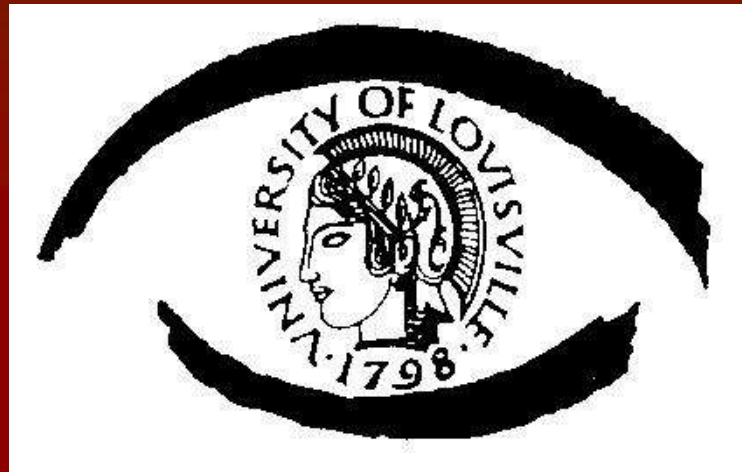


# Grand Rounds



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# Subjective

CC: “ decreased vision in R eye”

HPI: 78 year-old AAF presented to clinic c/o decreased vision OD. Pt. stated that her vision gradually became blurry, but was unsure of time-frame. She denied any pain. She c/o occasional floaters, but denied photopsia.

POH: Mild NPDR OU

PMH: Type II DM, HTN, ESRD on HD, GERD

MEDS: Glipizide, Lipitor, Pantoprazole, Phoslo, Isosorbide mononitrate, Metoprolol, Warfarin

All: NKDA

# Objective

BCVA  $\left\{ \begin{array}{l} 20/200 \\ 20/40 \end{array} \right.$

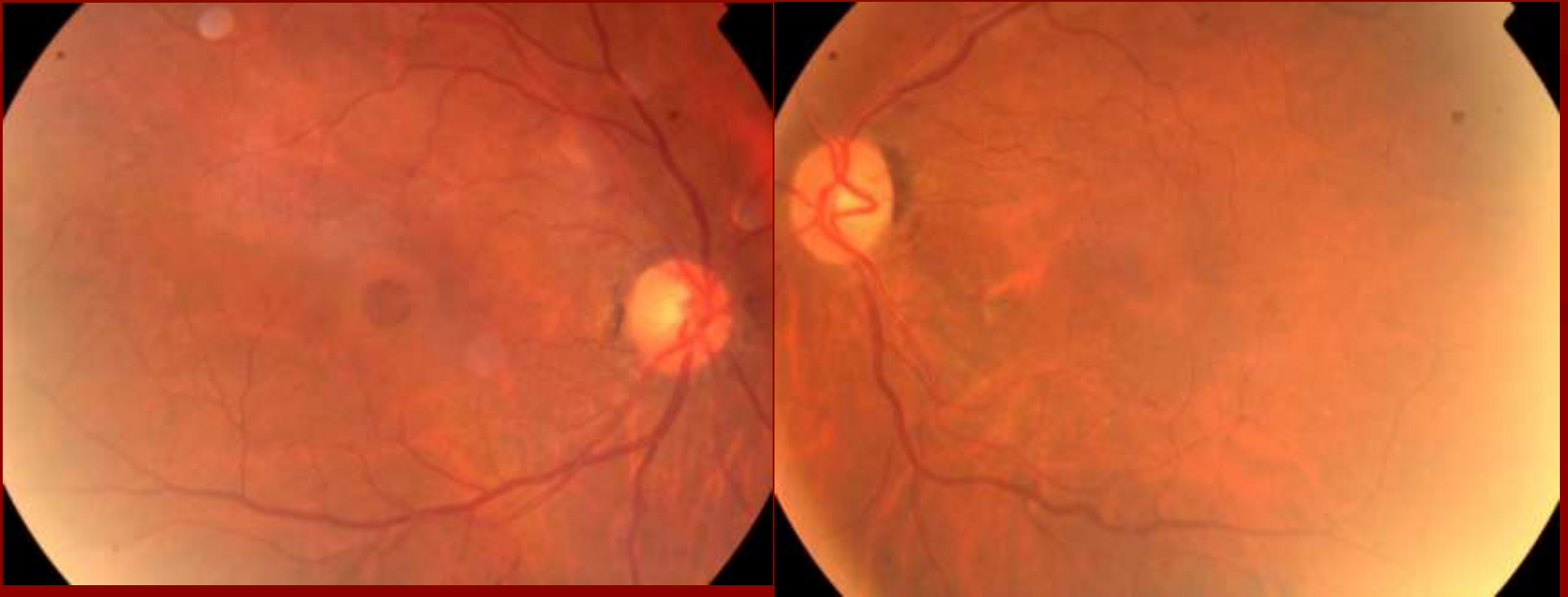
P  $\left\{ \begin{array}{l} 3 \rightarrow 2 \\ 3 \rightarrow 2 \end{array} \right.$   $\emptyset$  RAPD

T  $\left\{ \begin{array}{l} 23 \\ 17 \end{array} \right.$

Motility: Full OU

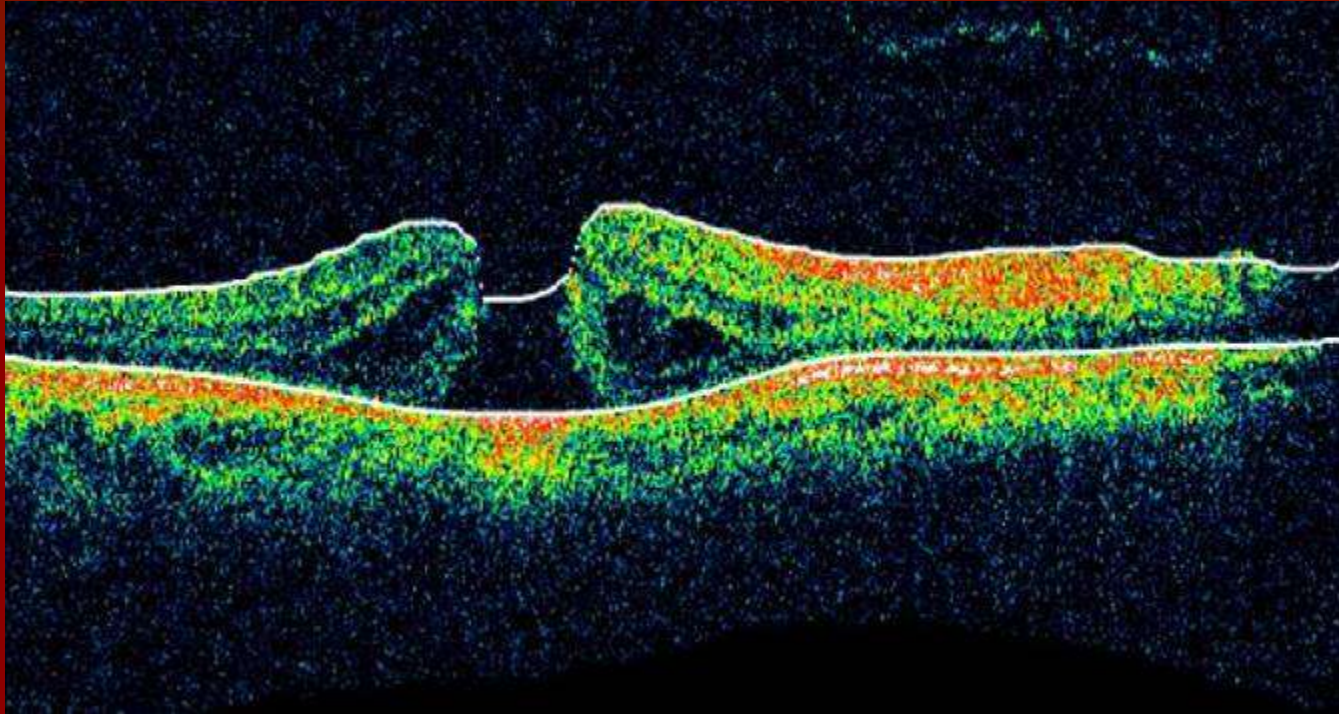
Anterior Exam: significant for 2+ NS cataracts OU

# Color Fundus Photos



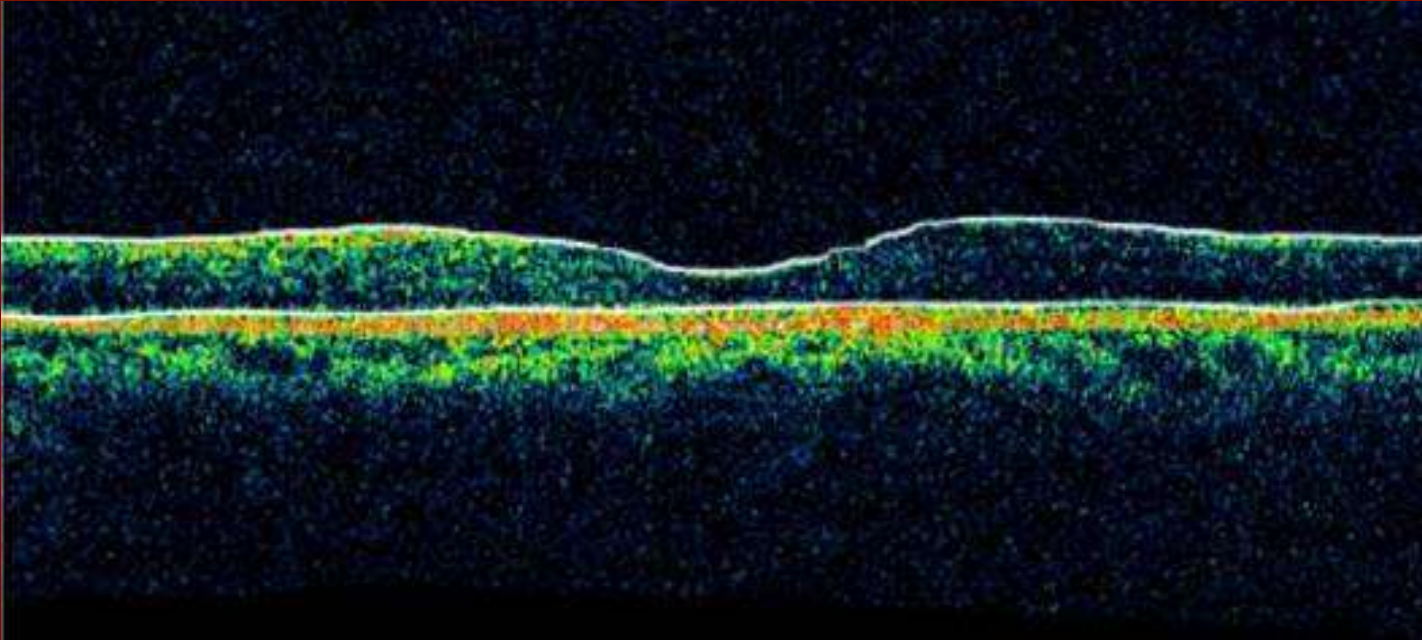
Color fundus photos demonstrates macular hole OD and few small scattered microaneurysms OU

# OCT OD



OCT OD demonstrates a full thickness macular hole

# OCT OS



# Assessment

- 78 yo AAF with a full thickness macular hole OD

# Management

- The patient underwent 25G PPV, membrane peel and gas tamponade
- On POD#1, macular hole OD almost completely closed, VA HM

# Macular Holes

- A full-thickness depletion of the neural retinal tissue in the center of the macula
- Majority are idiopathic
- Affects individuals in 6<sup>th</sup> and 7<sup>th</sup> decade (mean age 65); 2:1 F:M ratio; incidence 7.8 per 100,000 per year.
- 10%-20% develop bilateral disease, but rarely simultaneous

# Clinical Presentation

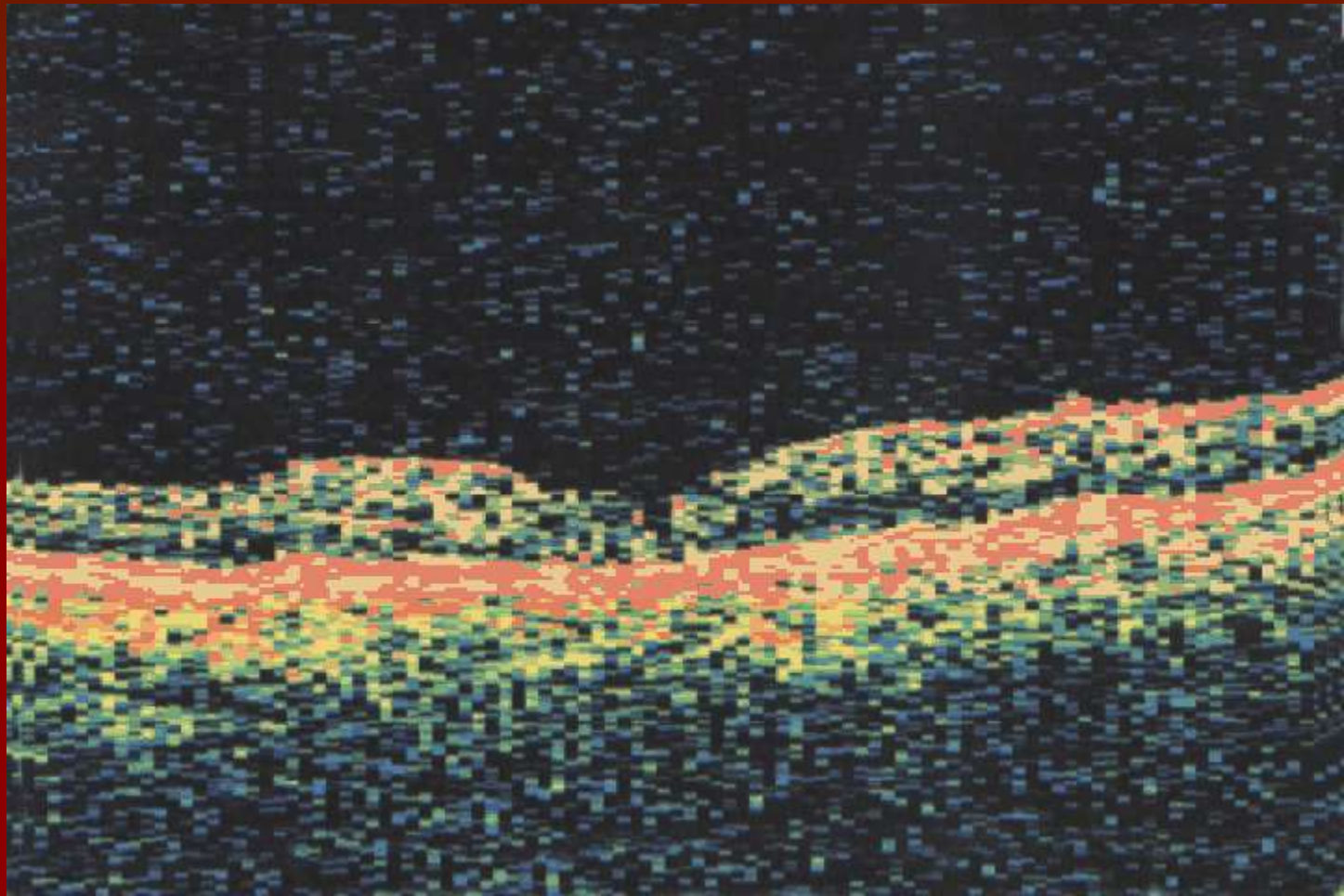
- Acute or subacute painless central visual distortion
- Visual loss often goes undetected unless cross-covering performed
- VA acuity worsens as the hole progresses over weeks to months stabilizing around 20/200-800

# Pathogenesis

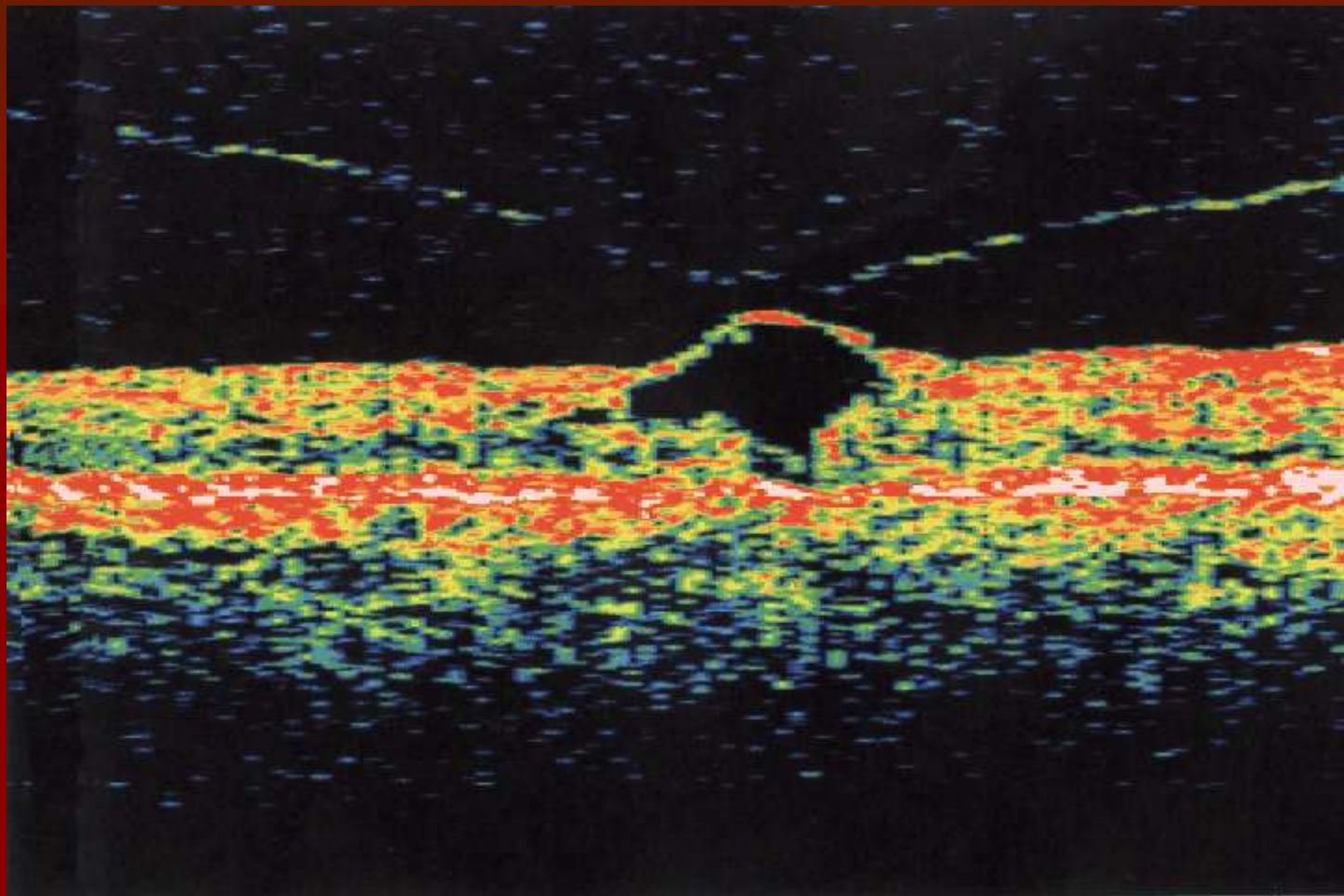
- Caused by abnormal tractional forces of the vitreous on the macula
- Begins with vitreofoveal separation and adherence of foveolar ILM to the posterior vitreous cortex
- Anterior-posterior traction usually causes hyaloid detachment from fovea
- These forces are transmitted to Muller cell cone and disrupts its structural support of foveolar photoreceptors
- Disruption of ILM and ELM becomes pivotal event

# Pathogenesis

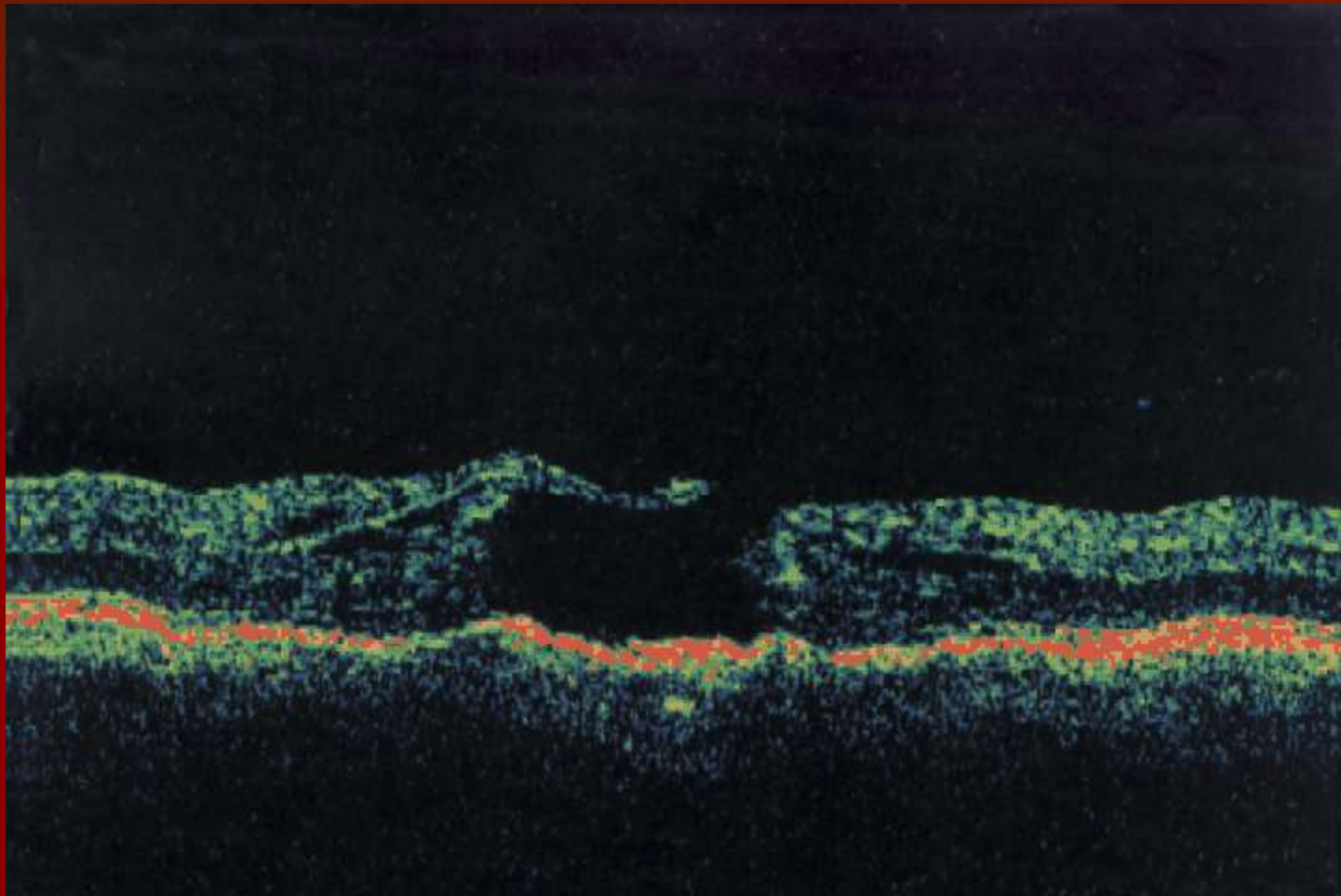
- The second phase can occur without traction
- Damage of ILM and ELM results in disruption of the seal between neurosensory retina and RPE pump causing hydration of perifoveolar retina with cystic changes subsequently enlarging the macular hole



Gentile et al. Macular hole formation,  
progression, and surgical repair BMC  
Ophthalmol. 2010 Sep 17;10:24.



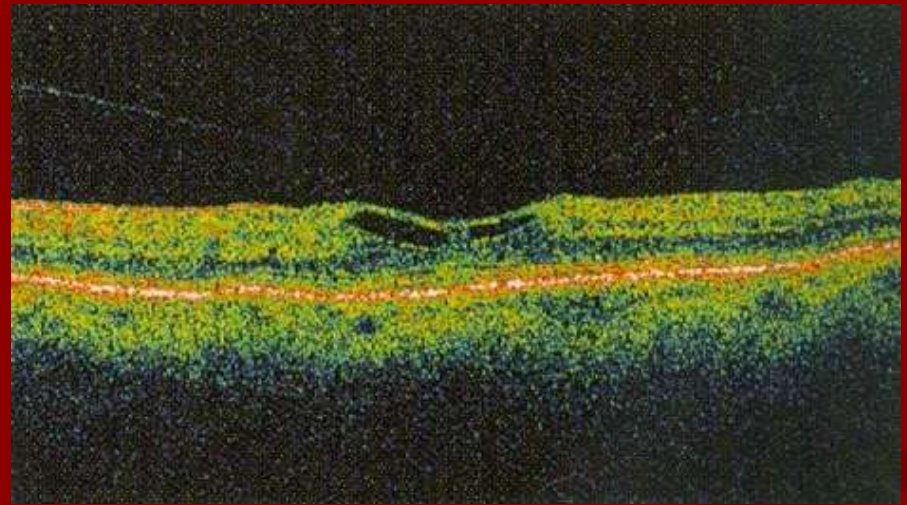
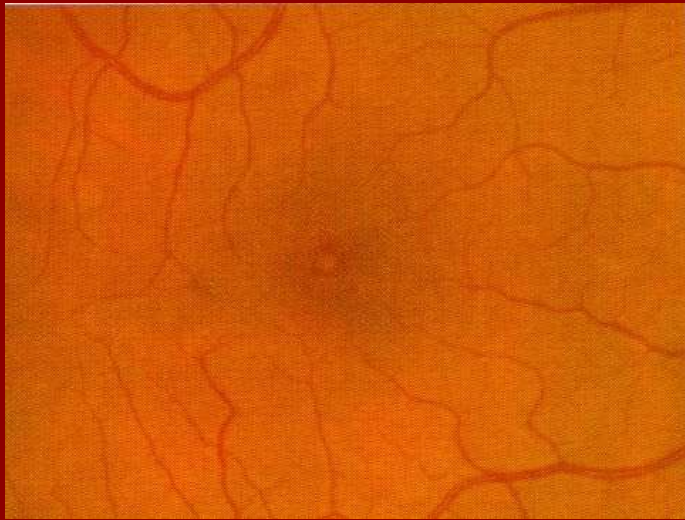
Gentile et al. Macular hole formation, progression, and surgical repair BMC Ophthalmol. 2010 Sep 17;10:24



Gentile et al. Macular hole formation,  
progression, and surgical repair BMC  
Ophthalmol. 2010 Sep 17;10:24

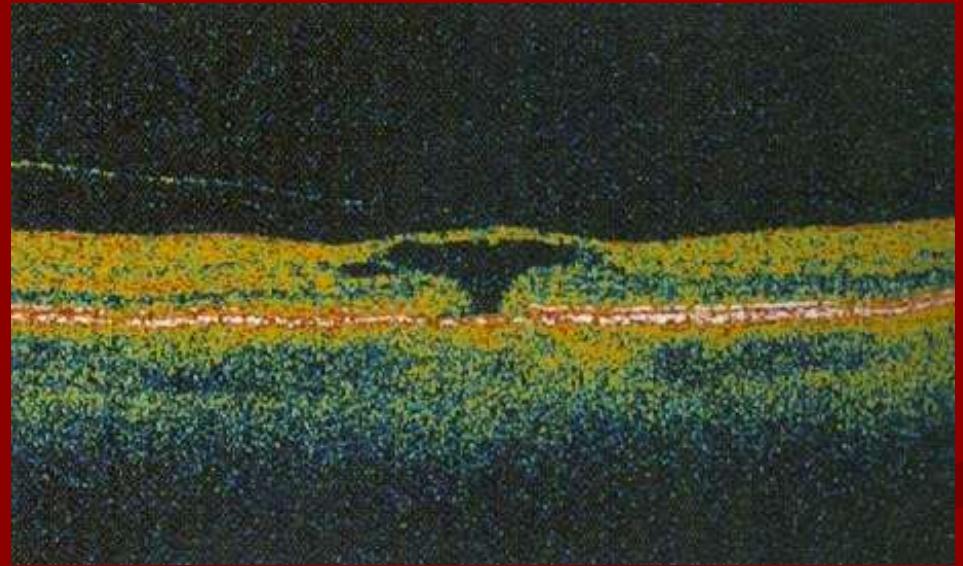
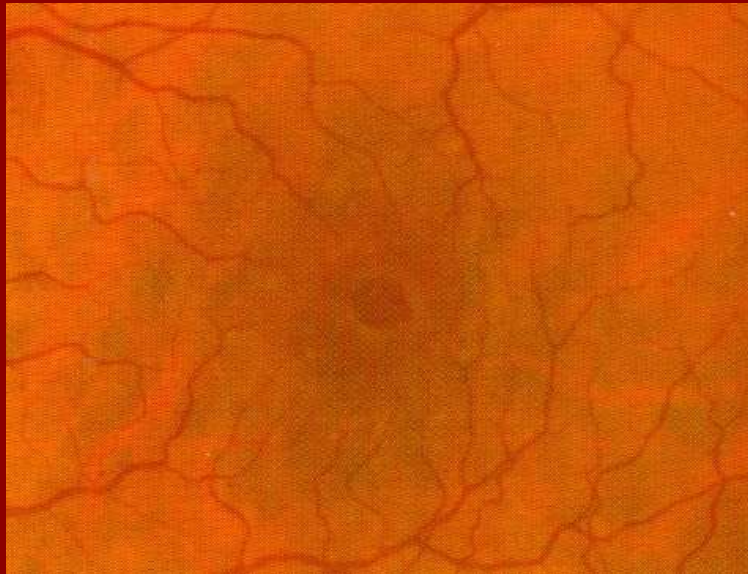
# Classification

- Stage 1A: (impending macular hole) has central yellow spot and loss of foveal depression, vitreous attached over the fovea



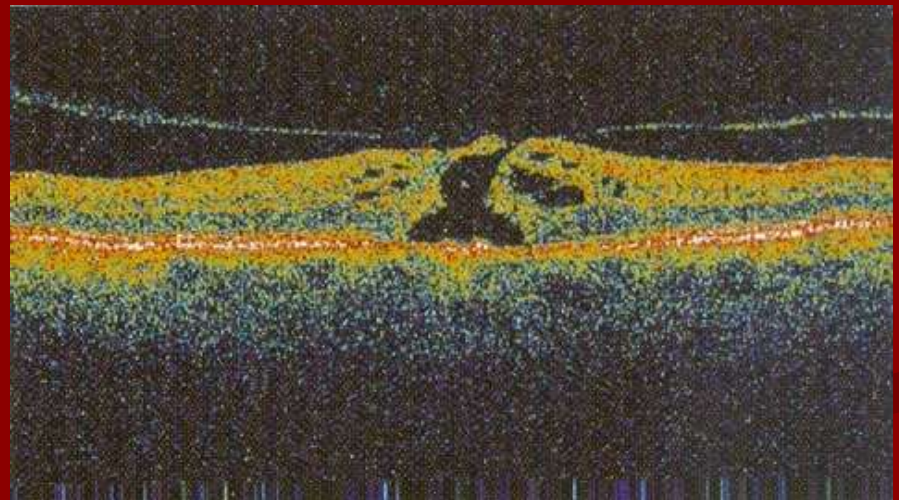
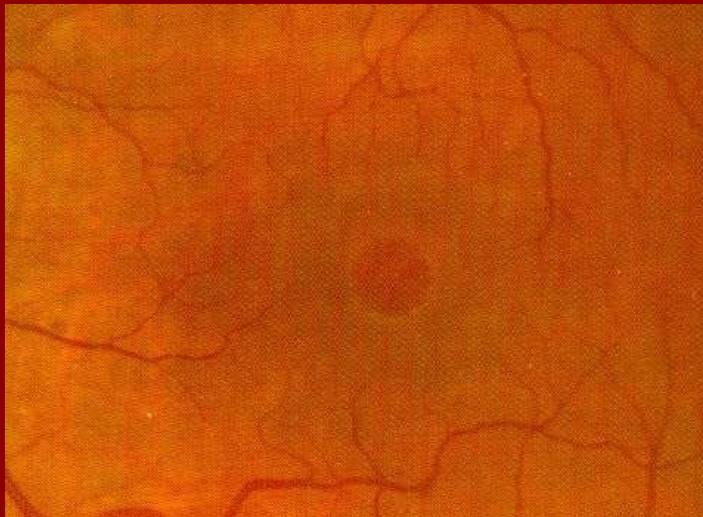
# Classification

- Stage 1B: Yellow ring with bridging interface, loss of foveal depression, vitreous attached over the fovea



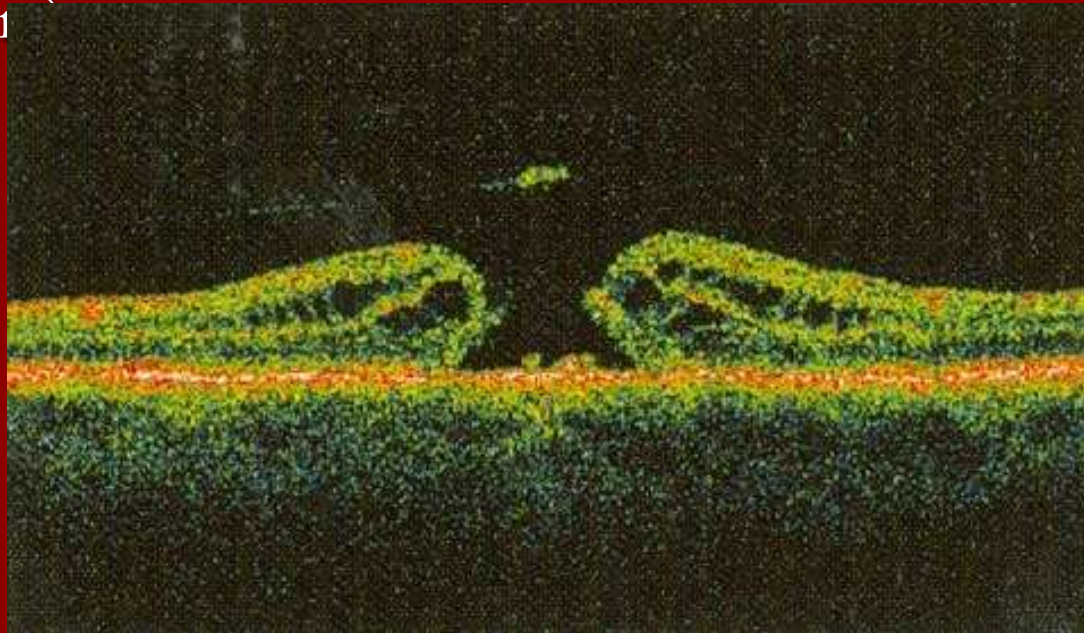
# Classification

- Stage 2: Retinal defect seen within yellow ring, may be round, crescent shaped, eccentric oval, or horseshoe in appearance. There may or may not be a prefoveal opacity (operculum)



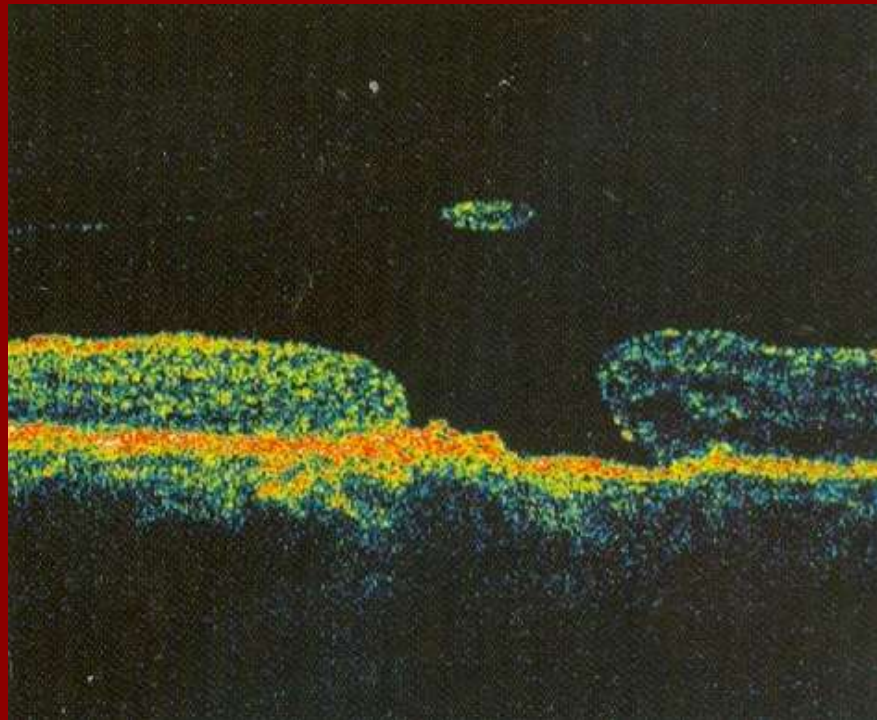
# Classification

- Stage 3: Central round full thickness retinal defect  $> 400$  microns, rim of elevated retina. Posterior hyaloid detached from the macular region, no Weiss ring present. (vitreous still attached to nerve head) There may or may not be a prefoveal opacity (operculum)



# Classification

- Stage 4: Central round full thickness retinal defect  $> 400$  microns, rim of elevated retina. The premacular vitreous is detached and a Weiss ring is present. (vitreous detached from nerve head) There may or may not be a prefoveal opacity (operculum)



# Differential Diagnosis

- Epiretinal Membrane
- Cystoid macular edema
- Vitreomacular traction syndrome
- Foveal drusen
- Choroidal neovascular membrane
- Central areolar pigment epitheliopathy
- Pattern dystrophy
- Solar retinopathy
- Central serous chorioretinopathy
- Lamellar (aborted) macular hole
- Choroiditis

# Treatment

- Stage 1 macular hole is observed with 50% spontaneous resolution rate
- Surgery should be considered for stage 1 hole in it persists for months and causes decreased VA
- Stage 2, 3 and 4 macular holes are treated with vitrectomy ( with or without ILM peel ) and sulfur hexafluoride (SF<sub>6</sub>) or perfluoropropane (C<sub>3</sub>F<sub>8</sub>) gas tamponade

# Benefits of ILM Peeling

- Objective: To determine whether internal limiting membrane (ILM) peeling is effective and cost-effective compared with no peeling for patients with idiopathic stage 2 or 3 full-thickness macular holes
- Methods: participants were randomized to ILM peeling or no peeling (1:1 ratio) in addition to phaco-vitreectomy, including detachment and removal of the posterior hyaloid and gas tamponade. Primary outcome was distance VA at six months post-surgery and major secondary outcomes included hole closure, re-operations, complications, resource use and participant reported health status, visual function and costs

Lois et al. Internal limiting membrane peeling versus no peeling for idiopathic full thickness macular hole: A pragmatic randomised controlled trial. Invest Ophthalmol Vis Sci. 2010 Nov

# Results

- 141 patient randomized, but 127 finished six-month follow-up
- VA not significantly different between two groups at six months
- Statistically significant higher rate of hole closure in the ILM peel group 85% vs. 48% at one month,  $p < 0.001$
- Fewer re-operation in the ILM peel group at six months 12% vs. 48%,  $p < 0.001$

# Conclusion

- Given the higher anatomical closure and lower re-operation rates in the ILM peel group, ILM peeling seems to be the treatment of choice for macular holes.

Thank You

# References

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