REFER THIS, NOT THAT: RETINAL DECISION MAKING

Julie Rodman OD, MSc, FAAo

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RODMAN DISCLOSURES

- iCare
- Visionix
- Astellas
- Apellis
- LKC
- LENZ
- Notal Vision

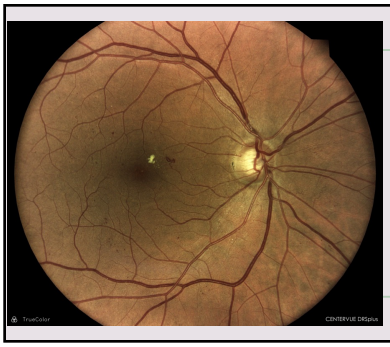
The following financial relationships have been mitigated

2

PATIENT PRESENTATION

- 58-year-old female
- Presents for comprehensive, diabetic eye exam
  - Patient reports **blurry vision OD>OS**, doesn't seem to go *away*
- Type 2 DM x 10 years
  - FBS that morning: 188, HbA1c 8.0
- BCVA measured **20/40 OD** and 20/20 OS

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WHAT IS THE DIAGNOSIS?

DR/DME?

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Non-Proliferative Diabetic Retinopathy (NPDR): AOA

Mild NPDR	MAs only
Moderate NPDR	MAs plus exudates, cotton wool spots, retinal hemorrhages, IRMA, VB
Severe NPDR	Any of the following and NO signs of PDR: (**4-2-1**) • Severe intraretinal hemorrhages and microaneurysms in each of **4** quadrants • Definite venous beading in **2** more quadrants • Prominent IRMA (≥ETDRS standard photograph 8A) in at least one quadrant.
Very-Severe NPDR	2 or more criteria (4-2-1) rule are met

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Proliferative Diabetic Retinopathy (PDR)

PDR	Neovascularization of the disc (NVD) or neovascularization elsewhere (NVE)
High-Risk PDR (DRS)	1. NVD> 1/4-1/3 disc area 2. Any NVD associated with VH or preretinal heme 3. Any NVE associated with VH or preretinal heme

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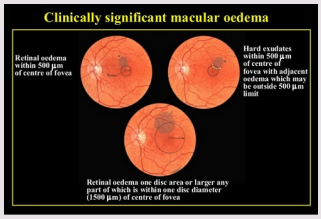


STEP 1:
GRADE THE RETINOPATHY

MODERATE

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DON'T FORGET THE MACULA!



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DIABETIC MACULAR EDEMA

Diabetic macular edema can be further classified as:
- **Non-central-involved** – retinal thickening in the macula that does not involve the center subfield zone that is 1mm in diameter
- **Central-involved** – retinal thickening in the macula that does involve the central subfield zone.

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DME:
CENTER INVOLVING (CI-DME) OR NON-CENTER INVOLVING (NCI-DME)



ETDRS grid map with numerical data for central subfield retinal thickness within innermost circle (1 mm diameter)

CI-DME: retinal thickening in the macula that does involve the central subfield zone.

NCI-DME: retinal thickening in the macula that does not involve the center subfield zone that is 1mm in diameter

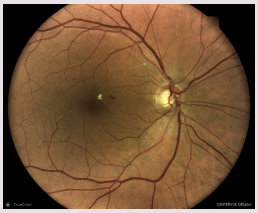
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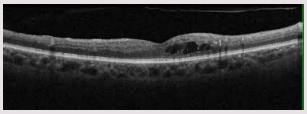
WHY DOES IT MATTER? (ETDRS)

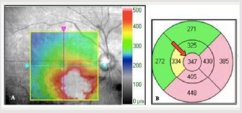
Eyes with central-involved DME had nearly a ten-fold greater risk for developing moderate visual loss compared to eyes without center involvement, stressing the importance of determining central involvement in eyes with macular edema.

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Step 2: Assess the Macula: CI or NCI DME?



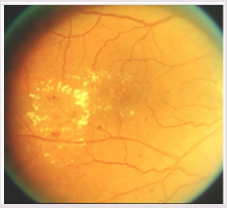


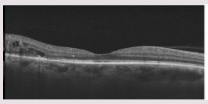


With CI-DME!

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CI or NCI DME?





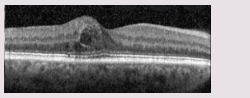


With NCI-DME!

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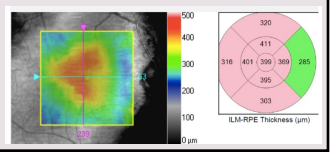


With CI-DME!



Grade the level of retinopathy:

Severe NPDR



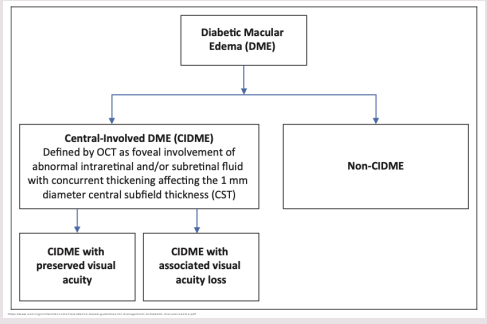
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PEARLS

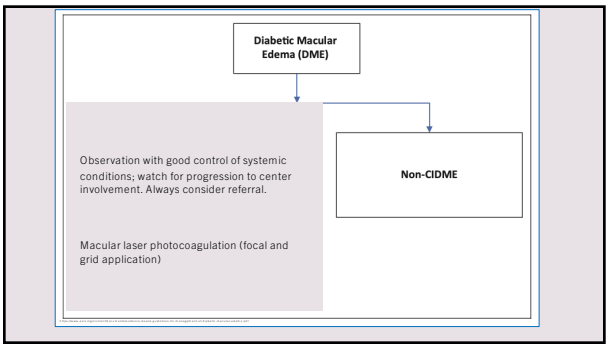
Optometrists should consider referring DME at any stage, especially if it is center involved, causing reduced BCVA or is progressing

AOA Clinical Practice Guidelines recommend consultation within 2-4 weeks when CI-DME is present

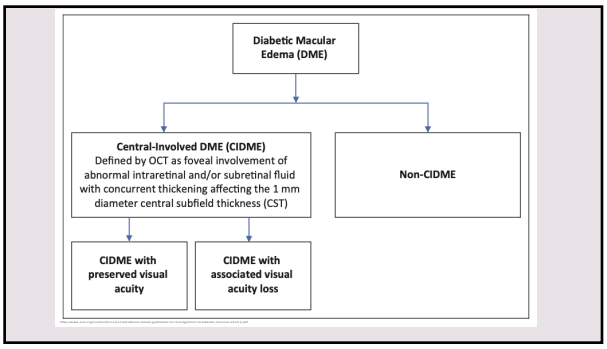
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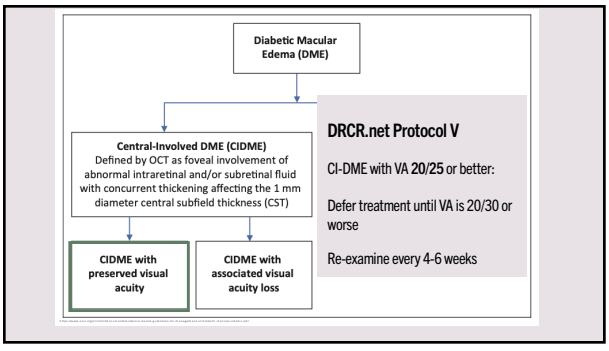
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Diabetic Macular Edema (DME)
Observation with good control of systemic conditions; watch for progression to center involvement. Always consider referral.
Macular laser photocoagulation (focal and grid application)
Non-CIDME

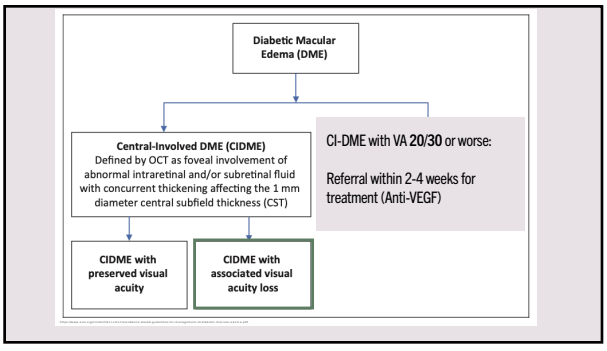
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Diabetic Macular Edema (DME)
Central-Involved DME (CIDME)
Defined by OCT as foveal involvement of abnormal intraretinal and/or subretinal fluid with concurrent thickening affecting the 1 mm diameter central subfield thickness (CST)
Non-CIDME
CIDME with preserved visual acuity
CIDME with associated visual acuity loss

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Diabetic Macular Edema (DME)
Central-Involved DME (CIDME)
Defined by OCT as foveal involvement of abnormal intraretinal and/or subretinal fluid with concurrent thickening affecting the 1 mm diameter central subfield thickness (CST)
CIDME with preserved visual acuity
CIDME with associated visual acuity loss
DRCR.net Protocol V
CI-DME with VA 20/25 or better:
Defer treatment until VA is 20/30 or worse
Re-examine every 4-6 weeks

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Diabetic Macular Edema (DME)
Central-Involved DME (CIDME)
Defined by OCT as foveal involvement of abnormal intraretinal and/or subretinal fluid with concurrent thickening affecting the 1 mm diameter central subfield thickness (CST)
CIDME with preserved visual acuity
CIDME with associated visual acuity loss
CI-DME with VA 20/30 or worse:
Referral within 2-4 weeks for treatment (Anti-VEGF)

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Some anti-VEGF drugs used to treat DME include:

Aflibercept (Eyelea)
Approved by the FDA in 2014, this drug may help control retinal swelling better than other anti-VEGFs

Bevacizumab (Avastin)
Used off-label for ocular conditions since 2005, this drug may lead to greater improvements in macular thickness than laser treatment

Ranibizumab (Lucentis)
Approved by the FDA in 2012, this drug can help prevent visual loss and improve vision

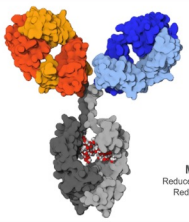
Brolucizumab
Approved by the FDA in 2022, this drug may help control retinal swelling better than other anti-VEGFs

Faricimab
Approved by the FDA in 2022, this drug works similarly to aflibercept and ranibizumab, but also targets the Ang-2 pathway

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Faricimab
Bispecific Monoclonal Antibody
Two Different Antigen-Binding Sites: VEGF-A and Angiopoeitin-2

Anti–Ang-2 Fab
Enhances vascular stability
Reduces inflammation and vascular leakage



Anti–VEGF-A Fab
Inhibits vascular leakage and neovascularization

Modified Fc
Reduces systemic exposure
Reduces inflammatory potential

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PATIENT PRESENTATION

- 60-year-old female
- **Photopsia, mild metamorphopsia OS**
- BCVA measured 20/20 OD and **20/50 OS**

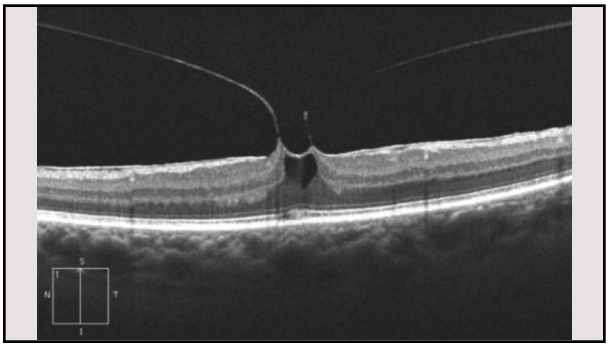
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WHAT IS THE DIAGNOSIS?

ERM/VMT

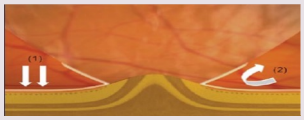
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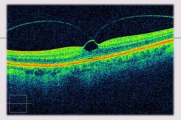
ERM

ERMs are sheet-like structures that develop on the inner surface of the neurosensory retina.
"Proliferation of glial cells"



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VMT



VMT occurs when the posterior cortical vitreous partially separates from the retina, yet some tractional areas remain adherent to the central 3mm of the fovea causing anatomic changes.

Epiretinal membranes and VMT often occur together in the same eye.

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Epiretinal Membrane: Risk Factors

- The risk of developing an ERM increases with age, and persons with predisposing ocular conditions may develop ERM at an earlier age.

>50 years: 2% have ERM	>75 years: 20% have ERM

- *In about 10% to 20% of cases, both eyes have ERMs, but they can be of varying degrees of severity.*

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MANAGEMENT: ERM/VMT

- The current standard of care: (AOA/AAO)
  - Early stage, asymptomatic ERM and/or VMT
    - Monitor every 2-6 months
    - Shorter follow-up intervals (patients with focal VMT or history of MH in the fellow eye)
    - Use OCT biomarkers to help guide decision making

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GOVETTO OCT STAGING SYSTEM: BEYOND VA

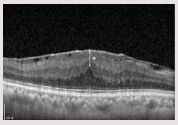
The **Govetto OCT staging system** is a very useful way to describe the **structural severity of an epiretinal membrane (ERM)**. It is based primarily on **three OCT features**:

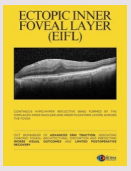
1. **Is the normal foveal pit present?**
2. **Are the individual retinal layers still identifiable?**
3. **Is there an ectopic inner foveal layer (EIFL)?**

The stages progress from **1 → 4**, with increasing distortion of the foveal architecture.

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ECTOPIC INNER FOVEAL LAYER

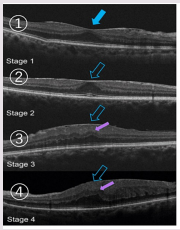




EIFL is a continuous band of inner retinal tissue crossing the entire central foveal area. It corresponds to tissue extending from the **INL/IPL region across the fovea**

https://www.researchgate.net/figure/Ectopic-inner-foveal-layer-The-EIFL-asterisk-on-SD-OCT-was-defined-by-the-presence-of_fig1_339243503

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Stage 1
Presence of the foveal pit.
Well-defined retinal layers.

Stage 2
Absence of the foveal pit.
Well-defined retinal layers.

Stage 3
Absence of the foveal pit.
Well-defined retinal layers.
Presence of ectopic inner foveal layers.

Stage 4
Absence of the foveal pit.
Disrupted retinal layers.
Presence of ectopic inner foveal layers.

When you're reading a scan, ask:

1. Is there an ERM?
↓
2. Is the foveal pit still present?
- Yes → **Stage 1**
- No → keep going

3. Can I clearly identify the retinal layers?
- Yes → could be **Stage 2 or 3**
- No → **Stage 4**

4. Is there a continuous band of inner retinal tissue crossing the foveal center?
- No → **Stage 2**
- Yes → **Stage 3**

https://www.cureus.com/articles/189795-evaluation-of-new-and-preexisting-epiretinal-membranes-following-glaucoma-filtration-surgery

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HOW DO WE USE THIS TO MAKE CLINICAL DECISIONS?

By the time a patient progresses to stage 3, the prognosis after surgery is guarded, relative to operating at earlier stages. Therefore, using visual acuity alone to drive surgical decision making may not optimize patient outcomes. Considering OCT biomarkers and other functional visual symptoms of metamorphopsia may allow for a more sophisticated approach to ERMs.

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TREATMENT CONSIDERATIONS...

When is treatment indicated?

*decrease in visual acuity (<20/40)
*metamorphopsia
*double vision
*difficulty using their eyes together (stereopsis)

Vitrectomy with membrane peel for ERM or VMT usually leads to improvement of the metamorphopsia and visual acuity.

• On average, approximately 80% of patients with ERM or VMT will improve by at least two lines of visual acuity following vitrectomy surgery.

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CLINICAL PEARL

The majority of epiretinal membranes remain relatively stable and do not require treatment. In eyes with vitreomacular traction (VMT) ≤1,500 µm, spontaneous release of traction occurs in approximately 30%–40% of eyes over 1–2 years.

American Academy of Ophthalmology Retina/Vitreous Panel. *Idiopathic Epiretinal Membrane and Vitreomacular Traction Preferred Practice Pattern®*. Ophthalmology. 2025.

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WHAT IS THE DIAGNOSIS?

ERM

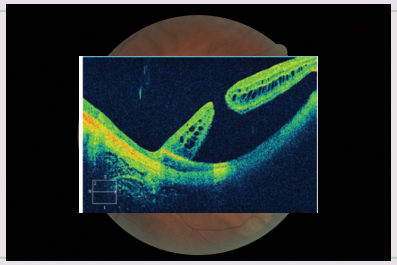
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ASK YOURSELF THESE QUESTIONS:

1. Is the patient symptomatic?
2. What is his visual acuity?

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AND WHAT CAN GO WRONG.....

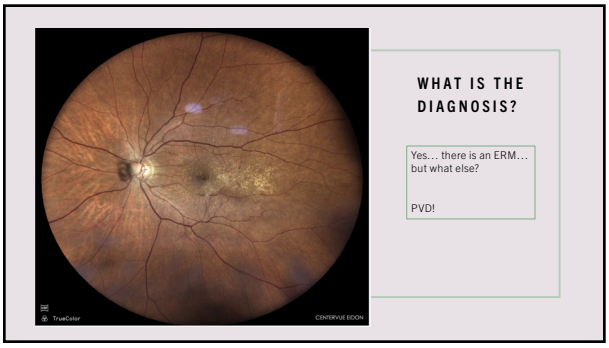


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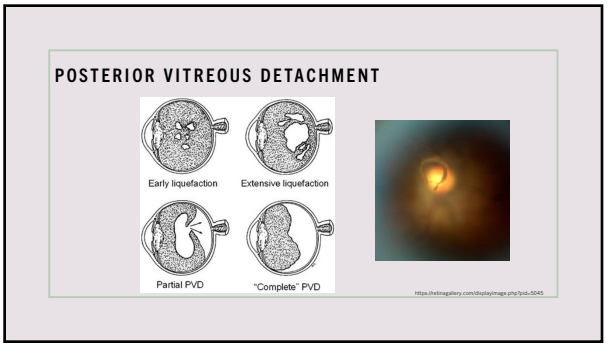
PATIENT PRESENTATION

- 76-year-old male
- Recent onset floaters OS x 2 days; feels like big spider web in vision; no flashes
- BCVA 20/20 OD; 20/40 OS

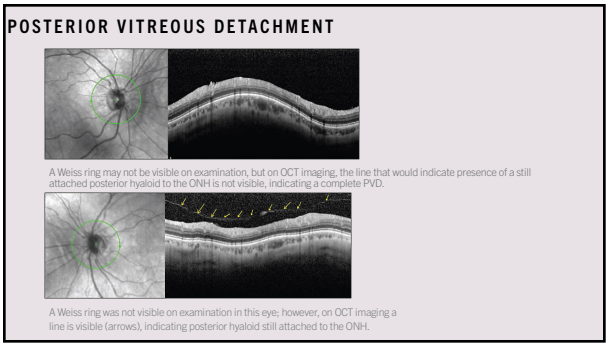
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WHAT IS THE DIAGNOSIS?
Yes… there is an ERM… but what else?
PVD!

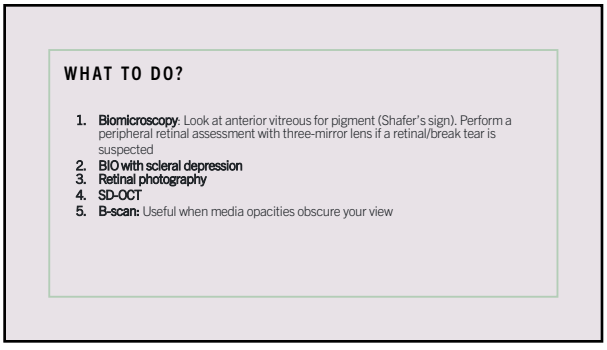
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POSTERIOR VITREOUS DETACHMENT
Early liquefaction
Extensive liquefaction
Partial PVD
"Complete" PVD

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POSTERIOR VITREOUS DETACHMENT
A Weiss ring may not be visible on examination, but on OCT imaging, the line that would indicate presence of a still attached posterior hyaloid to the ONH is not visible, indicating a complete PVD.
A Weiss ring was not visible on examination in this eye; however, on OCT imaging a line is visible (arrows), indicating posterior hyaloid still attached to the ONH.

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WHAT TO DO?
1. Biomicroscopy: Look at anterior vitreous for pigment (Shafer's sign). Perform a peripheral retinal assessment with three-mirror lens if a retinal/break tear is suspected
2. BIO with scleral depression
3. Retinal photography
4. SD-OCT
5. B-scan: Useful when media opacities obscure your view

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WHAT TO DO?

Acute PVD with no complications; (retinal breaks/tears):
-Follow-up with them in **one to two weeks** initially, depending on other risk factors, associated signs or symptoms, or both, **until the condition becomes asymptomatic and no retinal breaks/tears are found.**

Advise these patients to return immediately if they experience an increase in signs or symptoms, such as flashes, floaters or a curtain, in their vision.

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MANAGEMENT

A retinal consult is necessary for **acute symptomatic PVD with retinal breaks/tears** because of the risk of a retinal detachment.

Laser treatment is performed to prevent progression to a retinal detachment. Even when a patient has undergone adequate treatment, **long-term follow-up is important because between 5% and 14% of patients who have an initial retinal break/tear will develop additional breaks/tears during long-term follow-up**.

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PATIENT PRESENTATION

- 73-year-old Caucasian male
- Presented for comprehensive eye examination
  - **CC: Difficulty seeing at night**
- Social history: **(+)Smoker x 40 years**
  - Still smokes 1 pack/day
- PFH: **(+)GF: AMD**
- BCVA **20/40 OD**; 20/20 OS

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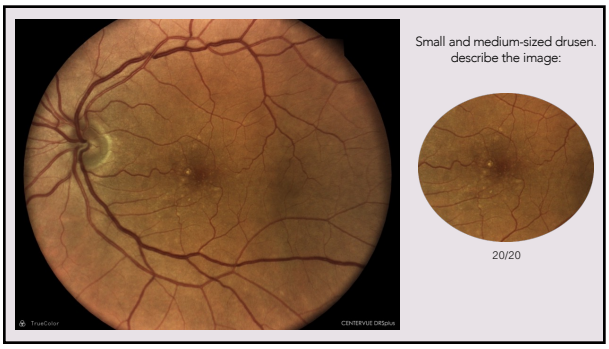
What is the diagnosis?

AMD, What type?



20/30 20/20

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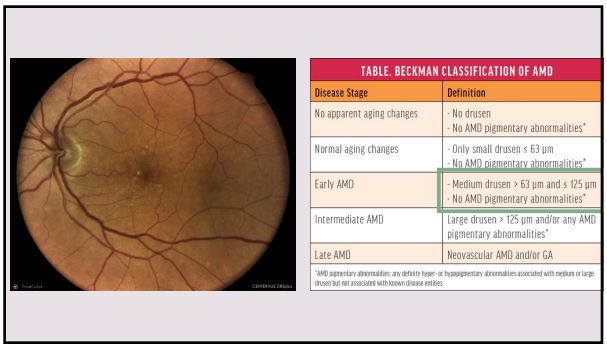
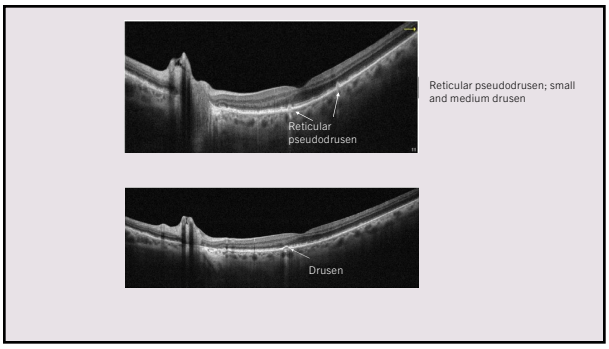


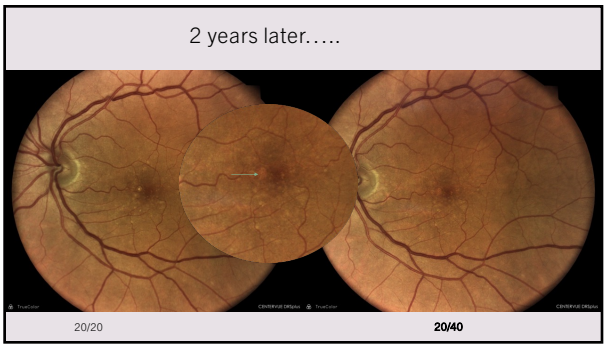
TABLE. BECKMAN CLASSIFICATION OF AMD	
Disease Stage	**Definition**
No apparent aging changes	- No drusen - No AMD pigmentary abnormalities*
Normal aging changes	- Only small drusen ≤ 63 µm - No AMD pigmentary abnormalities*
Early AMD	- Medium drusen > 63 µm and ≤ 125 µm - No AMD pigmentary abnormalities*
Intermediate AMD	Large drusen > 125 µm and/or any AMD pigmentary abnormalities*
Late AMD	Neovascular AMD and/or GA

*AMD pigmentary abnormalities: any definite hyper- or hypopigmentary abnormalities associated with medium or large drusen but not associated with known disease entities.

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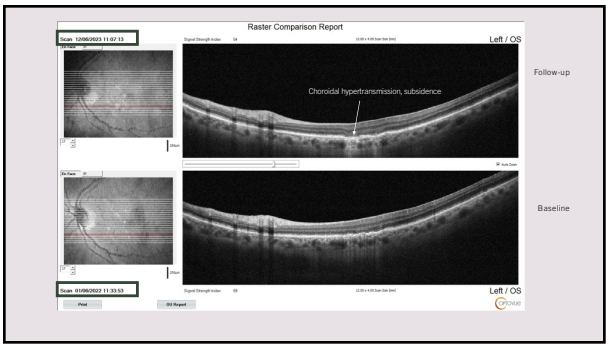
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The patient developed a focal extrafoveal area of GA

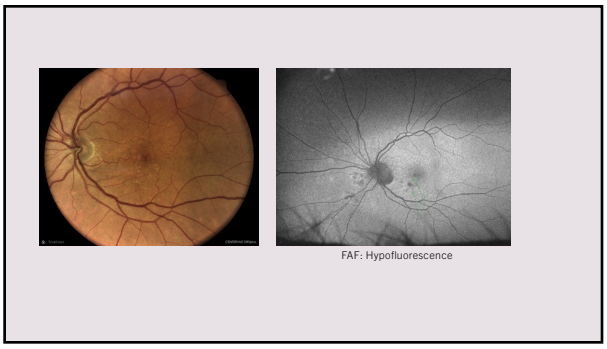
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Drusen collapse
Choroidal hypertransmission, subsidence

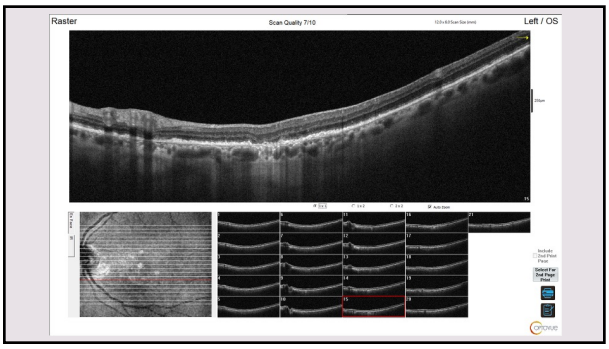
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Raster Comparison Report
Left / OS
Choroidal hypertransmission, subsidence
Follow-up
Baseline
Left / OS

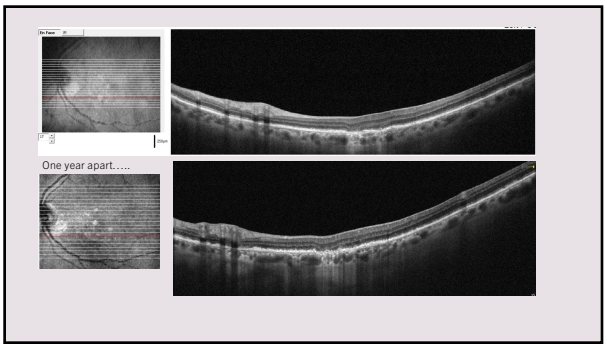
55


FAF: Hypofluorescence

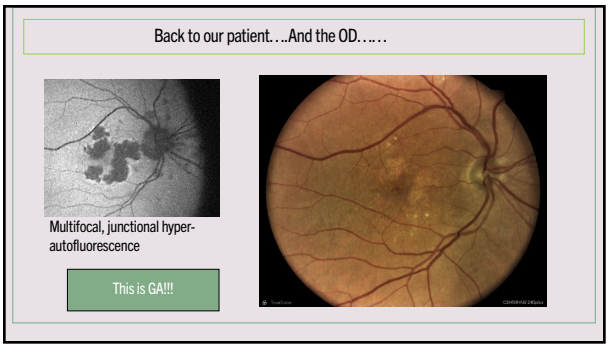
56


Raster
Scan Quality 7/10
Left / OS

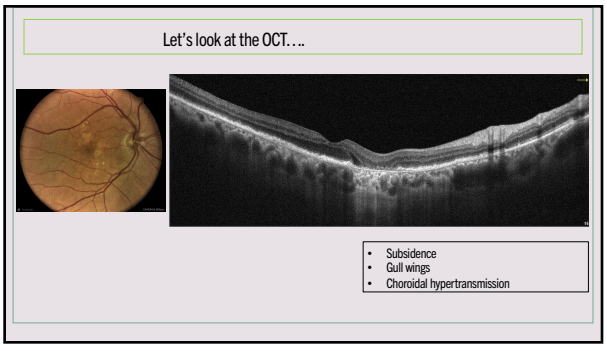
57


One year apart…..

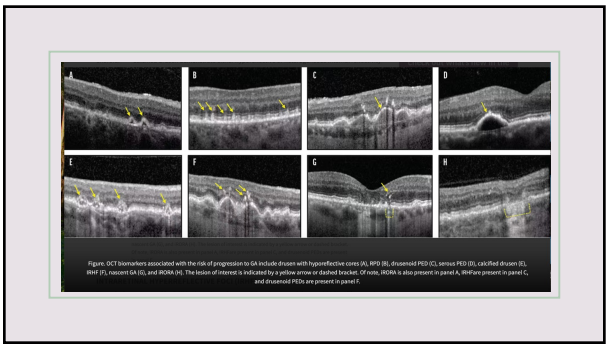
58


Back to our patient….And the OD……
Multifocal, junctional hyper-autofluorescence
This is GA!!!

59

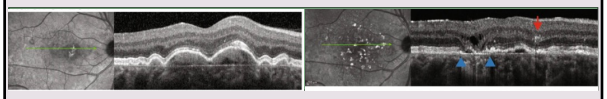

Let's look at the OCT…..
Subsidence
Gull wings
Choroidal hypertransmission

60



61

DOES THIS MAKE SENSE?



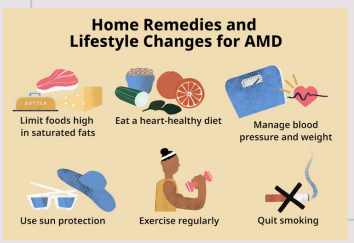
The en-face image looks worse, but the OCT looks better???? Or does it???

62

TREATMENTS?

63

PAST TREATMENT OF GA...





64

Identify early, refer soon, slow course of disease!

AND NOW....



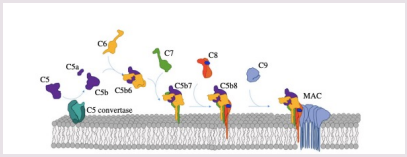


Intravitreal complement inhibitors that SLOW the progression of the disease

65

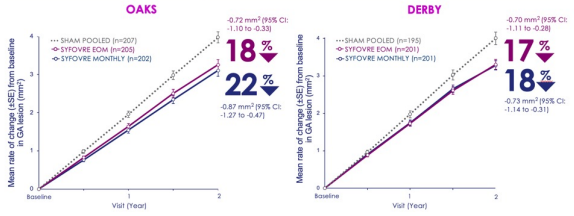
WHAT IS THE COMPLEMENT CASCADE?

- Immune response to recognize and remove pathogens
- The cascade is made up of over 50 proteins
  - These proteins make up the membrane attack complex



66

SYFOVRE Achieved Continuous Reductions in Lesion Growth Rate* vs Sham Pooled† Through Year 2[1,2‡]

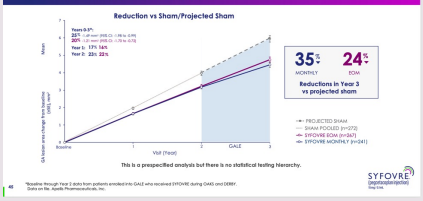


*Based on a mixed effects model for repeated measures assuming a piecewise linear trend in time with knots at Month 6, Month 12, and Month 18.[1]
†SHAM EOM and SHAM Monthly were pooled for analysis.[2]
‡Slope for baseline to Year 2 is an average of slope of baseline to Month 6, Month 6 to Month 12, Month 12 to Month 18, and Month 18 to Month 24.[1]
CI=confidence interval; SE=standard error.
1. SYFOVRE (pegcetacoplan injection) [package insert]. Waltham, MA: Apellis Pharmaceuticals, Inc; 2023. 2. Data on file. Apellis Pharmaceuticals, Inc.

SYFOVRE (pegcetacoplan injection)

67

GALE 1-Year
Up to 35% Reductions in Lesion Growth Rate In Year 3 With Increasing Effect Over Time

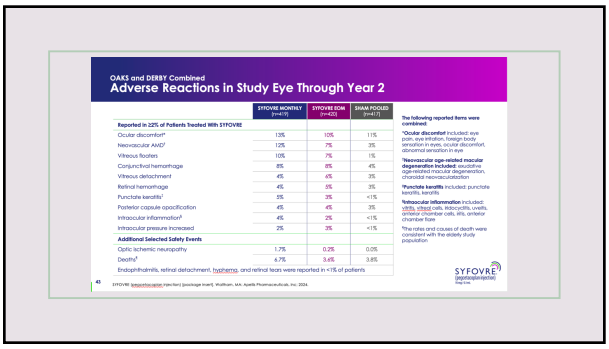


This is a prespecified analysis but there is no statistical testing hierarchy.

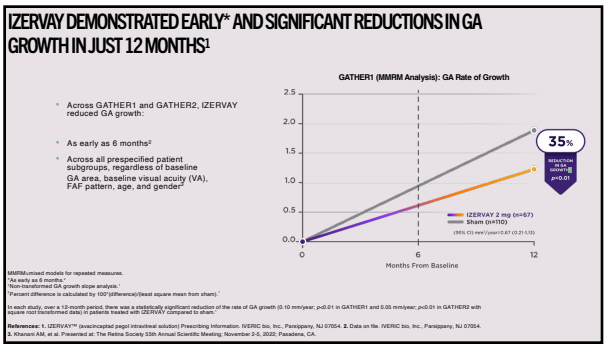
*Baseline through Year 2 data from patients enrolled in to GALE who received SYFOVRE during OAKS and DERBY.
Data on file. Apellis Pharmaceuticals, Inc.

SYFOVRE (pegcetacoplan injection)

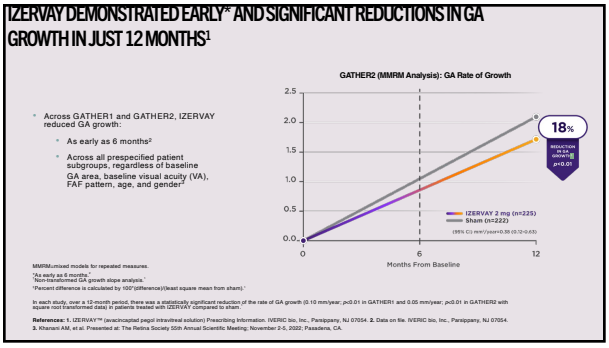
68


OAKS and DERBY Combined
Adverse Reactions in Study Eye Through Year 2
SYFOVRE MONTHLY (n=479)
SYFOVRE EOM (n=432)
SHAM POOLED (n=911)
Reported in ≥2% of Patients Treated With SYFOVRE
Ocular discomfort*
Neovascular AMD†
Vitreous floaters
Conjunctival hemorrhage
Vitreous detachment
Retinal hemorrhage
Punctate keratitis‡
Posterior capsule opacification
Intraocular inflammation§
Intraocular pressure increased
Additional Selected Safety Events
Optic ischemic neuropathy
Deaths¶
Endophthalmitis, retinal detachment, hyphema, and retinal tears were reported in <1% of patients.
The following reported items were combined:
*Ocular discomfort included: eye discomfort, eye pain, foreign body sensation in eyes, ocular discomfort, abnormal sensation in eye
†Neovascular age-related macular degeneration included: exudative age-related macular degeneration, choroidal neovascularization
‡Punctate keratitis included: punctate keratitis, keratitis
§Intraocular inflammation included: vitritis, retinal cells, iridocyclitis, uveitis, anterior chamber cells, iritis, anterior chamber flare
¶The rates and causes of death were consistent with the elderly study population
SYFOVRE

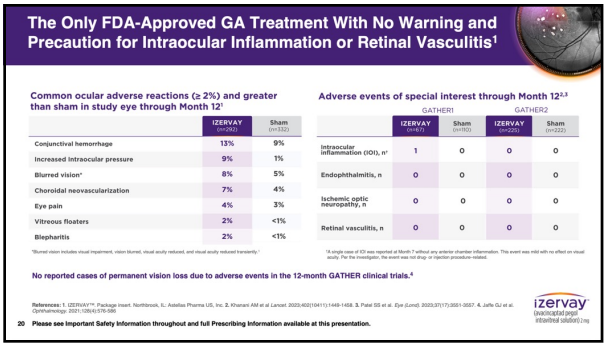
69


IZERVAY DEMONSTRATED EARLY* AND SIGNIFICANT REDUCTIONS IN GA GROWTH IN JUST 12 MONTHS1
GATHER1 (MMRM Analysis): GA Rate of Growth
Across GATHER1 and GATHER2, IZERVAY reduced GA growth:
As early as 6 months2
Across all prespecified patient subgroups, regardless of baseline GA area, baseline visual acuity (VA), FAF pattern, age, and gender2
35%
REDUCTION IN GA GROWTH
p=0.01
IZERVAY 2 mg (n=67)
Sham (n=110)
Months From Baseline
MMRM=mixed models for repeated measures.
*As early as 6 months.†
†Non-transformed GA growth slope analysis.†
‡Percent difference is calculated by 100*(difference)/(least square mean from sham).†
In each study, over a 12-month period, there was a statistically significant reduction of the rate of GA growth (0.10 mm/year; p<0.01 in GATHER1 and 0.05 mm/year; p<0.01 in GATHER2 with square root transformed data) in patients treated with IZERVAY compared to sham.†
References: 1. IZERVAY™ (avacincaptad pegol intravitreal solution) Prescribing Information. IVERIC bio, Inc., Parsippany, NJ 07054. 2. Data on file. IVERIC bio, Inc., Parsippany, NJ 07054. 3. Khanani AM, et al. Presented at: The Retina Society 55th Annual Scientific Meeting; November 2-5, 2022; Pasadena, CA.

70


IZERVAY DEMONSTRATED EARLY* AND SIGNIFICANT REDUCTIONS IN GA GROWTH IN JUST 12 MONTHS1
GATHER2 (MMRM Analysis): GA Rate of Growth
Across GATHER1 and GATHER2, IZERVAY reduced GA growth:
As early as 6 months2
Across all prespecified patient subgroups, regardless of baseline GA area, baseline visual acuity (VA), FAF pattern, age, and gender2
18%
REDUCTION IN GA GROWTH
p=0.01
IZERVAY 2 mg (n=225)
Sham (n=222)
Months From Baseline
MMRM=mixed models for repeated measures.
*As early as 6 months.†
†Non-transformed GA growth slope analysis.†
‡Percent difference is calculated by 100*(difference)/(least square mean from sham).†
In each study, over a 12-month period, there was a statistically significant reduction of the rate of GA growth (0.10 mm/year; p<0.01 in GATHER1 and 0.05 mm/year; p<0.01 in GATHER2 with square root transformed data) in patients treated with IZERVAY compared to sham.†
References: 1. IZERVAY™ (avacincaptad pegol intravitreal solution) Prescribing Information. IVERIC bio, Inc., Parsippany, NJ 07054. 2. Data on file. IVERIC bio, Inc., Parsippany, NJ 07054. 3. Khanani AM, et al. Presented at: The Retina Society 55th Annual Scientific Meeting; November 2-5, 2022; Pasadena, CA.

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The Only FDA-Approved GA Treatment With No Warning and Precaution for Intraocular Inflammation or Retinal Vasculitis1
Common ocular adverse reactions (≥ 2%) and greater than sham in study eye through Month 12†
IZERVAY (n=392)
Sham (n=332)
Conjunctival hemorrhage 13% 9%
Increased intraocular pressure 9% 1%
Blurred vision* 8% 5%
Choroidal neovascularization 7% 4%
Eye pain 4% 3%
Vitreous floaters 2% <1%
Blepharitis 2% <1%
Adverse events of special interest through Month 12‡,3
GATHER1
GATHER2
IZERVAY (n=67)
Sham (n=110)
IZERVAY (n=225)
Sham (n=222)
Intraocular inflammation (IOI), n^ 1 0 0 0
Endophthalmitis, n 0 0 0 0
Ischemic optic neuropathy, n 0 0 0 0
Retinal vasculitis, n 0 0 0 0
No reported cases of permanent vision loss due to adverse events in the 12-month GATHER clinical trials.4
izervay

72

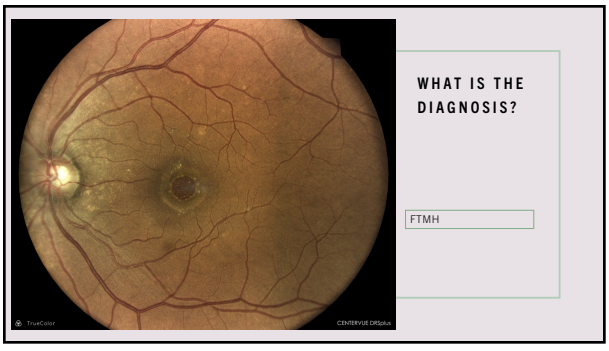


73

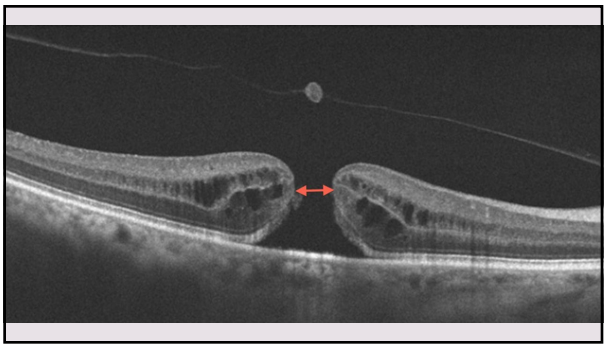
PATIENT PRESENTATION

- 75-year-old female
- Slowly progressive decrease in vision OS; spot in central vision
- Pseudophakic OU
- BCVA measured 20/25 OD and **20/400 OS**

74



75



76

SIZE OF THE DEFECT

What is the size of the aperture?
(Size critical for surgical outcome)

Small: Full thickness defect; < 250 um
Medium: Full thickness defect; >250 um and <400 um
Large: Full thickness defect; >400 um

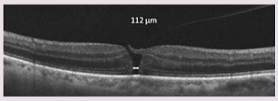
Measure at narrowest point on OCT
Parallel to RPE

77

SIZE OF DEFECT

Small: Full thickness defect; < 250 um

- *Visual acuity may be relatively good*
- *Very high probability of success with vitrectomy*

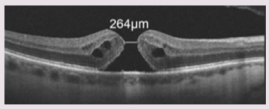


78

SIZE OF DEFECT

Medium: Full thickness defect; >250 um and <400 um

- *High probability of success with vitrectomy surgery*

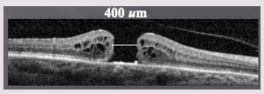


79

SIZE OF DEFECT

Large: Full thickness defect; >400 um

- *Less probability of successful closure with vitrectomy surgery*



80

MANAGEMENT

Full-thickness macular hole (FTMH)	- An interruption in all retinal layers from the internal limiting membrane to the photoreceptors. - FTMHs are further classified as either with or without VMT; by size (small = < 250 µm, medium = 250 to 400 µm, large = > 400 µm); and primary versus secondary.

Surgery involves a pars plana vitrectomy procedure with tamponade. This can be done with or without peeling of the internal limiting membrane.

81

Modified inverted ILM flap technique



82

PATIENT PRESENTATION

- 35-year-old male
- **Can't see OD x 1 month; thought it would go away on its own**
- **Had flashes and floaters which subsided OD**
- BCVA measured **20/400 OD** and 20/20 OS

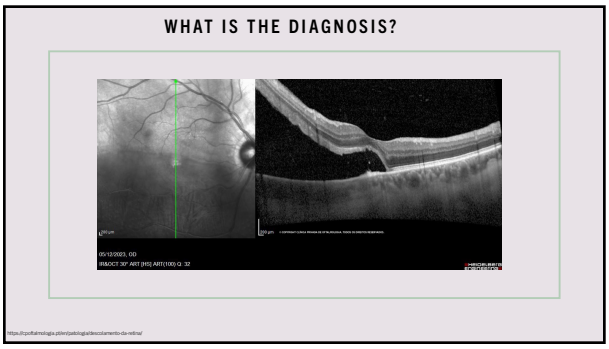
83

WHAT IS THE DIAGNOSIS?

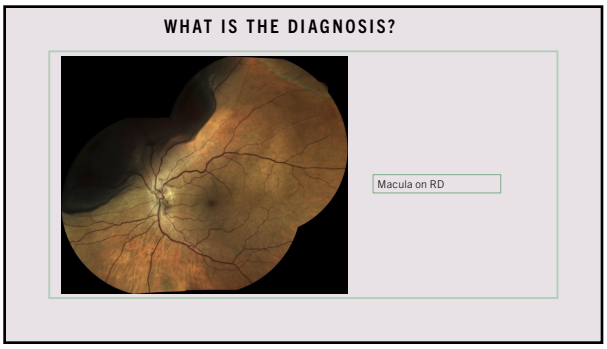


Macula off RD

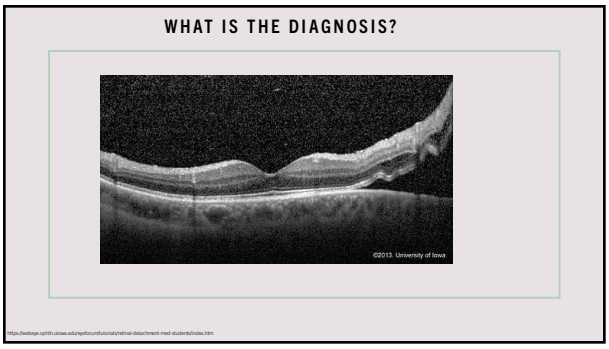
84


WHAT IS THE DIAGNOSIS?

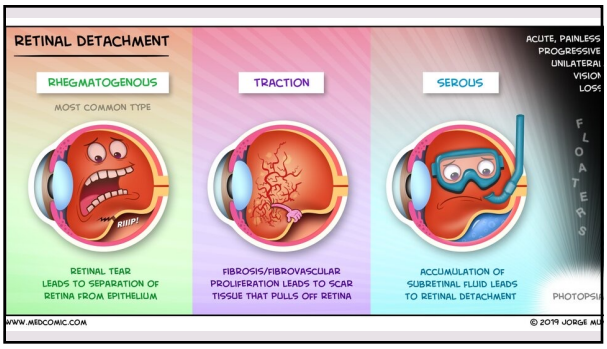
85


WHAT IS THE DIAGNOSIS?
Macula on RD

86


WHAT IS THE DIAGNOSIS?
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RETINAL DETACHMENT
RHEGMATOGENOUS
MOST COMMON TYPE
TRACTION
SEROUS
ACUTE, PAINLESS
PROGRESSIVE
UNILATERAL
VISION
LOSS
FLOATERS
RETINAL TEAR
LEADS TO SEPARATION OF
RETINA FROM EPITHELIUM
FIBROSIS/FIBROVASCULAR
PROLIFERATION LEADS TO SCAR
TISSUE THAT PULLS OFF RETINA
ACCUMULATION OF
SUBRETINAL FLUID LEADS
TO RETINAL DETACHMENT
WWW.MEDCOMIC.COM

88

MANAGEMENT

- **Macula off**
  - Some recommend referring patients with recent macula-off retinal detachments (RDs) **as soon as possible**, ideally within **48 to 72 hours of central vision loss.** This is because studies suggest that earlier surgery may lead to similar outcomes to macula-on RDs, which are historically considered to require surgery on the same day. However, others say that surgery doesn't need to be an emergency within the first 24 hours.
- **Macula on**
  - IMMEDIATE REFERRAL

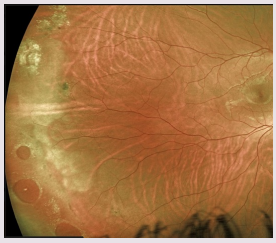
89

PATIENT PRESENTATION

- 29-year-old male
- -4.50 DS OU
- Longstanding floaters OU
- No flashes
- 20/20 OD and OS

90

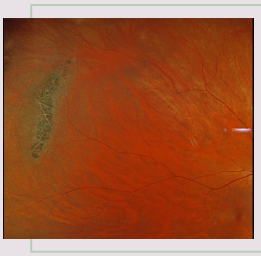
WHAT IS THE DIAGNOSIS?



Atrophic holes in lattice

91

FIRST ISSUE...LATTICE



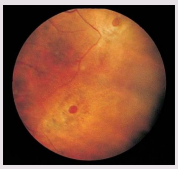
- **Lattice degeneration** is a common peripheral retinal thinning condition.
- Often found **incidentally** during DFE.
- It occurs in roughly 6–10% of people and is more common in myopes.

Monitor!

92

SECOND ISSUE… ATROPHIC HOLES

- Atrophic retinal holes are **full thickness retina breaks** often existing in the peripheral retina.
- Result of atrophic changes/**thinning** within the sensory retina that is **not induced by vitreous adhesions**.
- Often, these lesions are found in association with lattice degeneration.



https://doctorlib.org/ophthalmology/principles/59.html

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MANAGEMENT

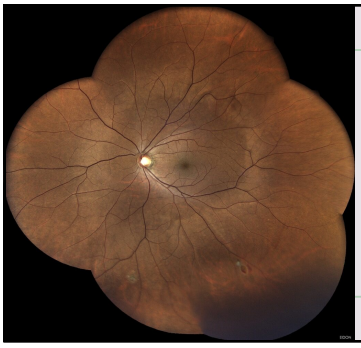
• **Asymptomatic** atrophic retinal hole:	**Observe in most cases**
• **Asymptomatic** atrophic retinal hole within lattice (no PVD, no subretinal fluid)	**Observe in most cases**
• **Symptomatic** atrophic retinal hole within lattice	**Refer for possible barricade laser**

94

How soon?

- **New flashes/floaters + atrophic hole in lattice:**
  - Arrange **retina/ophthalmology evaluation within a few days**, particularly if you cannot confidently exclude an associated horseshoe tear or acute PVD.
- **Same day / next day** if there is:
  - vitreous or retinal hemorrhage
  - pigment in the vitreous
  - significant/new PVD with suspicious vitreoretinal traction
  - increasing SRF around the hole
  - subclinical or clinical retinal detachment
  - visual field defect/"curtain"
  - significant sudden increase in floaters or flashes

95



What is the diagnosis?

Operculated retinal hole

96

OPERCULATED HOLES

- Small full-thickness breaks in the retina where a tiny piece of retinal tissue ("operculum") has been pulled free, usually by vitreous traction as the eye's gel changes with age.
- They're often found **incidentally** and may be low-risk once the pulling force has released, but they can sometimes allow fluid under the retina and lead to a retinal detachment.



- **Risk is higher if there are symptoms, surrounding fluid, multiple breaks, high myopia, prior detachment in the other eye, or recent eye trauma.**

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MANAGEMENT

- **Asymptomatic** operculated hole (no fluid): **Observe in most cases**
- **Symptomatic** operculated hole **Varies: If evidence of vitreoretinal traction or SRF, refer**

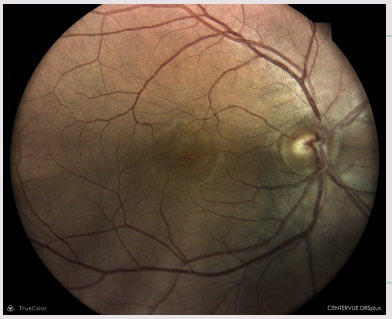
Operculated hole = traction has usually been released → observe if no SRF or residual traction.

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PATIENT PRESENTATION

- 30-year-old male
- Weight-lifter: **Type A personality; under a lot of stress at work**
- **Recent decrease in vision OD (getting worse over past month)**
- BCVA **OD 20/40**, OS 20/20

99

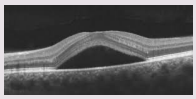


WHAT IS THE DIAGNOSIS?

CSR

100

Central serous chorioretinopathy is a retinal disorder characterized by localized serous detachment of the macula. It is secondary to retinal pigment epithelial decompensation and choroidal vascular changes.



101

MANAGEMENT

The majority of cases of CSCR resolve spontaneously:
*Observation is done for 3 months
*Reduction of stress
*Stoppage of steroids or other predisposing factors

80% cases resolve spontaneously.

- Laser
- PDT
- Anti-VEGF
- Anti-corticosteroids

Patients with CSCR commonly have endogenous hypercortisolism, thus drugs targeting cortisol pathways might be effective.

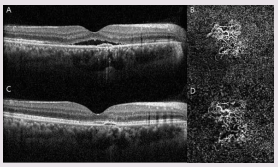
102

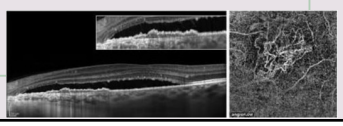
INDICATIONS FOR EARLY INTERVENTION

*Visual demand for profession (pilot) or the patient for early recovery
*Recurrent disease
*Severe decline of vision in the other eye due to chronic CSCR

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WHAT WE DON'T WANT TO HAPPEN!





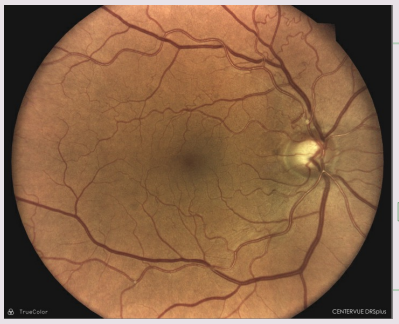
https://www.researchgate.net/figure/Chronic-central-serous-chorioretinopathy-and-irregular-pigment-epithelium-detachment_fig2_270402303

104

PATIENT PRESENTATION

- 72-year-old female
- **H/O stroke one year prior**
- Longstanding HTN, arteriosclerosis
- No visual complaints
- OD 20/20; OS 20/20

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WHAT IS THE DIAGNOSIS?

Retinal Plaques

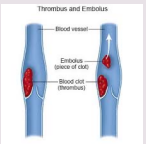
106

WHAT IS A RETINAL PLAQUE?

Plaques are created if plaque from in your body breaks free and blocks a blood vessel in your retina

When the clot travels from the site where it formed to another location in the body, it is called an embolism.

Retinal ischemia can occur if the embolus does not dislodge and blocks perfusion or retinal blood in the area



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DIFFERENTIAL DIAGNOSIS

- ***Cholesterol emboli/Hollenhorst plaque.***
  - Highly refractile, crystal-like emboli that typically are seen at arteriole bifurcations.
  - The emboli can be visually asymptomatic, because they often do not cause a significant obstruction of the retinal arteriole.
  - Also known as Hollenhorst plaques, cholesterol emboli originate from atheromatous lesions in the ipsilateral carotid artery or aorta.



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DIFFERENTIAL DIAGNOSIS

- ***Fibrin platelet emboli***.
  - Dull white in appearance and often present as long, smooth emboli simulating a plug in the retinal arteriole.
  - They are most commonly associated with carotid thrombosis.



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DIFFERENTIAL DIAGNOSIS

- ***Calcific emboli***.
- Non-scintillating, white in appearance, and typically present in the central retinal artery due to their large size.
- Such entities may remain in the retinal vasculature permanently, because they do not dissolve.
- They are associated with heart valve or aorta calcification. Transesophageal echocardiogram is needed to confirm the diagnosis.



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Retinal emboli are found within retinal arterioles

- Most commonly stem from vascular diseases
- Often incidental finding on fundus examination

Arise from carotid arteries, coronary arteries, or cardiac valves

- 80% cholesterol: Hollenhorst

Composition of emboli will dictate migration and resolution

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WHAT YOU NEED TO KNOW!

❖Risk factors for retinal emboli:

- ❖ Older age
- ❖ Male sex
- ❖ Hypertension
- ❖ Cholesterol levels
- ❖ Coronary artery disease
  - ❖ h/o bypass graft
- ❖ Smoking

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MANAGEMENT

- Urgent workup is indicated for **SYMPTOMATIC** plaques
  - *Unilateral vision loss
  - *Amaurosis Fugax
  - *Total loss of vision
  - *Visual Scotoma

OR: *headaches, numbness in face, arm, legs, or one side of body
- *dizziness
- *difficulty with balance or speaking
- *fainting

Send to stroke center STAT!

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MANAGEMENT

Urgent embolic evaluation including carotid ultrasound analysis is indicated, as approximately 25% may have substantial carotid artery stenosis. In patients with moderate to severe carotid artery stenosis, carotid endarterectomy can substantially reduce the risk of subsequent hemispheric cerebral vascular accident

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MANAGEMENT

- Approximately 75% of plaques seen in ophthalmic practice are asymptomatic

As high as 12% of patients with asymptomatic plaques are at risk of cerebrovascular events and 22% have significant carotid artery stenosis (>50%)

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MANAGEMENT

AAO management of an asymptomatic retinal embolus:

Asymptomatic Hollenhorst plaques should prompt a nonurgent workup for systemic atheromatous disease.

The patient should therefore be referred to their **primary care physician/internist** for evaluation and management of systemic vascular risk factors. The AAO specifically recommends evaluation for relevant systemic conditions rather than ocular treatment.

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CLINICAL PEARL

For an **asymptomatic Hollenhorst plaque**:
Fundus photo → document → check BP/risk factors → communicate with PCP → recommend systemic vascular evaluation ± carotid duplex → manage cardiovascular risk factors → monitor ocularly.

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DIAGNOSTIC TESTING

- Carotid doppler ultrasound
- Transesophogeal echocardiography (calcific plaque)
- Computed tomographic angiography and or MRA (degree of carotid stenosis)

Regardless of disease severity, sending for carotid workup is safe and is a justified approach to managing these patients. Always communicate with the patient's primary care provider.

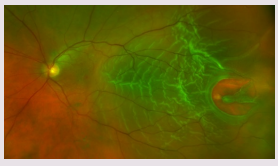
118

PATIENT PRESENTATION

- 30-year-old female
- **High myope**
- **New onset flashes, floaters, hazy vision OS**
- BCVA measured 20/20D and 20/20 OS

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WHAT IS THE DIAGNOSIS?



Horseshoe Retinal Tear

https://www.eyetec.net/index.php?option=com_content&view=article&id=625:tech-tips-august-2012&catid=53:tech-tips&Itemid=86

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• Horseshoe tears, also referred as flap or U-shaped tears, are **full thickness** breaks in the neurosensory retina that occur secondary to **vitreo-retinal traction**.



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MANAGEMENT

A **symptomatic retinal horseshoe tear (horseshoe retinal break)** should be referred to a retina specialist **the same day**, as it is considered an ophthalmic urgency.

If same-day evaluation is unavailable:
- The patient should generally be seen **within 24 hours**.
- The referring clinician should communicate directly with the retina specialist's office rather than simply providing a routine referral.

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MANAGEMENT

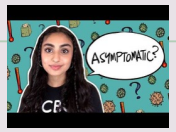
Symptomatic retinal tears:

- Symptomatic retinal horseshoe tears and persistent vitreoretinal traction can progress to a retinal detachment in **33 to 55 % of cases.**

Prompt retinopexy reduces that risk to **well under 10%**, often around **2–5%**, although exact rates vary among studies.

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MANAGEMENT



Asymptomatic tears:

Treating asymptomatic retinal horseshoe tears is less straight forward. Approximately 5% of these patients will progress to a retinal detachment.

An **asymptomatic horseshoe tear** is still usually referred promptly (often within a few days), but a **symptomatic horseshoe tear** warrants **same-day evaluation** because of the substantially higher risk of retinal detachment.

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PATIENT PRESENTATION

- 17-year-old female
- History of **blunt trauma to her left eye** one day prior
- She went to the ER the day of the injury and got imaging, but no fractures were found
- She reported photophobia, eye pain, eyelid tenderness and blurry vision in the left eye since the incident occurred
- She denied flashes, floaters, diplopia, or headaches.
- BCVA measured 20/20D and 20/20 OS

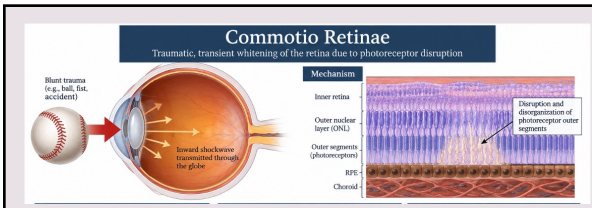
127



WHAT IS THE DIAGNOSIS?

Commotio Retinae

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Commotio retinae refers to traumatic retinopathy secondary to direct or indirect trauma to the globe. Retinopathy may be present at areas of scleral impact (coup) and or distant sites (contrecoup) including the macula. Commotio retinae in the posterior pole is also referred to as Berlin's edema.



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MANAGEMENT

Patients suffering substantial ocular trauma with commotio retinae should be followed with serial eye exams to evaluate for any additional treatable conditions. As commotio of macula resolves, partial or full thickness macular holes can develop.

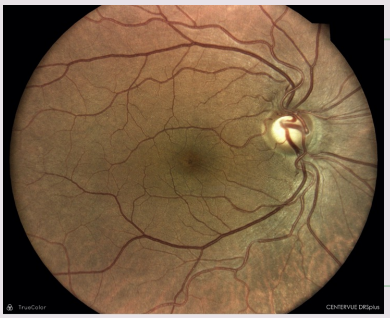
There is no **approved or commonly used medical treatment for commotio retinae.** However, in cases that do not resolve spontaneously, high dose IV steroids have been anecdotally shown to reduce retinal swelling and improve visual acuity.

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PATIENT PRESENTATION

- 45-year-old male
- Asymptomatic
- BCVA 20/20 OD; 20/20 OS

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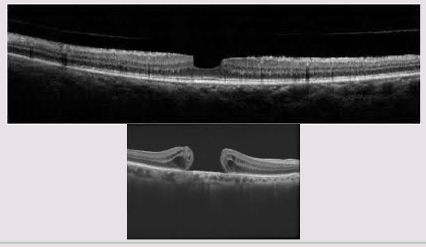
WHAT IS THE DIAGNOSIS?

Pseudohole/ERM

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Macular pseudoholes are partial thickness holes that resemble a "U" shape and can be related to ERM. Partial thickness macular hole have an **incomplete loss** of tissue from ILM to RPE. During clinical exam, it will appear as a circular or oval reddish lesion similar to a FTMH.

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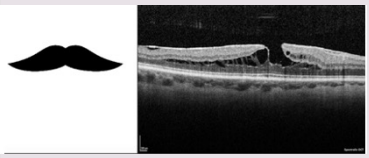
PARTIAL THICKNESS MACULAR HOLE

Lamellar Hole: Tractional and Degenerative

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LAMELLAR HOLE: 2 TYPES

Tractional: "Moustache" appearance

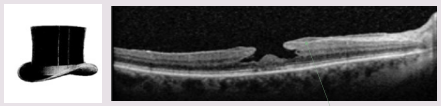


- Must Have:
  ERM and foveoschisis

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LAMELLAR HOLE: 2 TYPES

Degenerative: "Top Hat" appearance



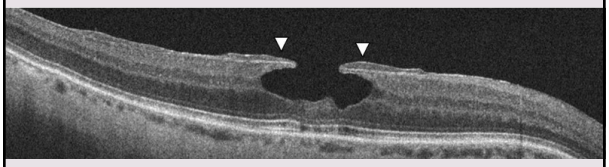
MUST HAVE:

- Irregular foveal contour
- Foveal cavitation with round edges
- Loss of foveal tissue (thinning)

??Epiretinal proliferation

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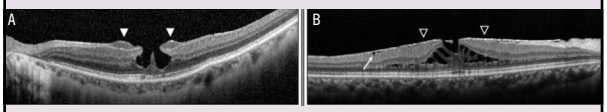
What is Epiretinal Proliferation?



A thick, homogeneous, isoreflective layer covered by a thin hyper-reflective line at the edges of the hole contiguous with the inner retina

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Quiz: Degenerative or tractional?



A: Degenerative

- Irregular foveal contour
- Foveal cavitation with round edges
- Loss of foveal tissue (thinning)

B: Tractional

- ERM and foveoschisis

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MANAGEMENT

Management of Lamellar Macular Hole
- Most LMHs can be observed if stable and minimally symptomatic
- Surgical intervention remains controversial because outcomes are variable
- Consider vitrectomy when there is **progressive anatomical deterioration or functionally significant symptoms**, including:

Metamorphopsia
Central scotoma
Visual acuity loss interfering with activities of daily living

Other guidelines recommend surgical intervention whenever VA <20/40 and there is evidence of ERM. Surgical options include phacoemulsification with vitrectomy and ERM and ILM peeling. ILM inverted flap.
*Preoperative defects in the PIL are associated with worse visual prognosis

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THANK YOU!!

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