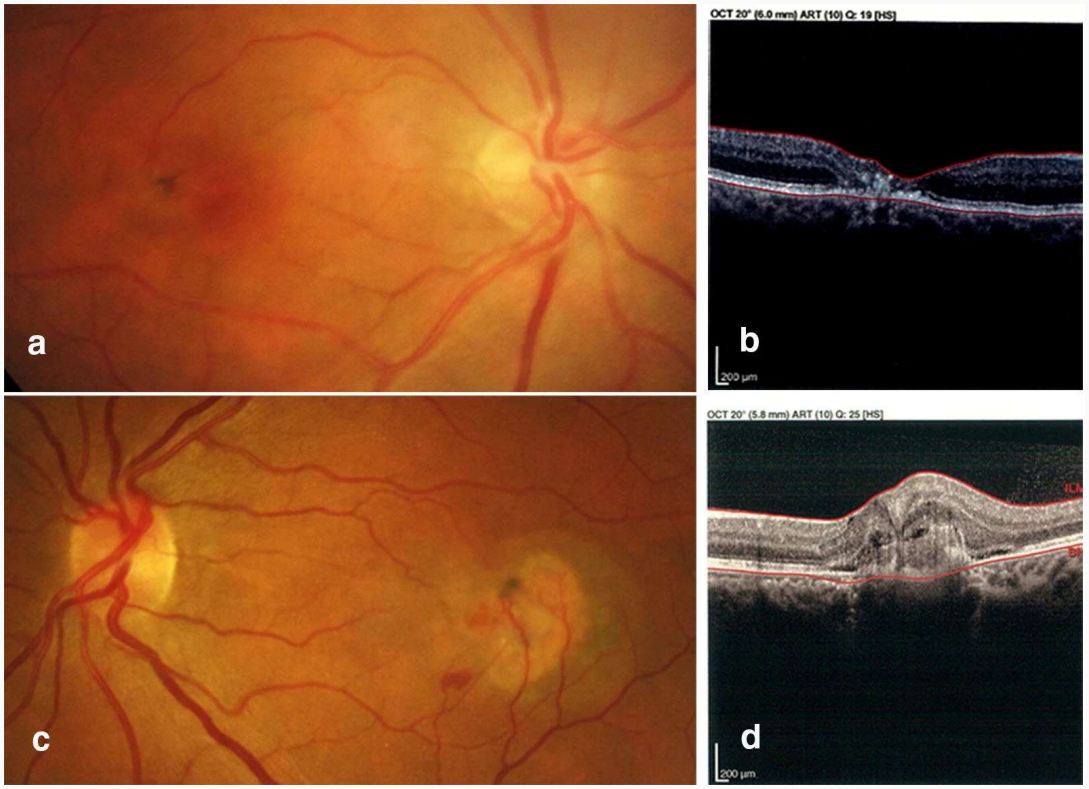


OCT-A

Who gets it?

- Macular telangiectasia type 2
  - Onset **age 40-50**
  - Female predilection

  - Often misdiagnosed



Management of Idiopathic Macular Telangiectasia Type 2 | Ophthalmology and Therapy | Springer Nature Link

Can we treat it?

- Macular telangiectasia type 2
  - Encelto: first treatment for MacTel
    - Approved in March 2025
    - Intravitreal implant
    - Slows ellipsoid zone loss
- Prognosis
  - Approximately 10% of patients develop CNV
  - Rare finding: Full thickness macular holes

Table 4. Best Corrected Visual Acuity of Patients with non-proliferative MacTel.

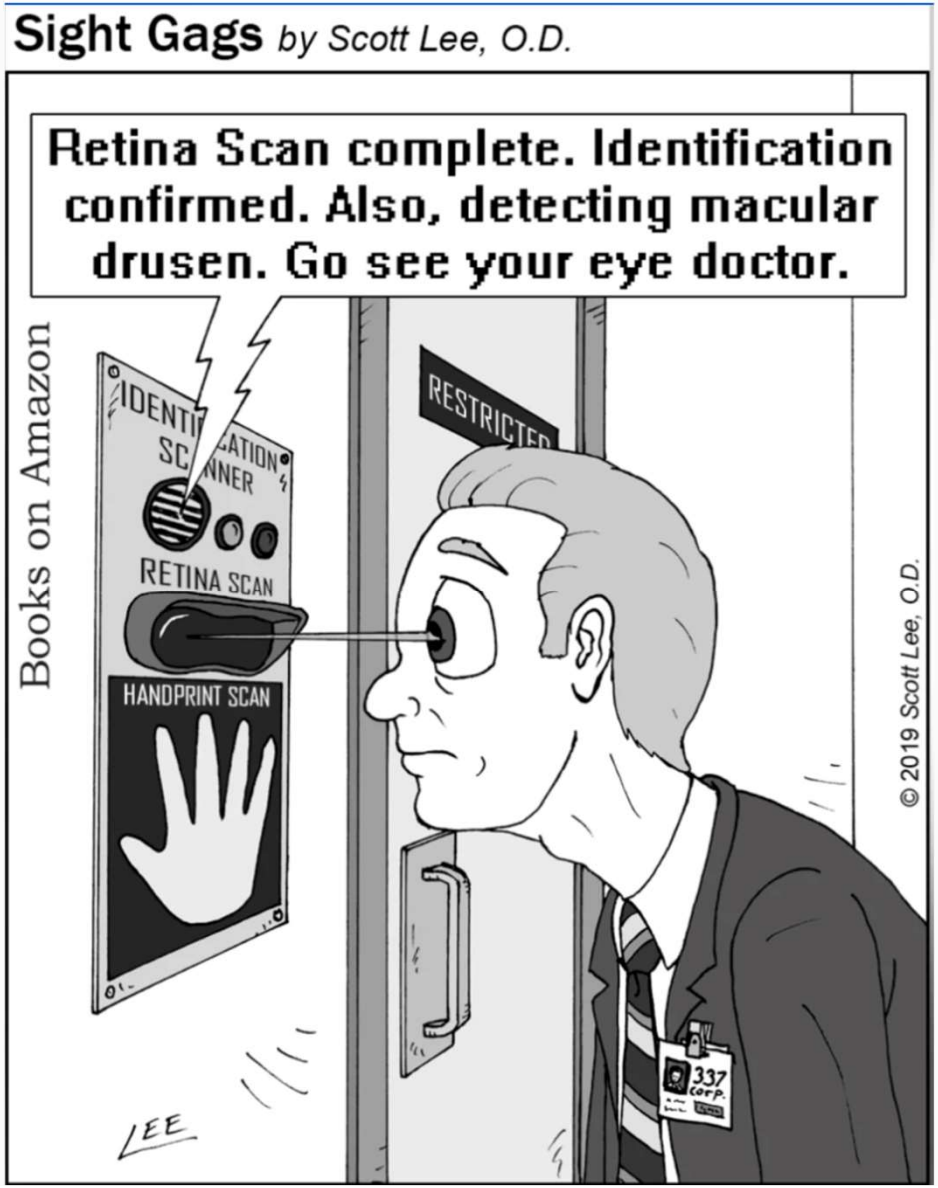
Non proliferative MacTel	BCVA mean log MAR (median,IQR)
BCVA at baseline	0.25 ± 0.25 (median: 0.2, IQR : 0-0.4)
BCVA at 1 year	0.25 ± 0.19 (median: 0.2, IQR : 0.1-0.3)
BCVA at 2 year	0.34 ± 0.37 (median: 0.2, IQR : 0.1-0.4)
BCVA at 3 year	0.41 ± 0.26 (median: 0.3, IQR : 0.2-0.62)
BCVA at 4 year	0.46 ± 0.42 (median: 0.35, IQR : 0.2-0.6)

20/30

20/50

Summary

Recommendations	Goals of Proper Diagnosis
• Consider your patient's demographics • Evaluate ancillary testing thoroughly • Understand the psychological implications of the diagnosis	• Minimize unnecessary treatments (vitamins, injections) • Realign patients' expectations for visual prognosis • Reduce unnecessary patient stress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Retina Scan complete. Identification confirmed. Also, detecting macular drusen. Go see your eye doctor.
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Questions?