
Retinal oddities and emergencies. A rural perspective. Part 3

Quiz A solo flat, pigmented lesion is found in the temporal periphery of one eye. It has both hypopigmented lacunae and a halo. Appropriate course of action is

a/ Image the lesion and review in 6 months
b/ Assess for FAF hypoautofluoresence with fluorescence of only the lacunae & halo
c/ Refer to GP on suspicion of Gardner's syndrome
d/ Refer to retinal Ophthalmologist as they are difficult to tell between typical & atypical
e/ all of the above ✓

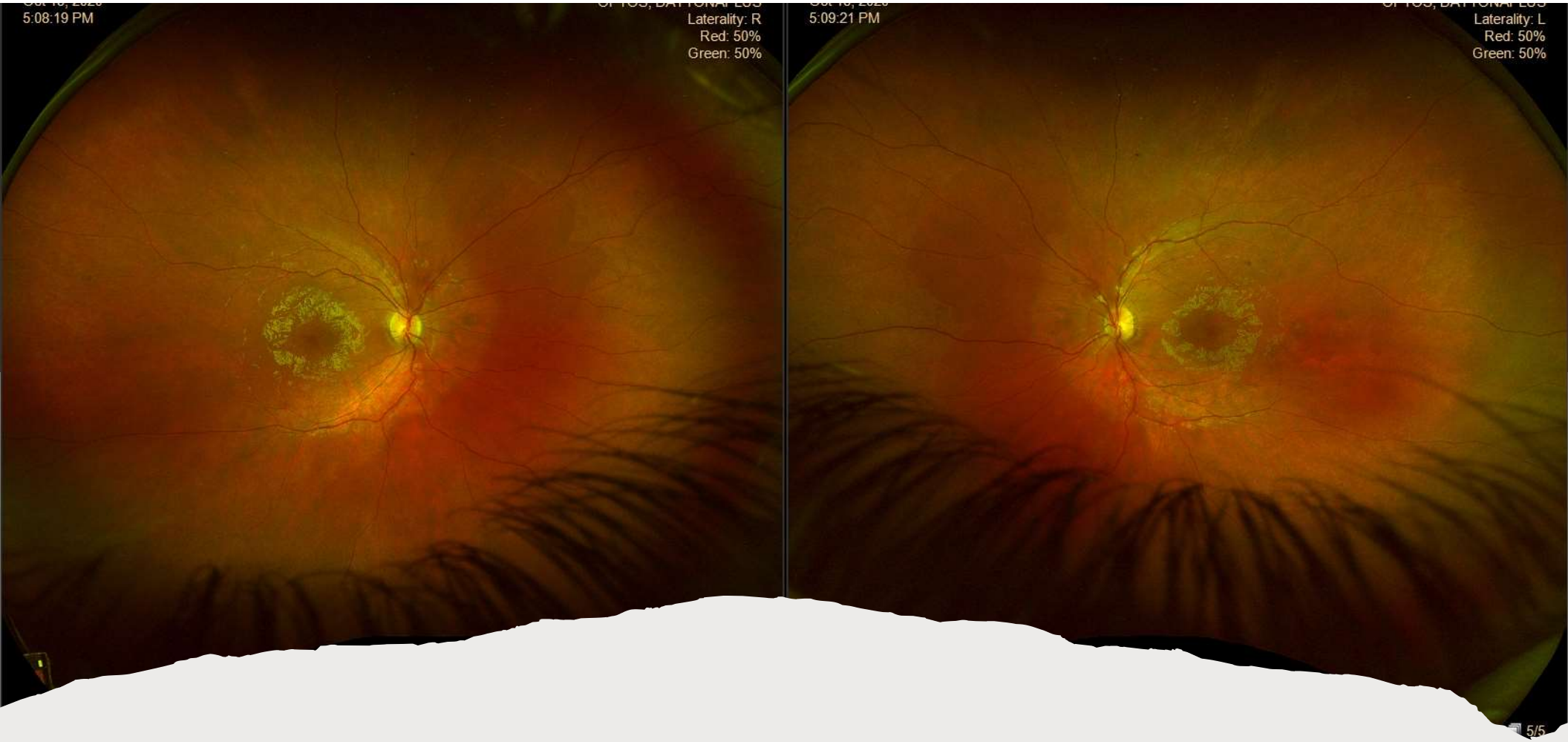
Case B. Further follow up post break out. Mr. J.S. Presented May 2021, age 76

POST BREAK OUT

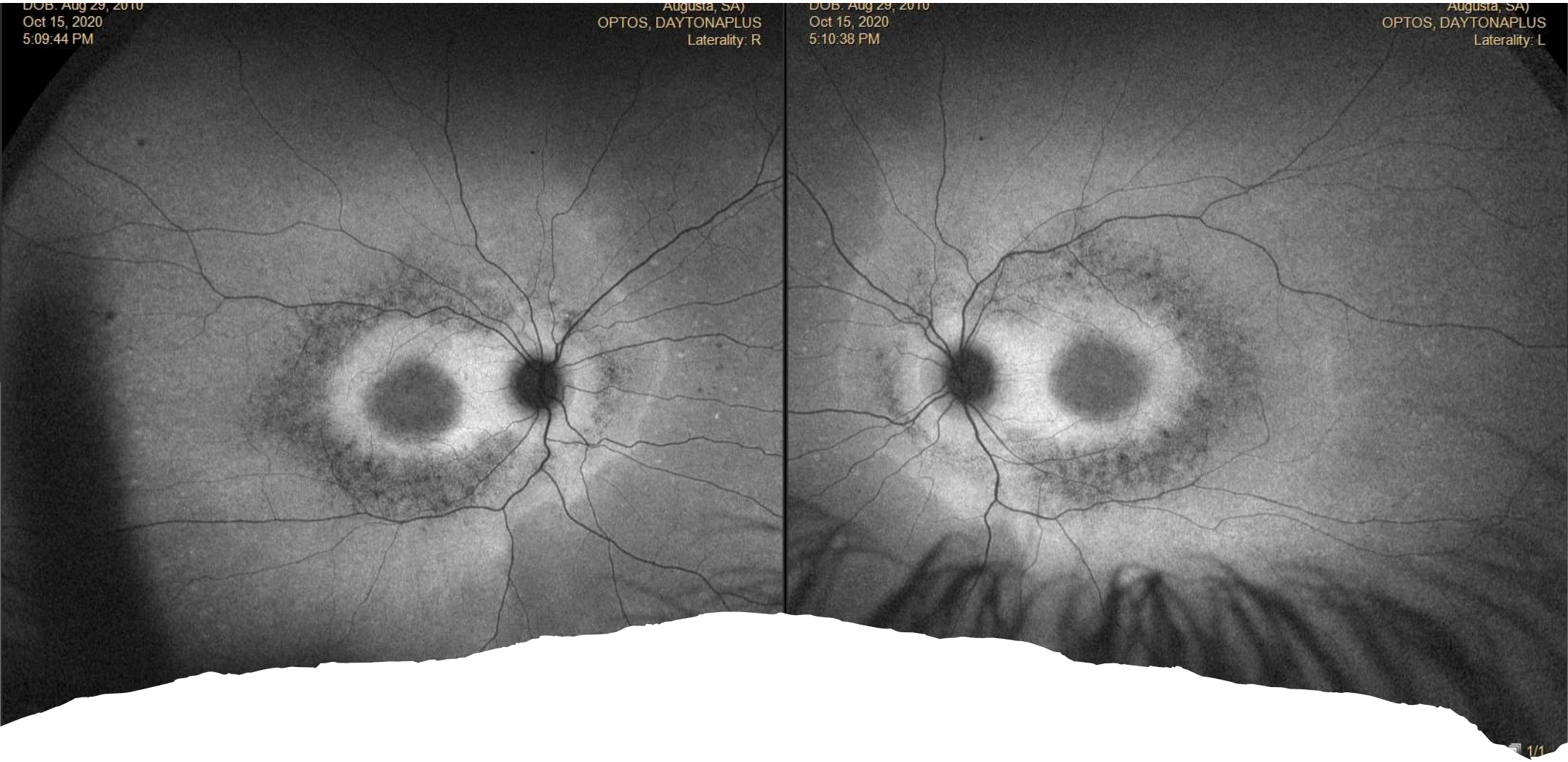
- Cancel follow appt with orthoptist. Stop timolol. Cancel ophthal review appt.
- Send carefully worded letter to Ophthal.
- GP letter suggesting second probable cadiovascular event. Examination internal carotid. Educate patient of heart attack and stroke risk.
- Review 2 months and glasses with prism in residual deviation remains

Case C. Post Breakout follow ON. Master K.M. Review 15/10/2021

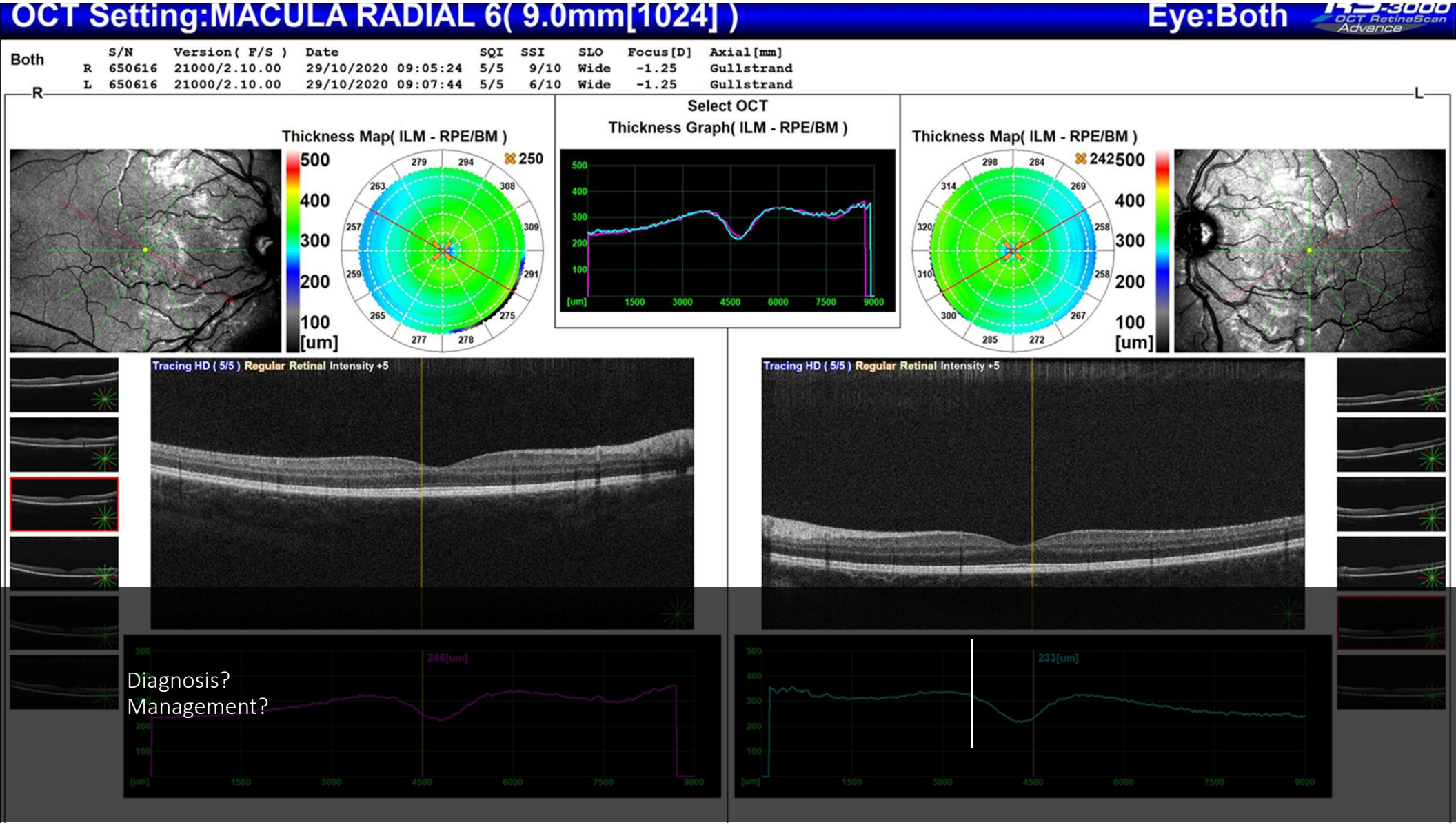
- PC: seen by paediatric Ophthalmologist. Concluded eye examination normal aside VA of 6/15 each eye. Suggested behavioural element and to work with paediatrician. Today is scheduled 6 month review from date referral sent.
- Examination:
  - VA: RE 6/39, LE 6/35 (eccentric fixation, single letter)
  - Anterior: unremarkable.
  - Posterior: **see next slides.**

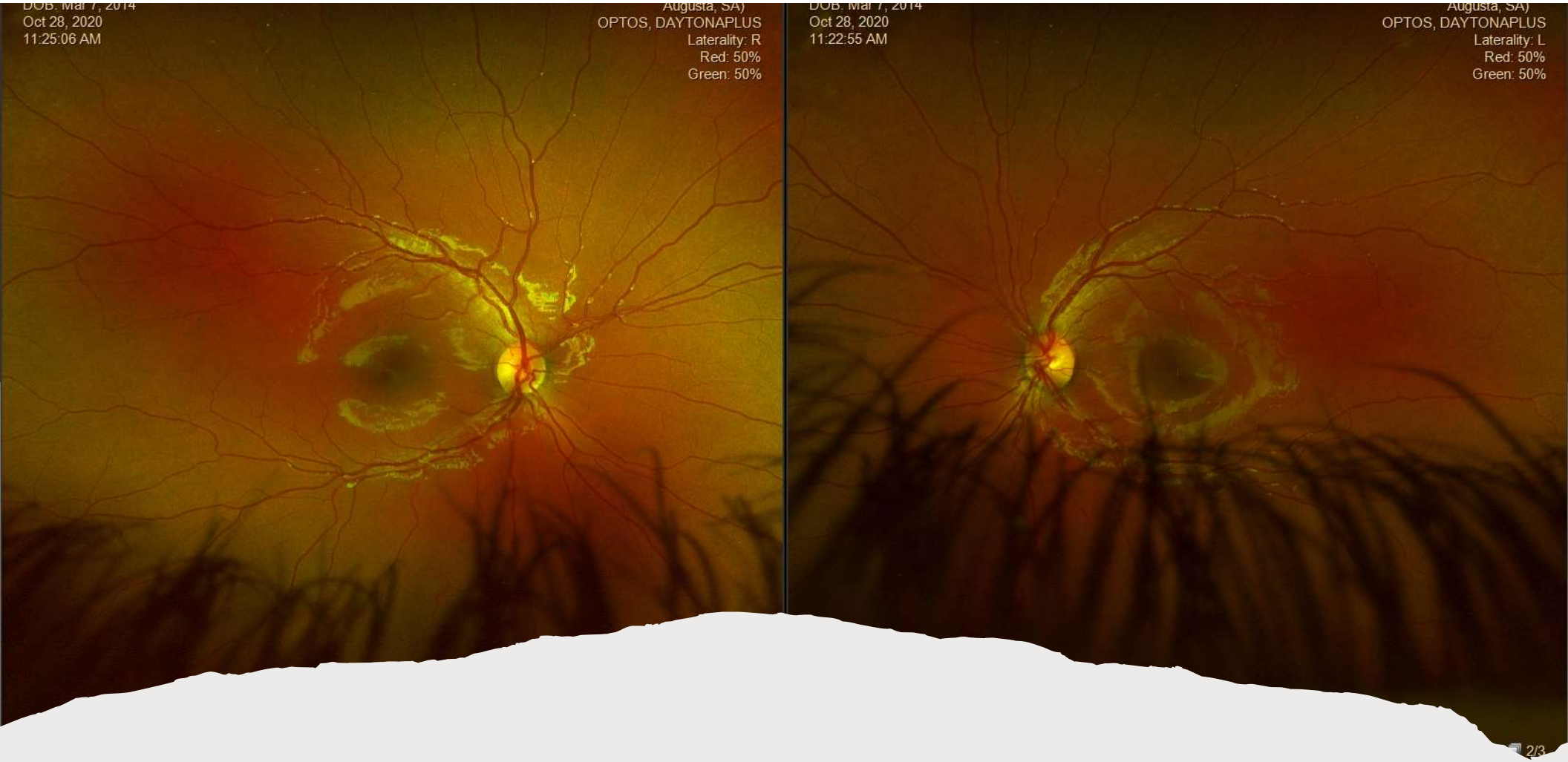

5:08:19 PM
Laterality: R
Red: 50%
Green: 50%
5:09:21 PM
Laterality: L
Red: 50%
Green: 50%
5/5

OPTOS Image

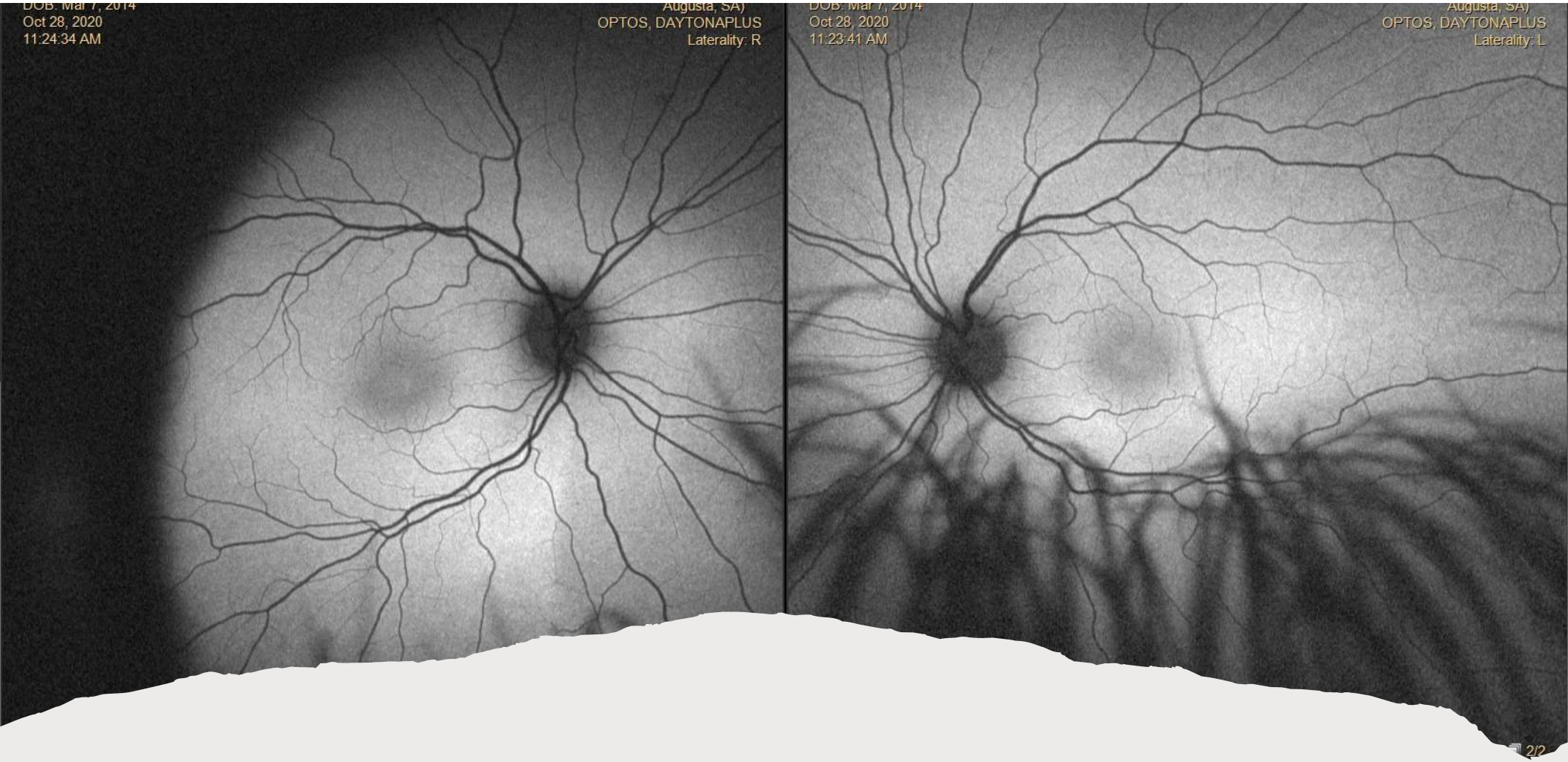


Case C. OPTOS Image FAF

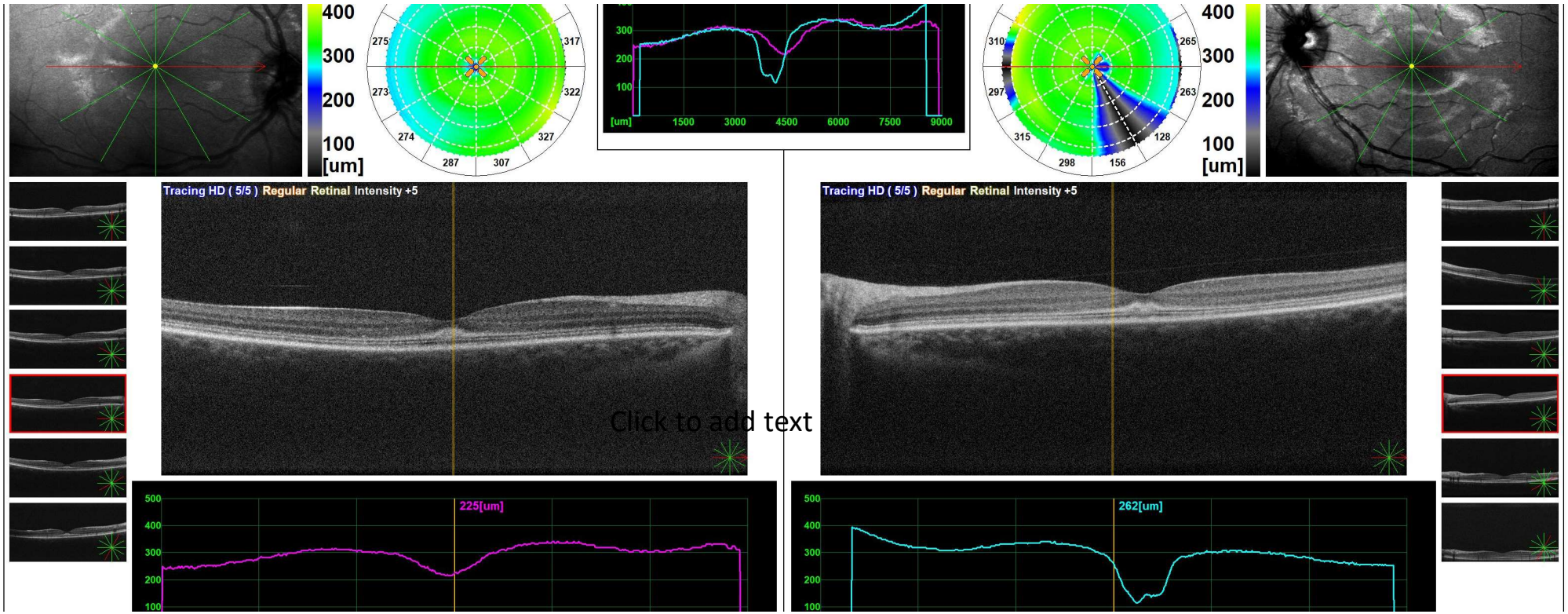

OCT Setting:MACULA RADIAL 6(9.0mm[1024])
Eye:Both
RS-3000 OCT RetinaScan Advance
Both
S/N Version(F/S) Date SQI SSI SLO Focus[D] Axial[mm]
R 650616 21000/2.10.00 29/10/2020 09:05:24 5/5 9/10 Wide -1.25 Gullstrand
L 650616 21000/2.10.00 29/10/2020 09:07:44 5/5 6/10 Wide -1.25 Gullstrand
R
L
Thickness Map(ILM - RPE/BM)
Select OCT
Thickness Graph(ILM - RPE/BM)
Thickness Map(ILM - RPE/BM)
500
400
300
200
100
[um]
Tracing HD (5/5) Regular Retinal Intensity +5
Tracing HD (5/5) Regular Retinal Intensity +5
246[um]
233[um]
Diagnosis?
Management?



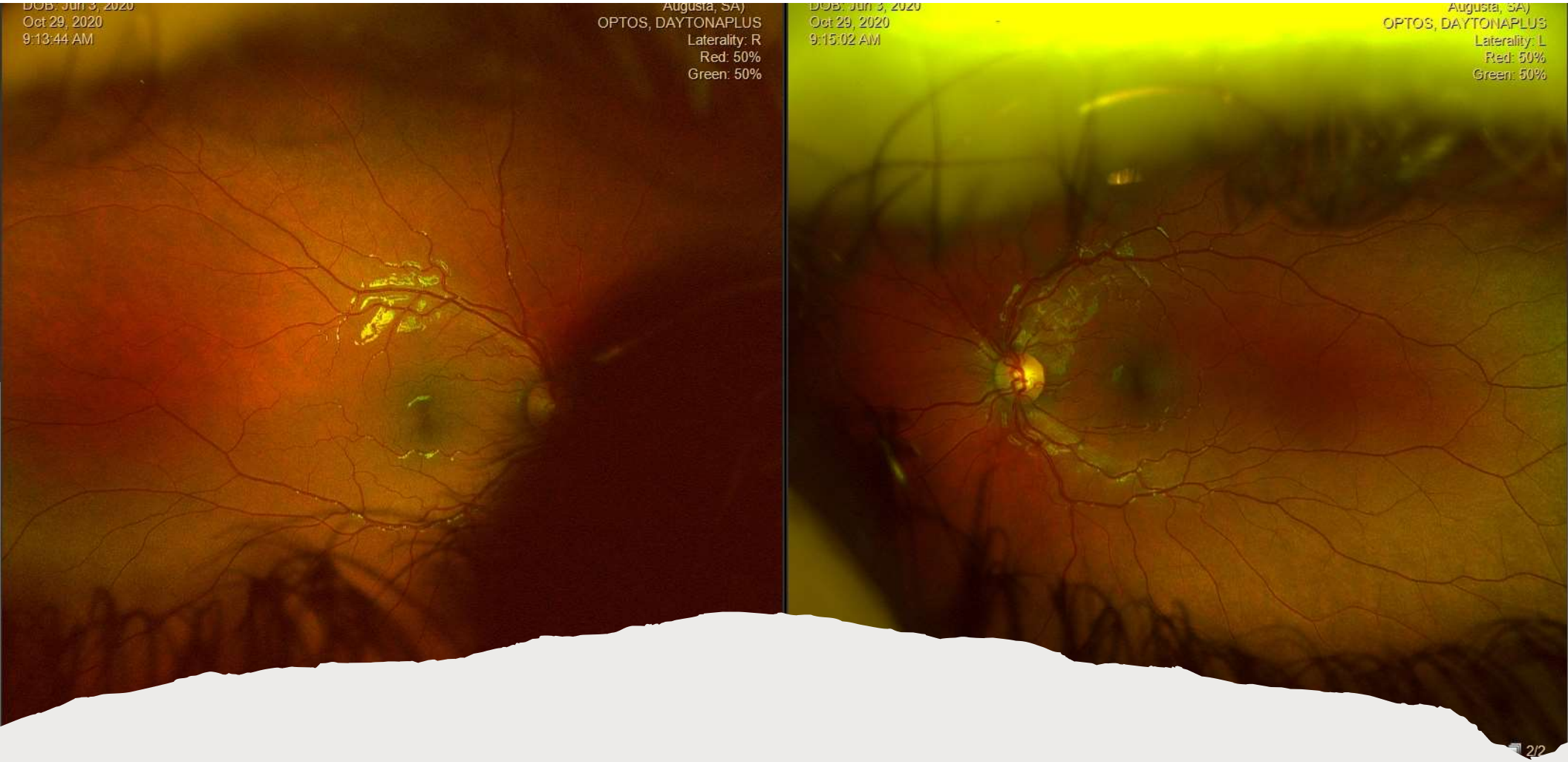
Master M.K.'s younger sibilings OPTOS



Master M.K.'s younger sibilings OPTOS red free

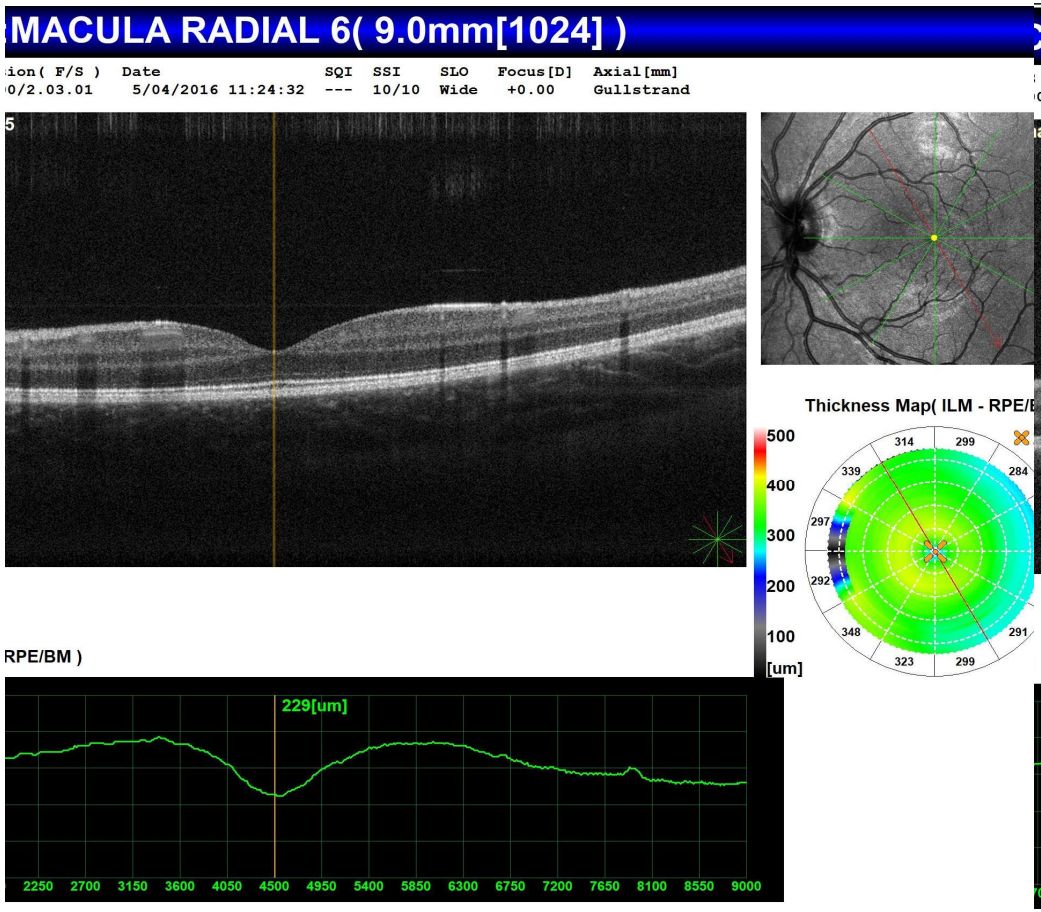


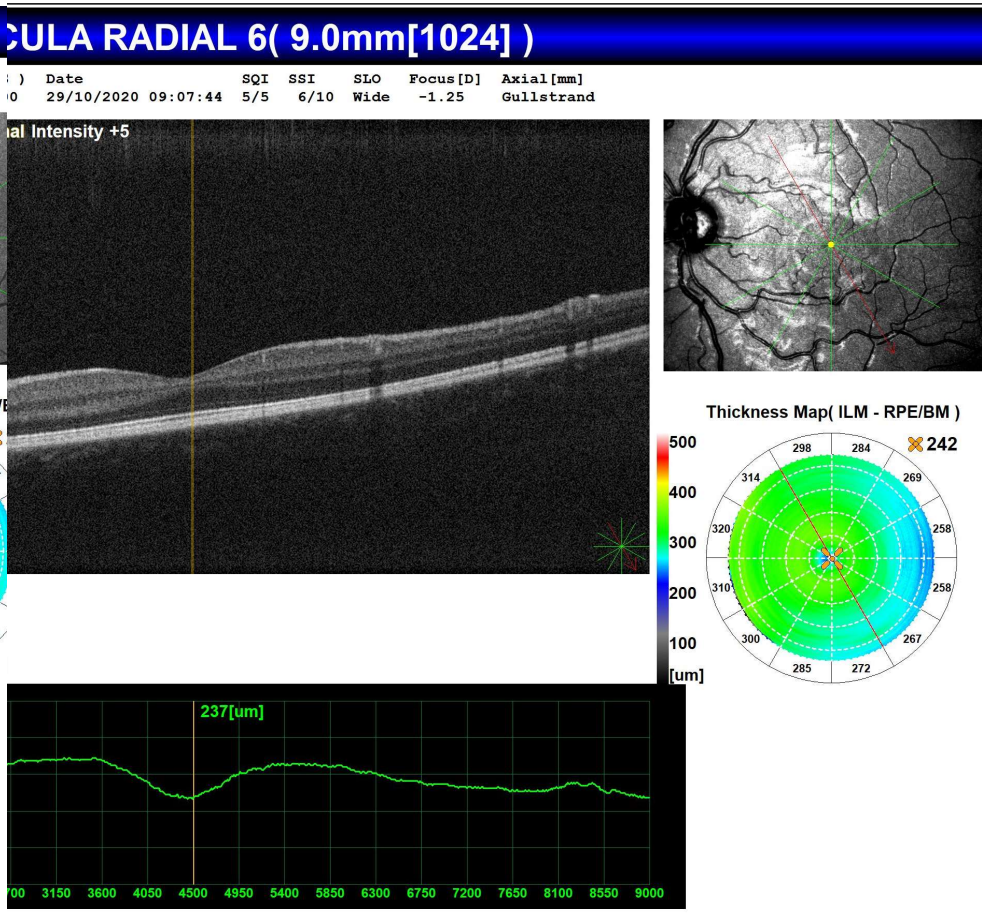
Master M.K.'s younger siblings OCT



Master M.K.'s younger sibiling #2 OPTOS

Comparing normal 4 year old to younger sibiling of Mr K.M

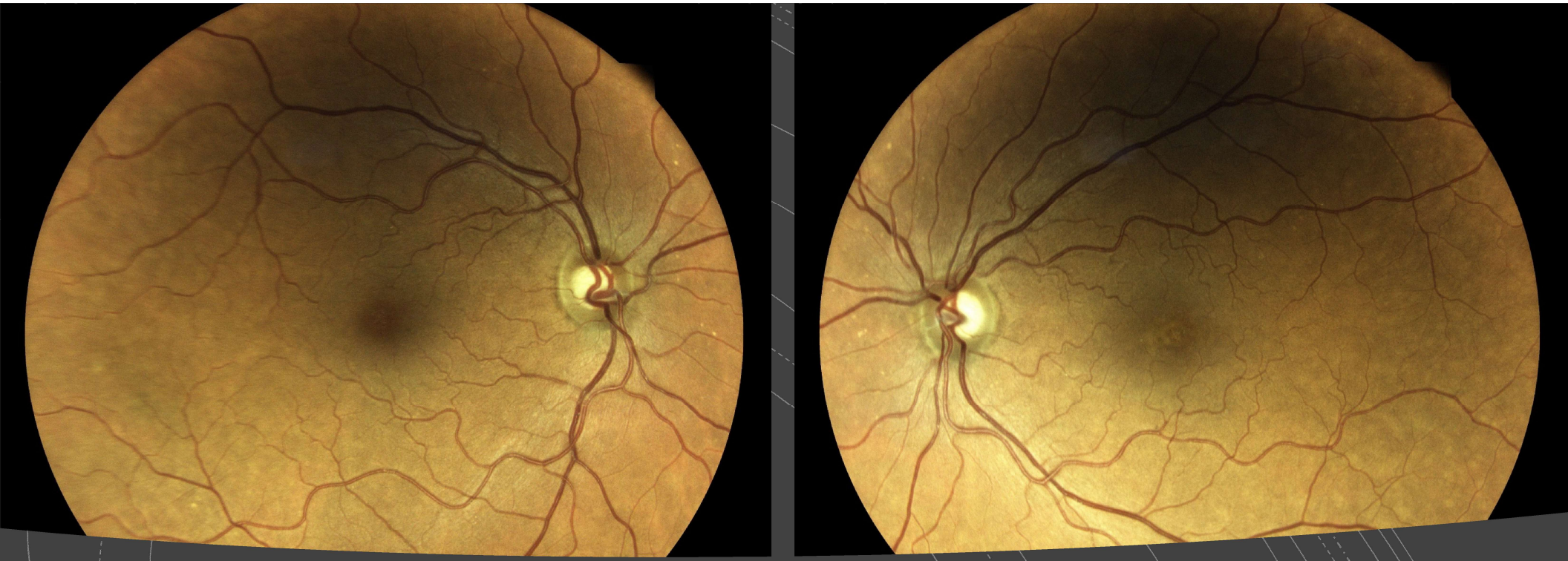




Areas of Hypoautofluoresence

Case C. Post Breakout follow ON. Master K.M. Review 15/10/2021

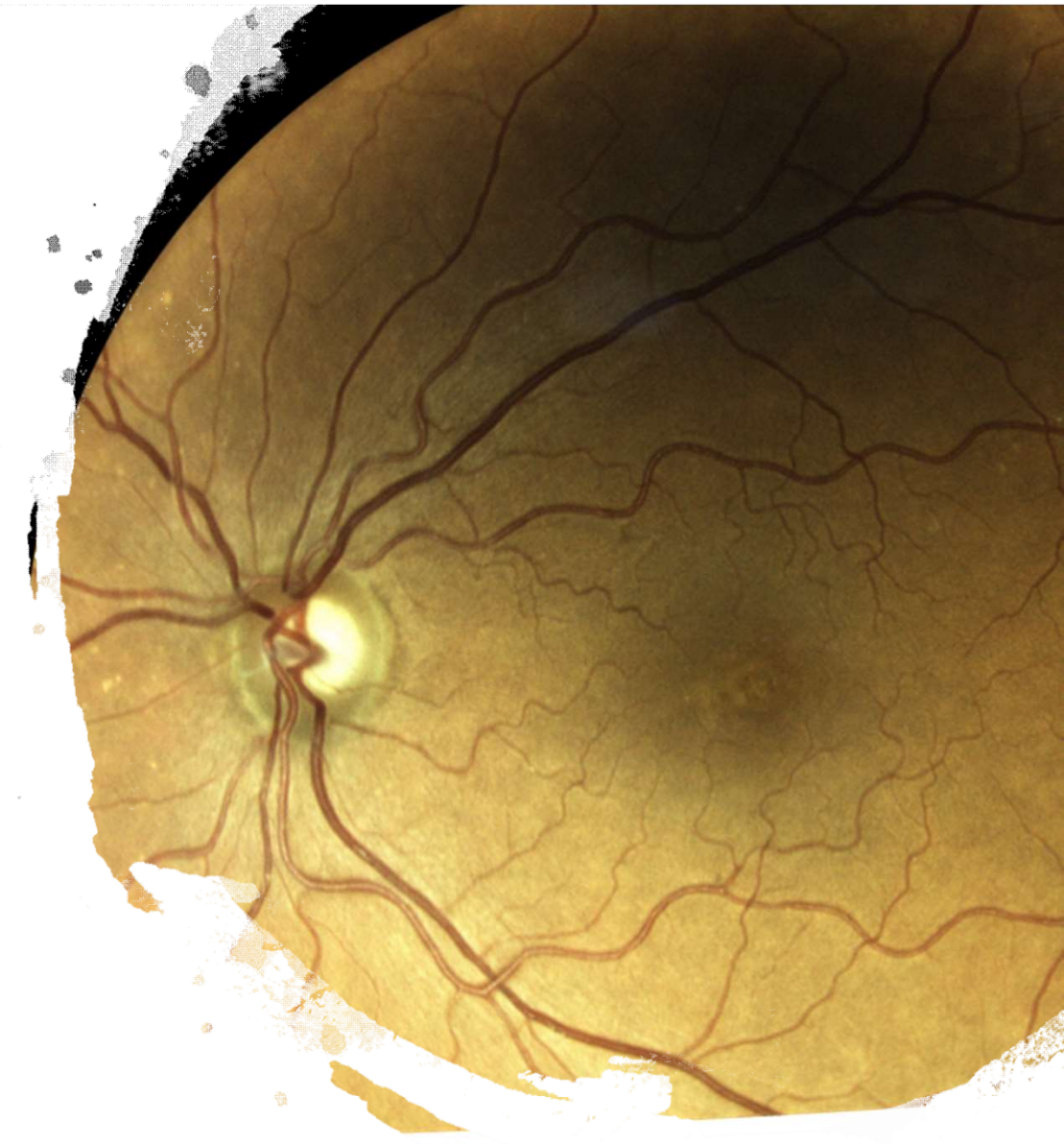
- Paediatric Ophthalmologist. Not sure how missed it but shows can happen to anyone.
- Lessons for us all, careful of malingering, good history helps a lot.
- Suspect cone –rod dystrophy
- **Genetics back. KM and younger sister both Stargarts. 4 year old not yet tested?**



Mrs. S.C.

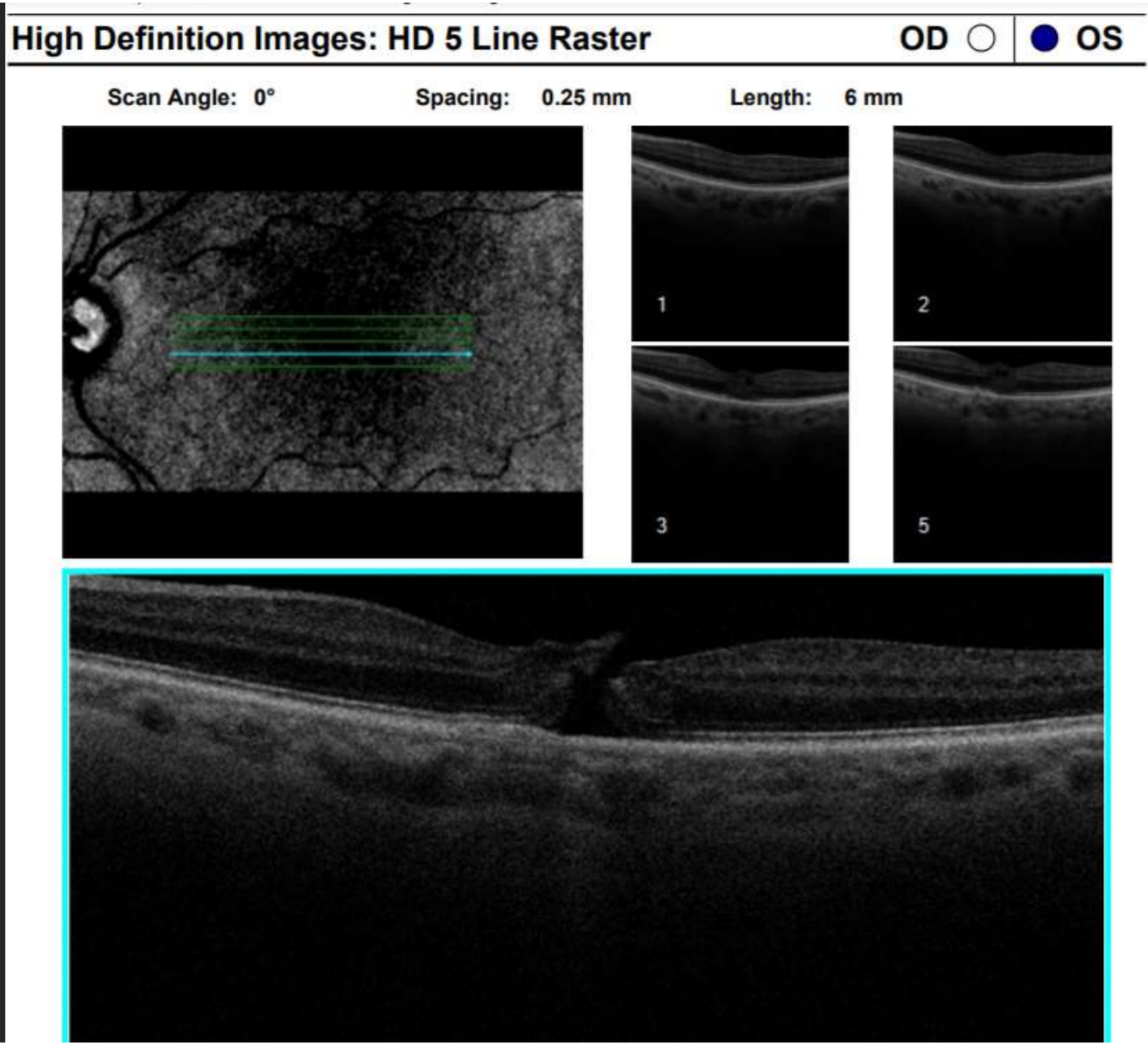
Mrs. S.C. aet 65

- Retired radiographer
- Font larger over last 6/12
- +7.00/-1.25 x 100 6/6 +7.25/-0.75 x 72.5 6/19
- PI OU
- ACLO narrow AC
- LE amsler +ve
- IOP 19 R 22 L



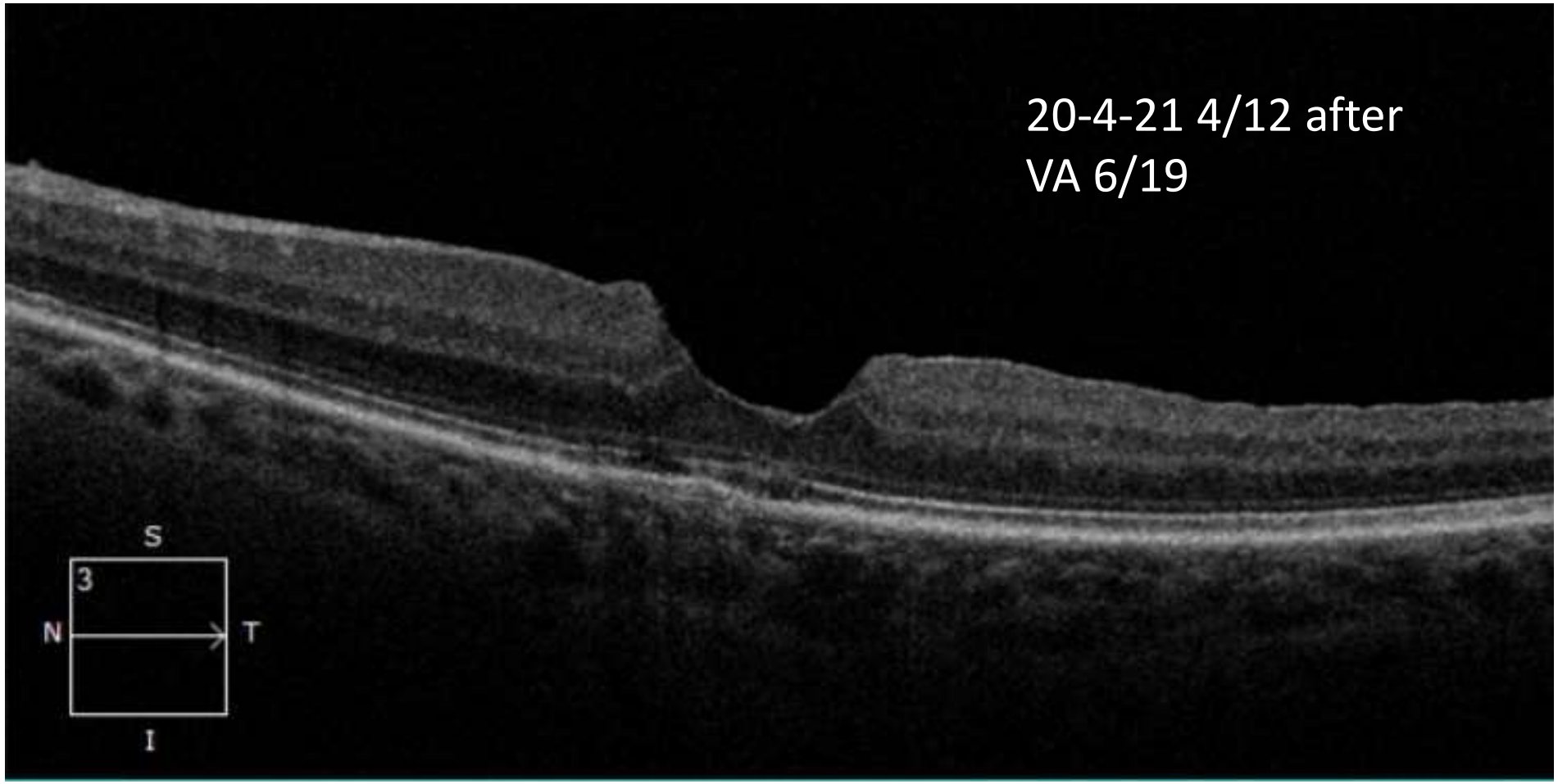
Mrs.S.C.

Urgency Or emergency?



So it is macula, Why include it?

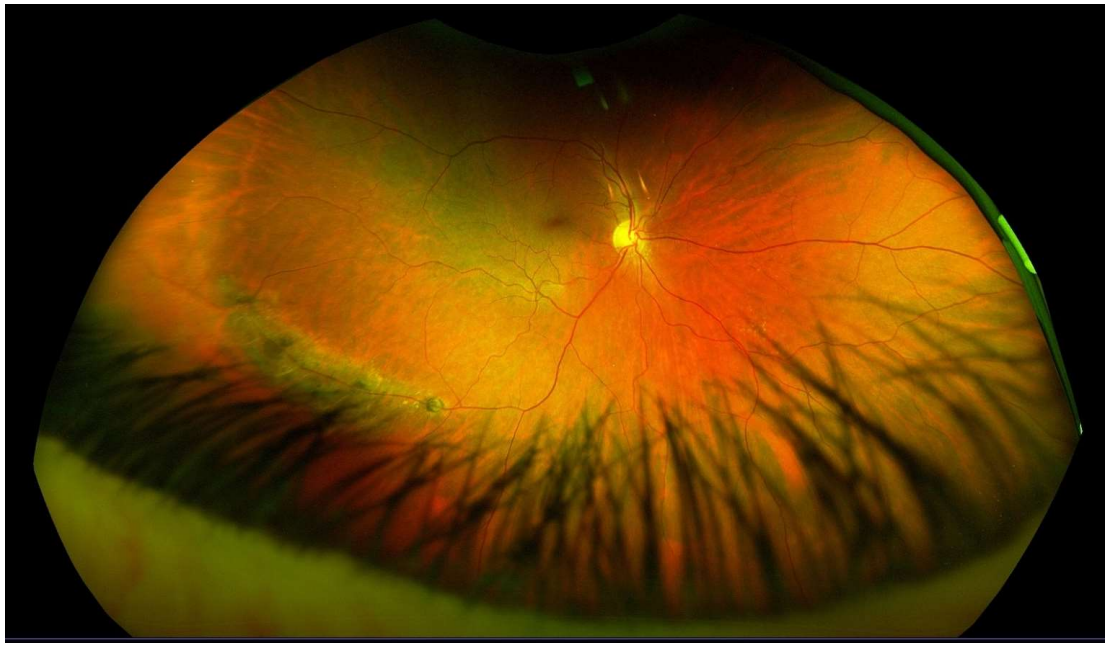
- Sx to repair macular hole scheduled 1/12 later
- High hyperope, shallow AC
- ILM peel, Vitrectomy, gas tamponade = AC closing and high IOP
- Cataract combined procedure
- The bubble acts as an internal, temporary bandage that holds the edge of the macular hole in place as it heals.
- Chin Face down position for 1/52
- Operate within 3/12 if acute or sooner
- PF or maxidex 4/52 and IV triamcinolone if CMO
- HIGH RATE RETINAL DETACHMENS IN POST OP PERIOD FOR UPTO 2 YEARS

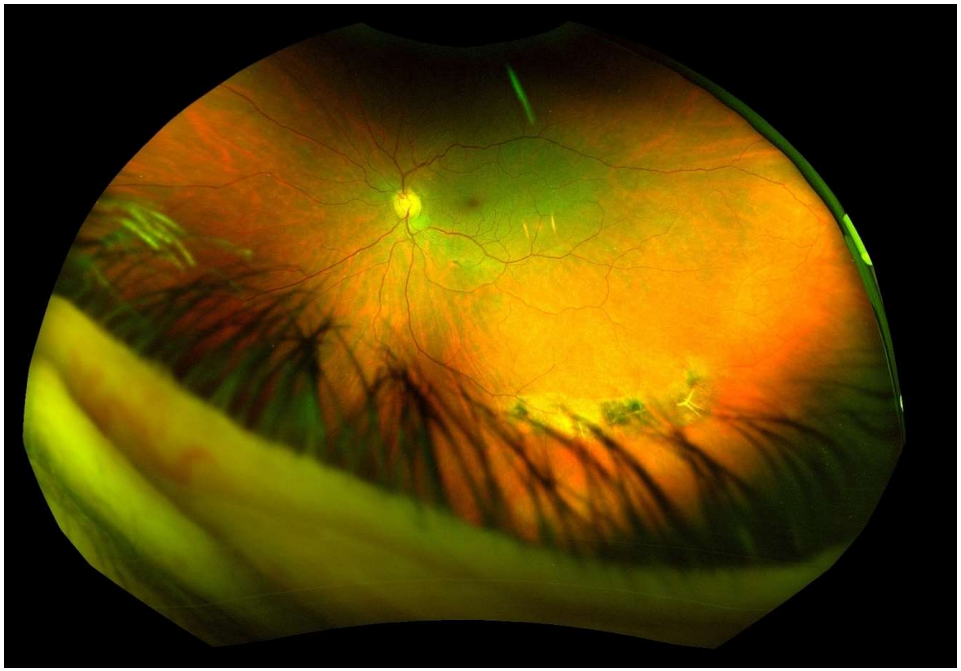

20-4-21 4/12 after
VA 6/19
S
3
N
T
I

Mrs. A.D. aet 58 17-7-20

Binocular flashing temporal side past 3/12
COVID couldn't attend
Vision fine
Esp in dark & head movement
Prior floaters, none recently
Prior Hx retinal issues

Mrs. A.D. What is this. Are we worried?





Lattice Degeneration and Myopia

Sclerosed vessels, irregular pigment, thin retina, atrophic holes

- General population 6-8 %
- Myopes → 25% including children
- Higher prevalence of LD in high myopes – greater than 35%

Hard to find studies showing rates lattice in different bands of myopia but higher myopia higher levels lattice.

Lattice Degeneration progress to RD?

Sclerosed vessels, irregular pigment, thin retina, atrophic holes

Lattice most important risk for RD ??

- Up to 60% in cases of rRD have LD via VR traction and stronger vitreal adhesion Sasaki et al 1995; Tilllery et al 1976; Byer 1974
- Search for tears/holes near edge or outside of lattice
- Those IN LD often pigmented over and SAFE
- Fellow eye?
- OCT shallow elevation like new OPTOS OCT or peripheral OCT although tricky to do.

Lattice Degeneration progress to RD?

Pathophysiology

It is well documented that lattice degeneration increases the risk of retinal tears or subsequent retinal detachments. This occurs via two mechanisms: (1) atrophic retinal hole or (2) retinal tear

Atrophic Retinal Hole

Atrophic round holes occur within the substance of the lattice lesion and likely represent the end-stage of retinal thinning and subsequent dissolution of tissue. [2] These holes rarely progress to a clinical retinal detachment though may develop a small cuff of subretinal fluid stemming from the overlying liquefied vitreous. As this cuff typically remains stable over time, it is believed that the overlying liquid vitreous does not communicate with the greater vitreous body.[2] Overall, a small percentage of retinal detachments are caused by lattice degeneration with atrophic holes (2.8% from one study).[10] And conversely, the risk of a retinal detachment developing in a patient with lattice degeneration associated with atrophic hole has been estimated to be less than 0.3%.[2] Interestingly, these retinal detachments occur more frequently in young, myopic patients, an observation possibly explained by the progressive strengthening of the bond between the retina and retinal pigment epithelium at the border of the hole with time.[2]

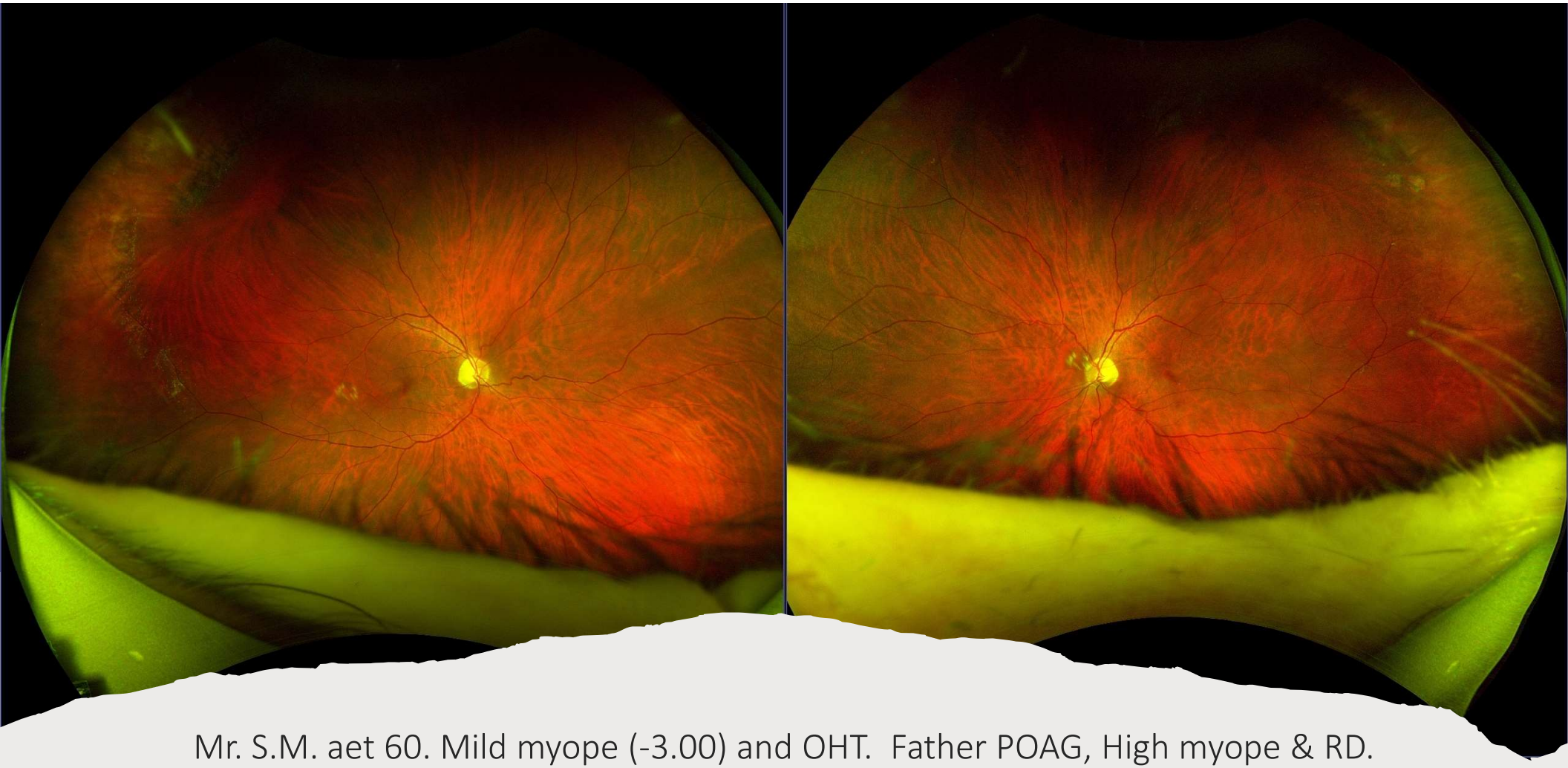
Lattice Degeneration progress to RD?

Retinal Tear

Retinal tears are believed to stem from traction at the margins of lattice lesions, an area typified by tight vitreoretinal adhesion. This traction may originate from a posterior vitreous detachment, and as such, the retinal tear is often on the posterior margin of the lattice lesion. However, not all retinal tears occurring in eyes with lattice degeneration occur adjacent to a lattice lesion, suggesting that these eyes may be at a generalized increased risk of retinal tears. Estimates for the percentage of tears that do occur adjacent to lattice lesions range widely, from to 28% to 82.5% [2] Overall, the risk of retinal tear occurring adjacent to a lattice lesion is very low, with one study suggesting a 1% incidence after 10 years.[11]

Retinal Detachment

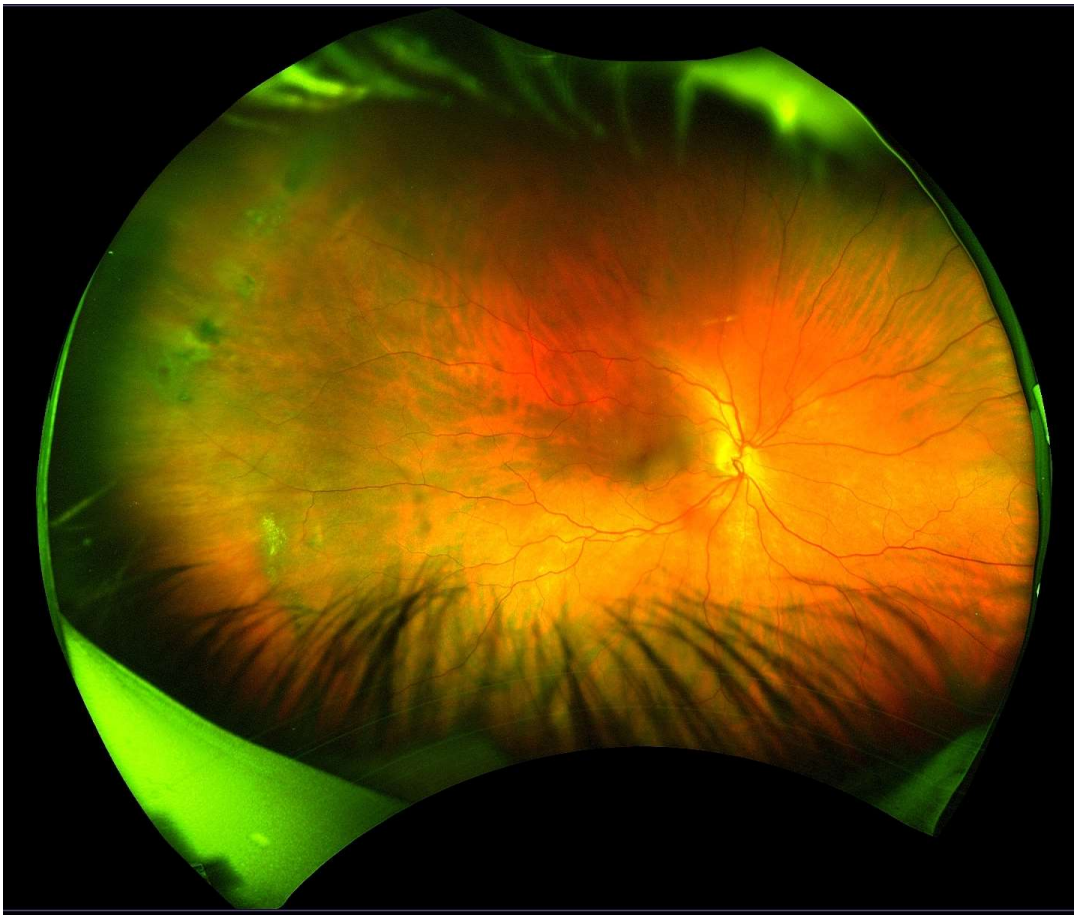
As mentioned previously, approximately 20 to 30% of patients with retinal detachment have evidence of lattice degeneration. Detachments associated with lattice degeneration more commonly occur due to retinal tears (16 to 18%) compared to atrophic round holes (range of 2.8 to 13.9%).[2][12][10] Studies measuring the converse (i.e., the risk of a retinal detachment developing in a patient with lattice degeneration though not necessarily from a lattice lesion-associated retinal break) have shown a fairly low overall risk ranging from 0.3 to 0.5%.[13]



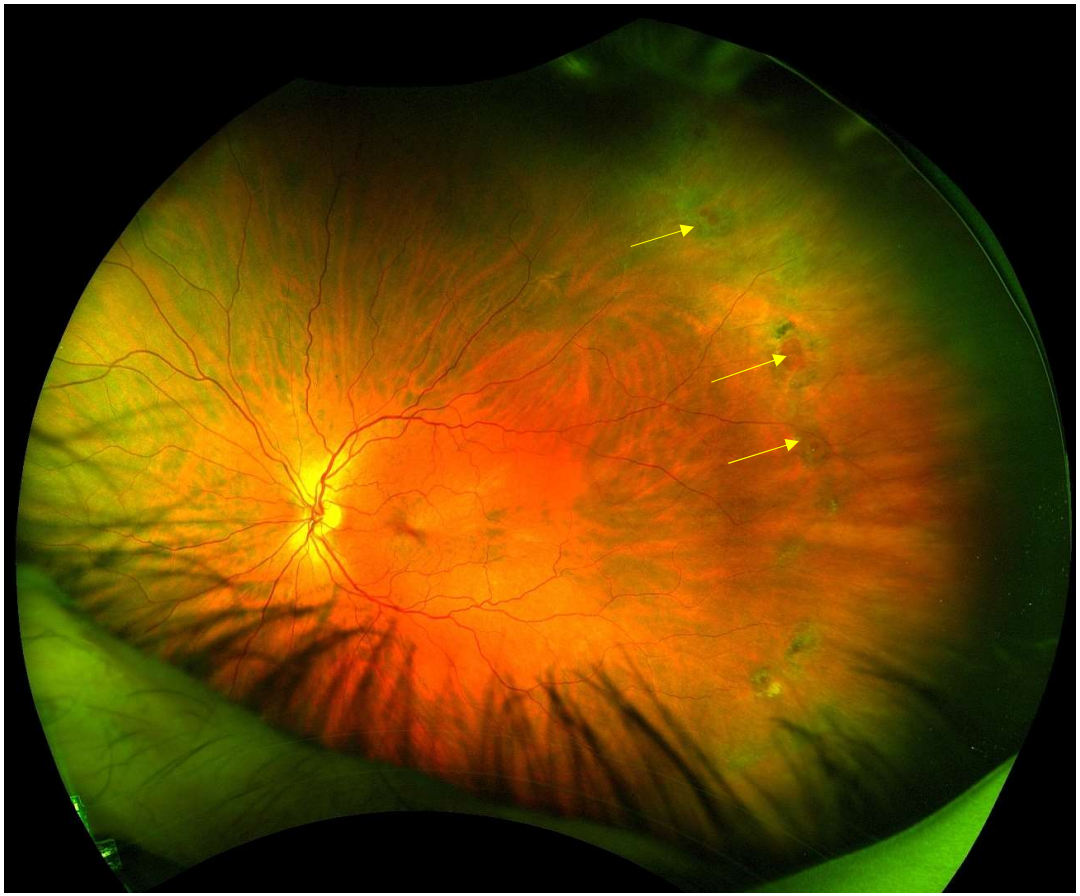
Mr. S.M. aet 60. Mild myope (-3.00) and OHT. Father POAG, High myope & RD.
No RD Symptoms or signs, vision good no PVD (yet)
"What can I do to not progress to retinal detachment?"

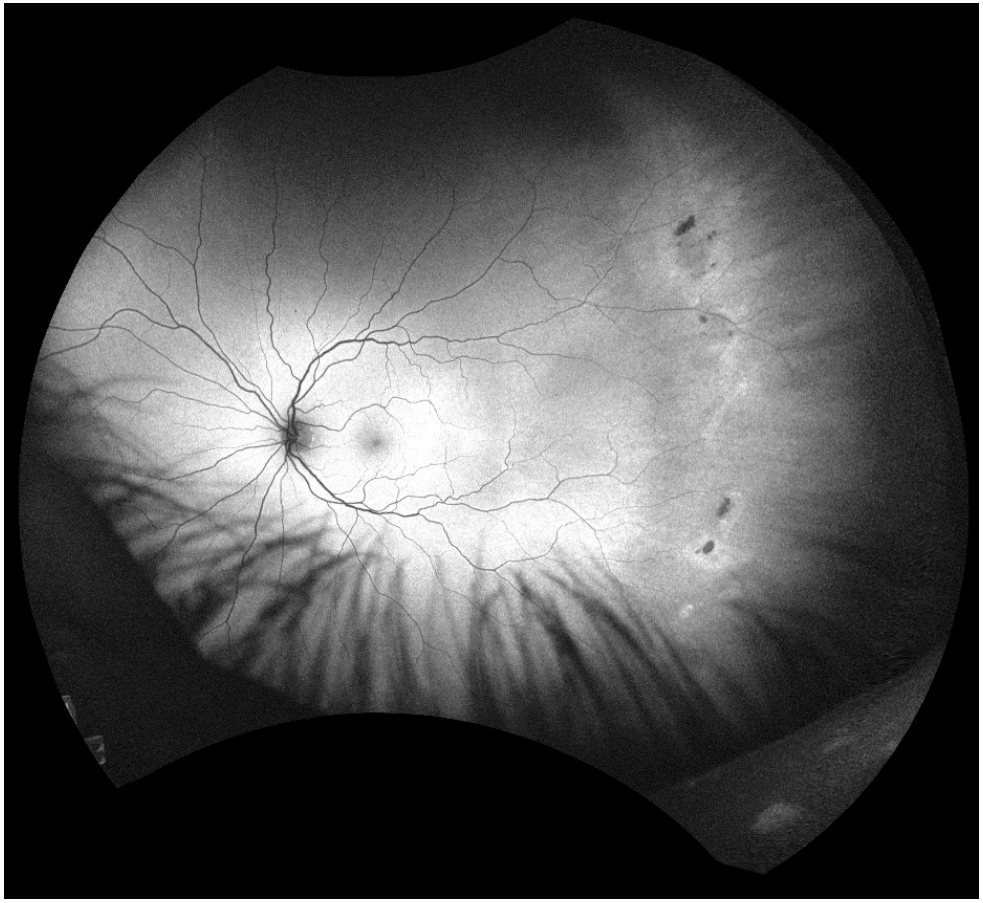
Mr. J.B. aet 63

attending clinic since aet 52 (2009), sister also has lattice and are both emmetropes areas of Hypoautofluoresence



Mr. J.B. aet 63 LE with atrophic holes within lattice and pigment hypertrophy surrounding





Long-term Natural History of Lattice Degeneration of the Retina

Norman E. Byer, MD AAOVOLUME 96, ISSUE 9, P1396-1402, SEPTEMBER 01, 1989

An initial series of patients with lattice degeneration was reported to the Academy in 1964 and a follow-up report given in 1973. A continuing prospective study of 276 consecutive untreated patients (423 eyes) is now reported with follow-up from 1 to 25 years (average, 10.8 years). Clinical retinal detachments (RDs) occurred in 3 (1.08%) of 276 patients and 0.7% of eyes. Tractional retinal tears were seen in eight (2.9%) patients and 1.9% of eyes; one of these led to a clinical RD. Clinical or progressive subclinical RD occurred in 3 (2%) of 150 eyes with atrophic holes. Subclinical RD was seen in 10 (6.7%) of 150 eyes with atrophic holes, involving 9 (7.5%) of 120 patients, and had a much less serious prognosis than clinical detachment. Prophylactic treatment of lattice with or without holes in phakic, nonfellow eyes should be discontinued.

Lattice and Detachment

- Lattice Degeneration (level of myopia not stated- 10 year average)
  - 0.7% of eyes developed retinal detachment
  - 1.9% of eyes develop retinal tears (some may progress to detachment
  - 2% clinical or progressive subclinical RD if had atrophic holes.
  - 6.7% of those with atrophic holes developed sub clinical RD and did well anyway.

  - The recommendation was, Prophylactic treatment of lattice with or without holes in phakic, non-fellow eyes should be **discontinued**.

Lattice and Retinal Detachment and Myopia

- The incidence of retinal detachment is higher in myopic eyes than in emmetropic eyes (3.2% [myopia greater than -6.00 D] vs 0.71%, respectively).[1,2]
- The yearly incidence of retinal detachment is approximately 0.015% in eyes with up to -4.75 D of myopia, 0.07% in eyes with -5.00 to -9.75 D, and 0.075% in eyes with myopia greater than -10.00 D.[3,4]

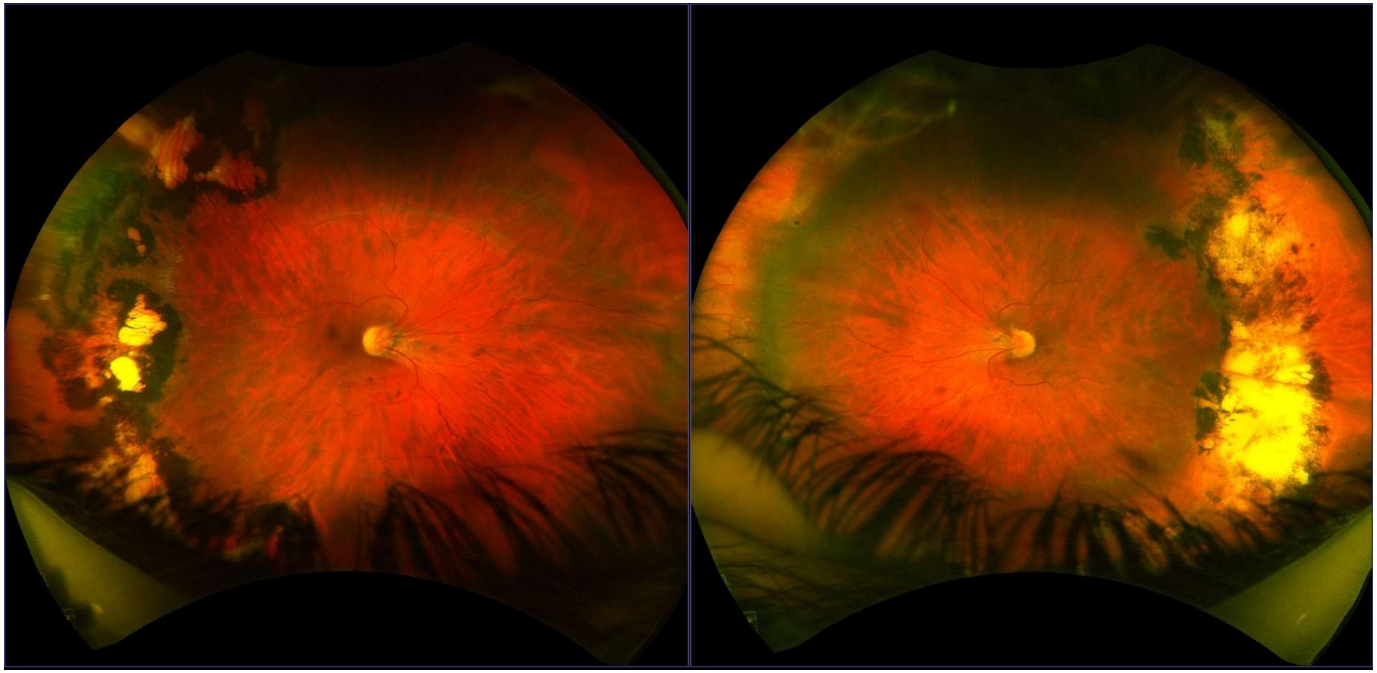
Lattice and Detachment and Myopia

- Lattice Degeneration (level of myopia not stated- **yearly incidence**)
  - 0.07% of eyes developed retinal detachment
  - 0.19% of eyes develop retinal tears (some may progress to detachment)
  - 0.2% clinical or progressive subclinical RD if had atrophic holes.
  - 0.67% of those with atrophic holes developed Sub Clinical RD and did well anyway.

- Myopia and **yearly incidence of retinal detachment**)
  - 0.015% in eyes with up to -4.75 D of myopia
  - 0.07% in eyes with -5.00 to -9.75 D
  - 0.075% in eyes with myopia greater than -10.00 D.[3,4]

What can I do to stop LD going to RD?

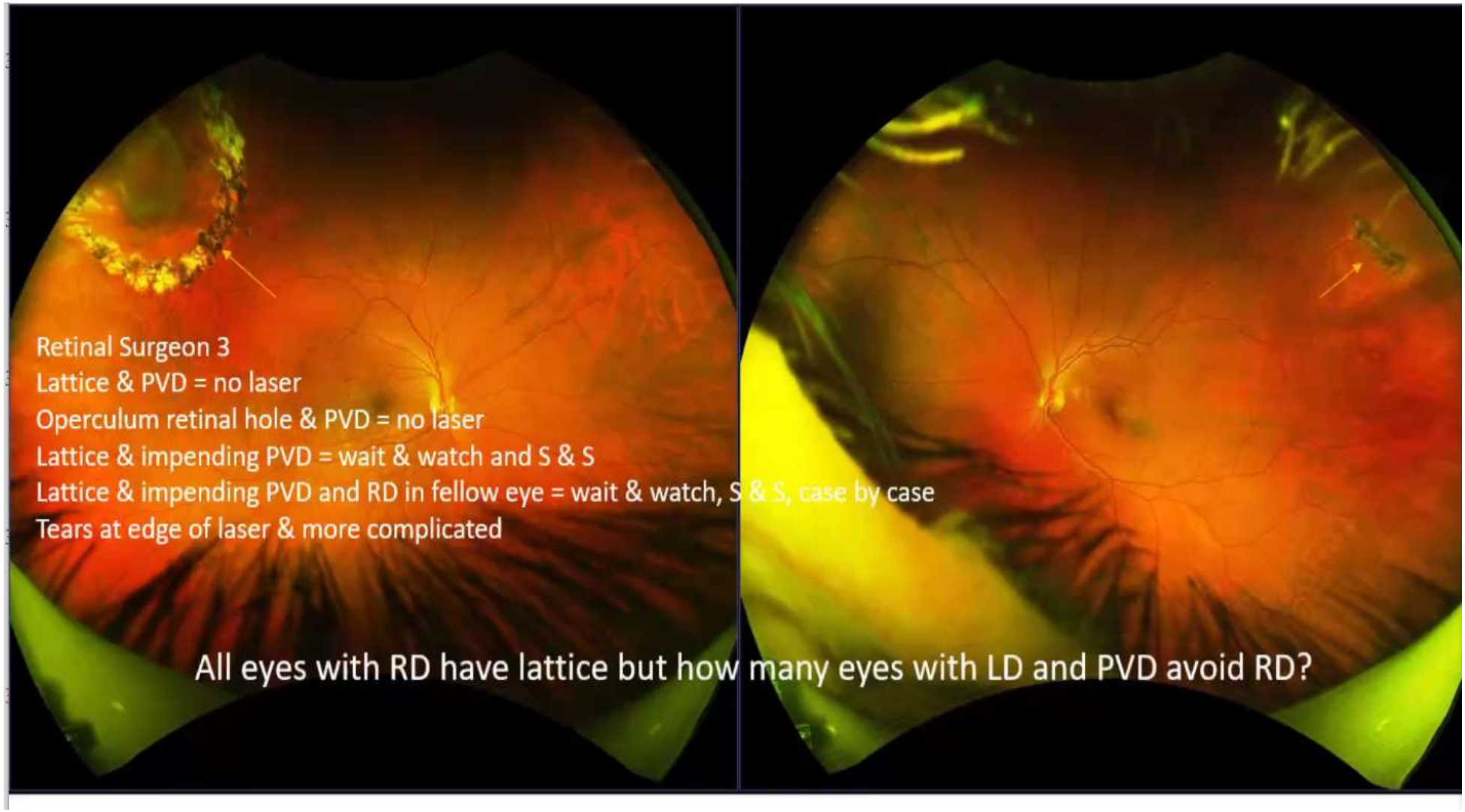
Olden days
Pt with ROP
-10.50/-1.00 x 32.2.5 -12.00/-0.75 x 45
RD LE and prophylactic cryopexy RE



Surgeon 1
No laser unless LD posterior to vitreous base and elevated or horse shoe tear
Widespread prophylactic laser doesn't prevent rRD & can produce more complex ones?

Surgeon 2
Leave lattice alone unless other eye has a rRD, then still no consensus

Warned S & S with PVD & return ASAP (within 48 hrs)
Any other ideas refer?
What if the patient is a great traveller?

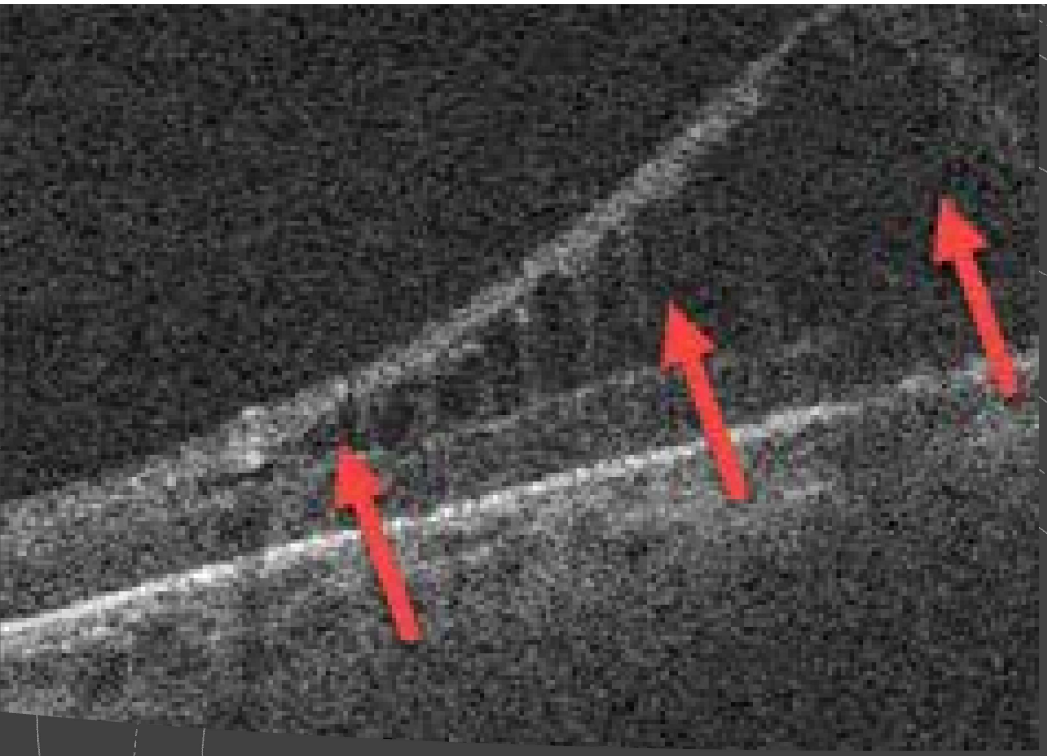

Retinal Surgeon 3
Lattice & PVD = no laser
Operculum retinal hole & PVD = no laser
Lattice & impending PVD = wait & watch and S & S
Lattice & impending PVD and RD in fellow eye = wait & watch, S & S, case by case
Tears at edge of laser & more complicated
All eyes with RD have lattice but how many eyes with LD and PVD avoid RD?

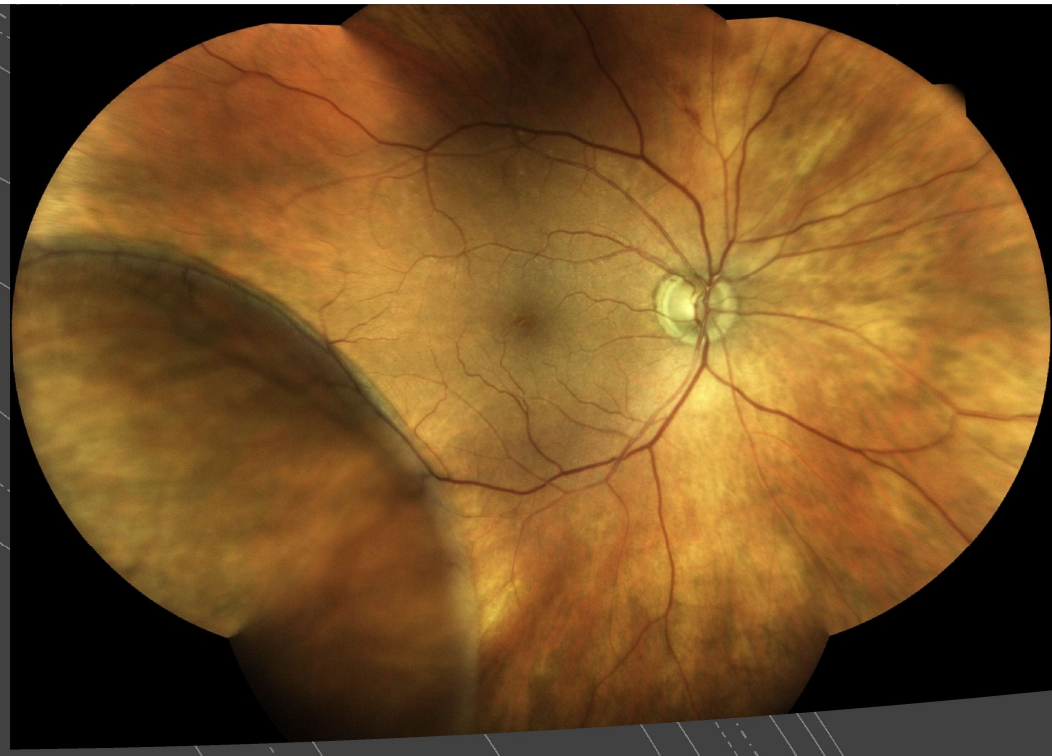
Retinal Surgeon 3 – Lattice and Intervention

- Operculated retinal hole and PVD already occurred = No laser
- Lattice and PVD = No Intervention and no Laser
- Lattice and Start of PVD = Wait and watch closely (+ Symptoms)
- Tears at edge laser more complicated
- Lattice and start of PVD and RD in other eye = watch but bit more case by case

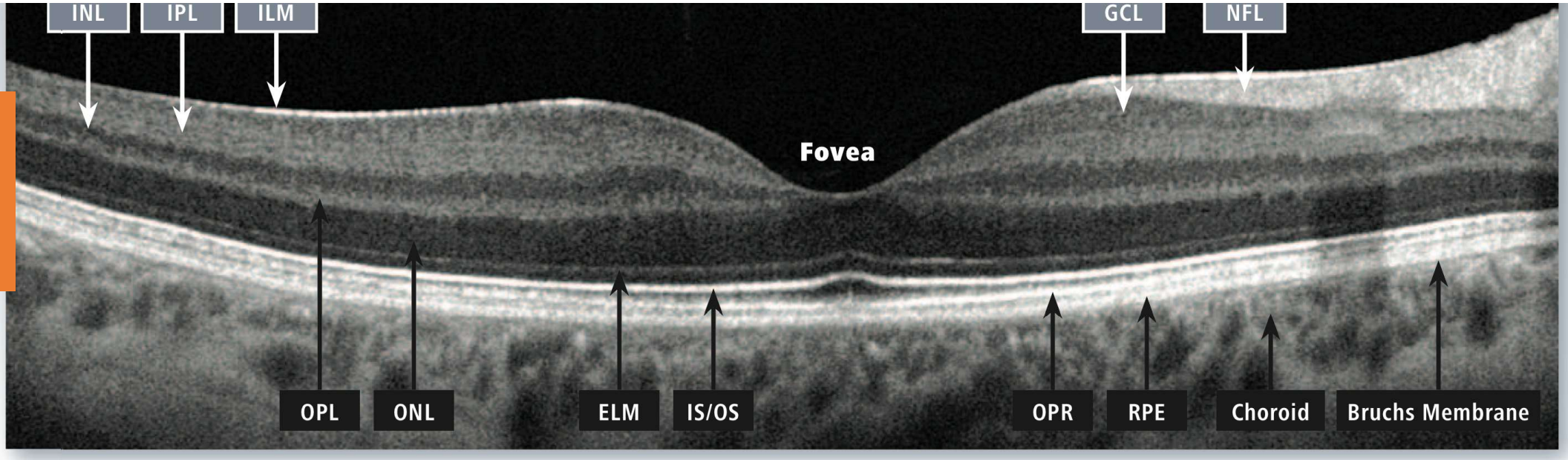
MCQ Which of these statements about lattice degeneration is false

- 1/ All lattice degenerations should be treated with retinal laser to prevent progression to rRD
- 2/ Approx 60% of eyes with rRD have lattice degeneration
- 3/ Lattice degeneration is more common amongst high myopes
- 4/ Lattice degeneration has stronger adhesion to the vitreal face and are more prone to retinal tears with PVD





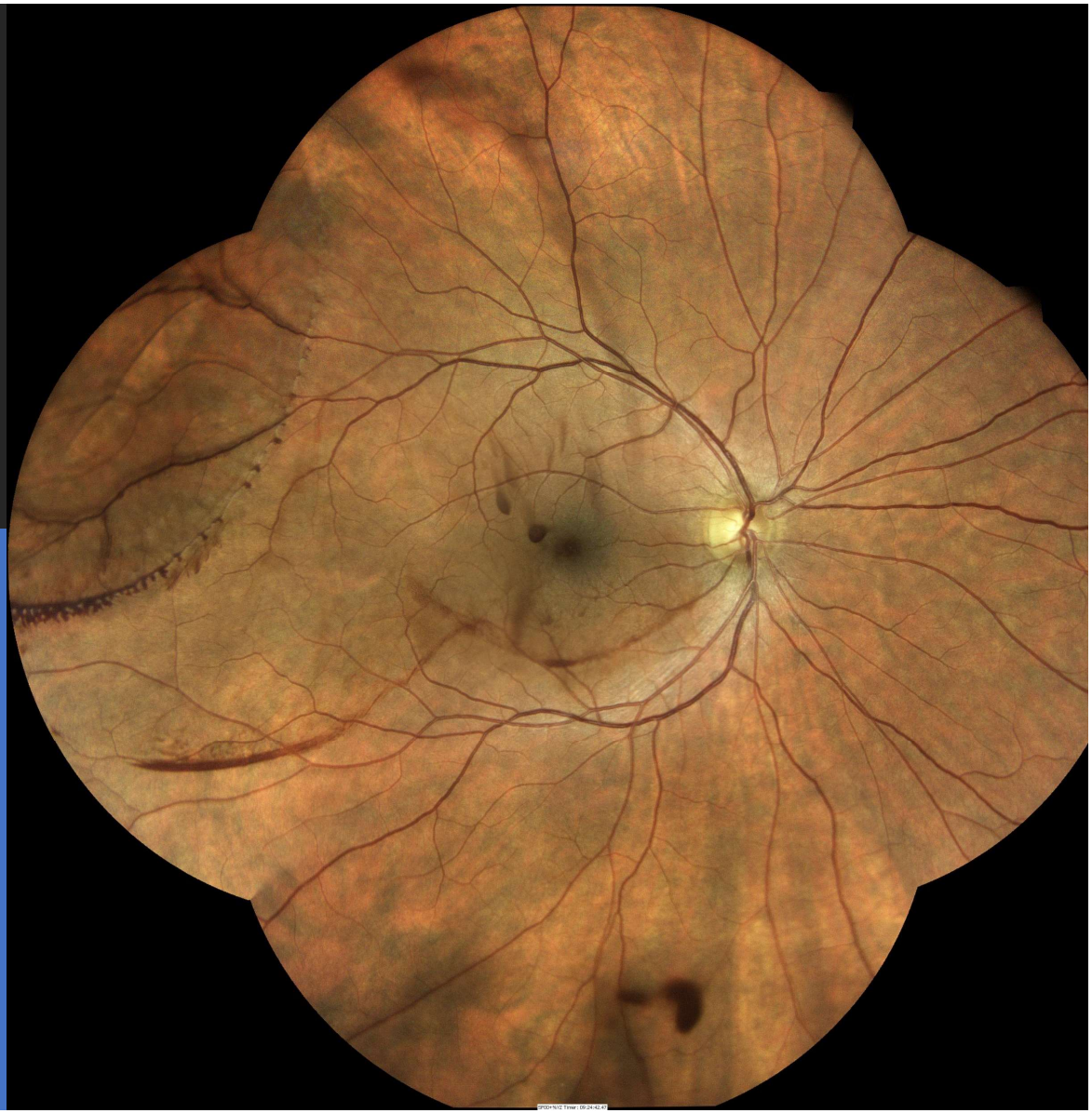
Retinoschisis behaving badly



Retinoschisis

Juvenile X linked rare poor vision RD?
acquired = degenerative, idiopathic splitting

- Splitting sensory retina at OPL
- Outer (choroidal layer) inner (vitreous layer)
- +ve scotoma
- 7% population > 40
- Hyperopes
- Generally outerplexiform
- Snowflakes on inner surface Beaten metal on outer surface
- ‘Wobbly’

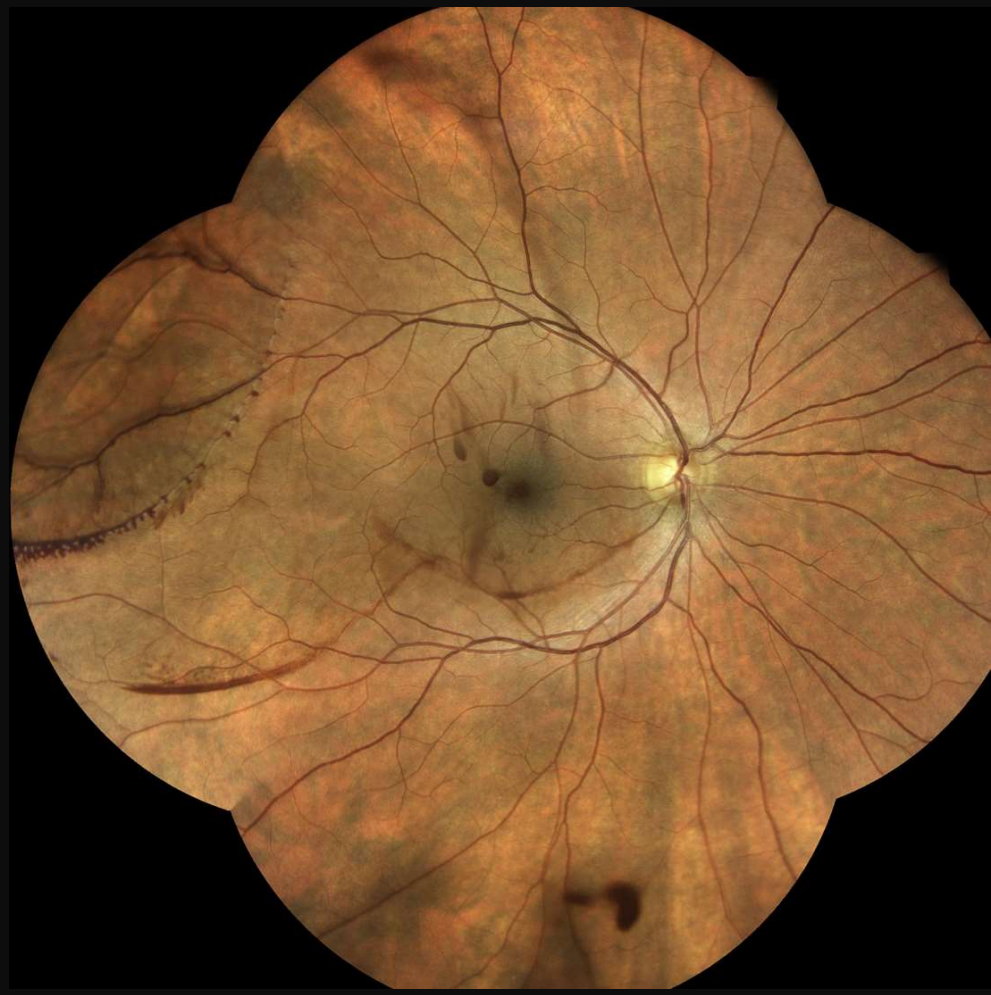


Mr. S.P. aet 46

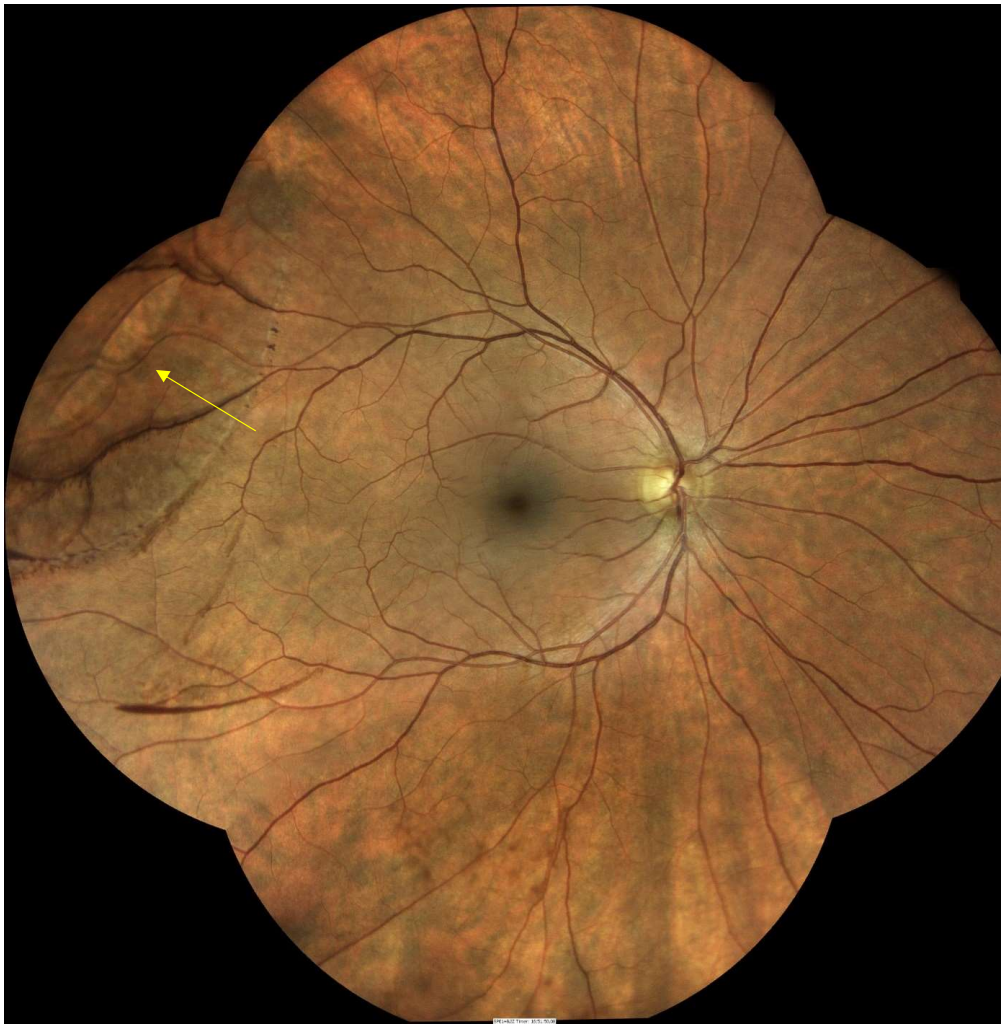
2-6-18
Black arc & 2 spots on awakening RE
no flashes after surfing
drinking heavily night before
Vis 6/6 OU
schafer's sign?
ST elevated lesion
horizontal tear
vitreous haem
VR surgeon
fast, limit head movement, off to Melbourne ASAP

VR ophthal report

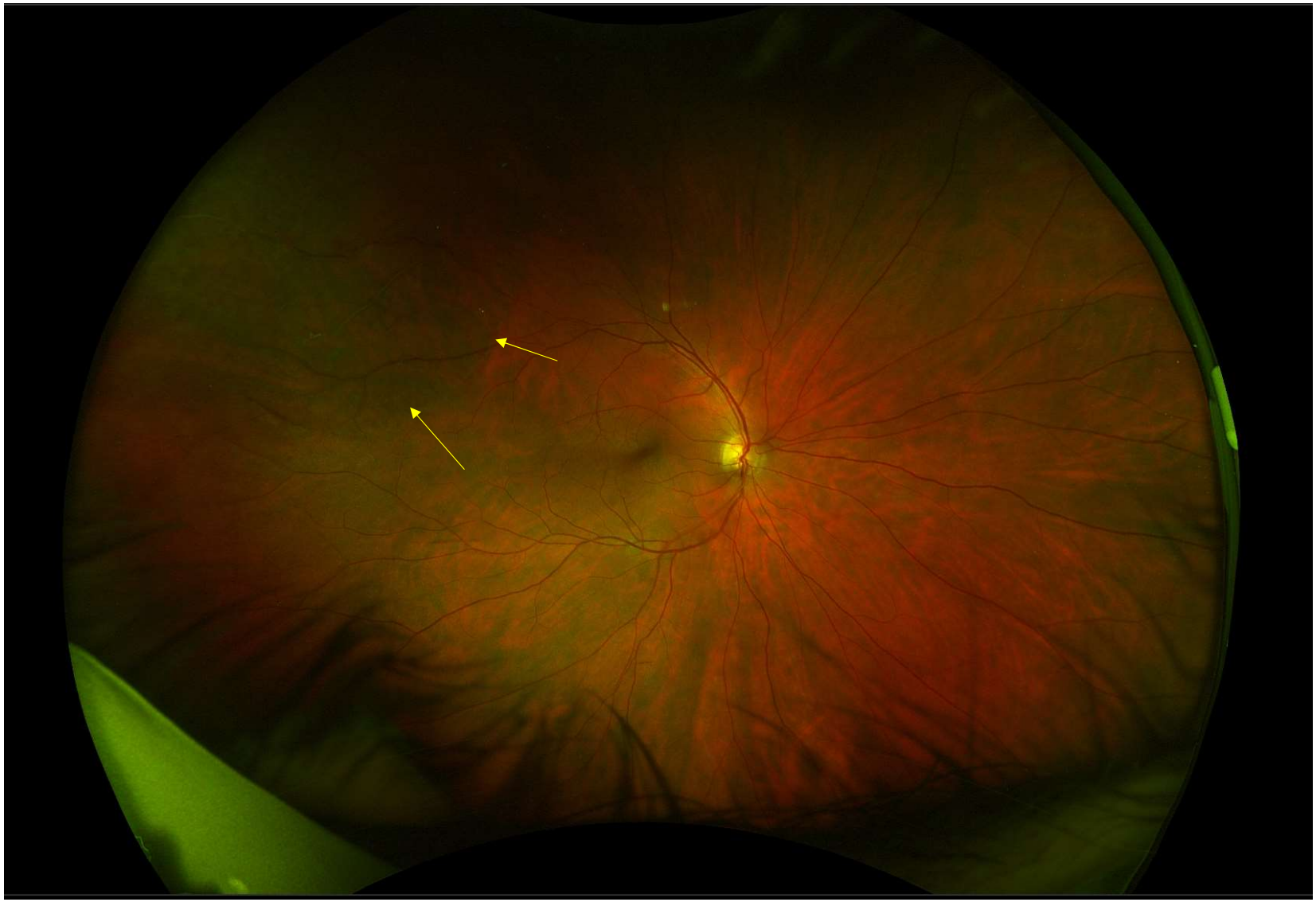
- Vitreous haem without PVD
- Vit haem obscuring macula
- ST chronic RS confirmed on OCT
- Inner retinal hole
- Blood along lower border of schisis cavity
- **Discussion**
- Spontaneous Vitreous haem = Valsalva
- Chance of schisis cavity extending is low
- Observe vit haem should resolve on own
- discharged



Mr. S.P.29-6-18
4 /52 later
Vitreous haem cleared
chronic inner leaf tear
no sx required
observe & RD symptoms & signs



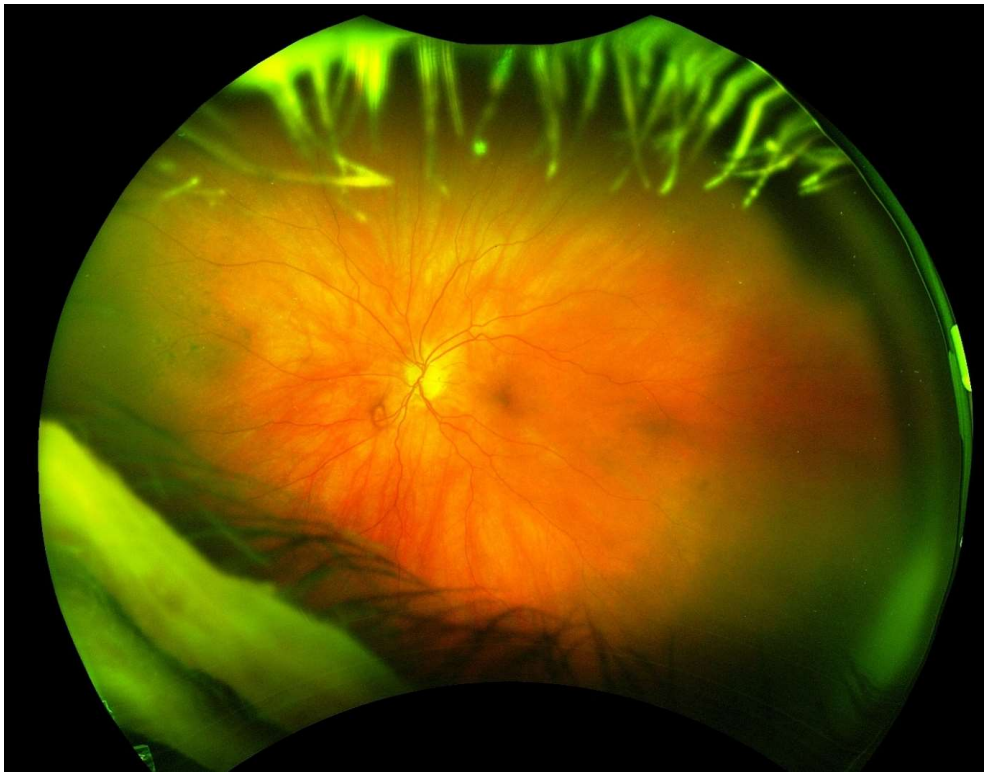
Mr. S.P.
25-2-20
20/12 later

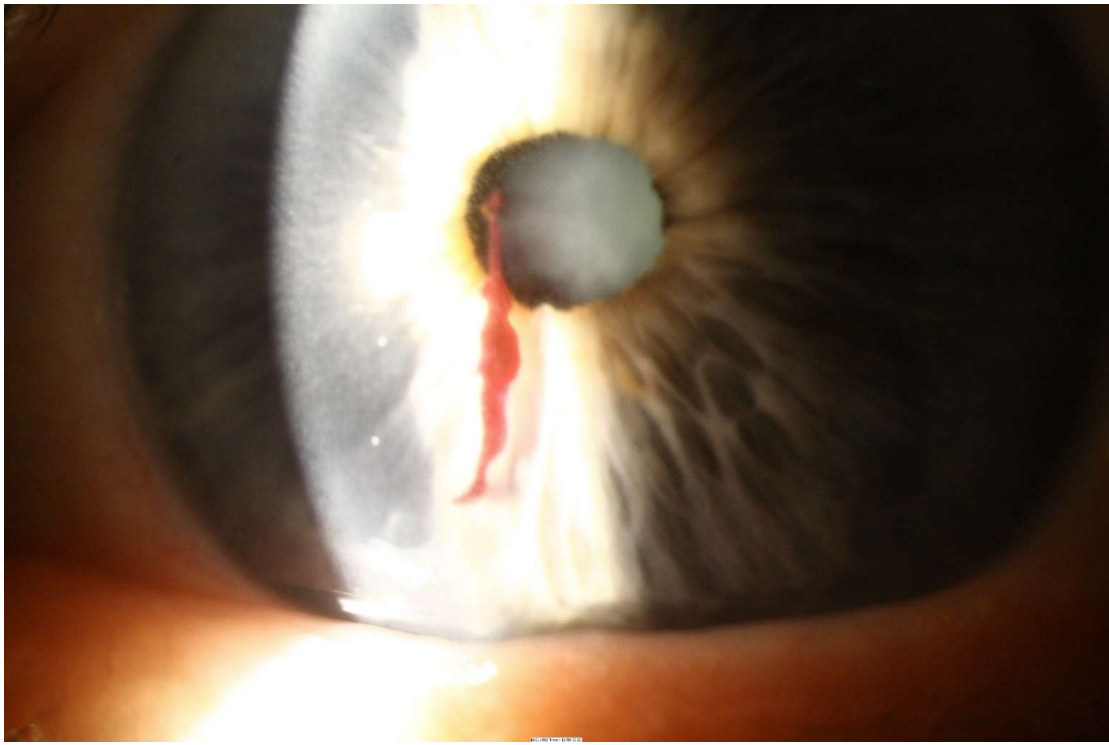


A little diversion

Mrs. B.K. aet 81 NIDDM 13 years , blurred L lower vision amsler NAD 6/7.5+
ACLO changes 3+ hyphaema no flare PERRLA PIOP R 16 L 26 mmHg
action : Hba1c <7% refer to ophthal

Alternate case – 55 years old female with new acute anterior uveitis





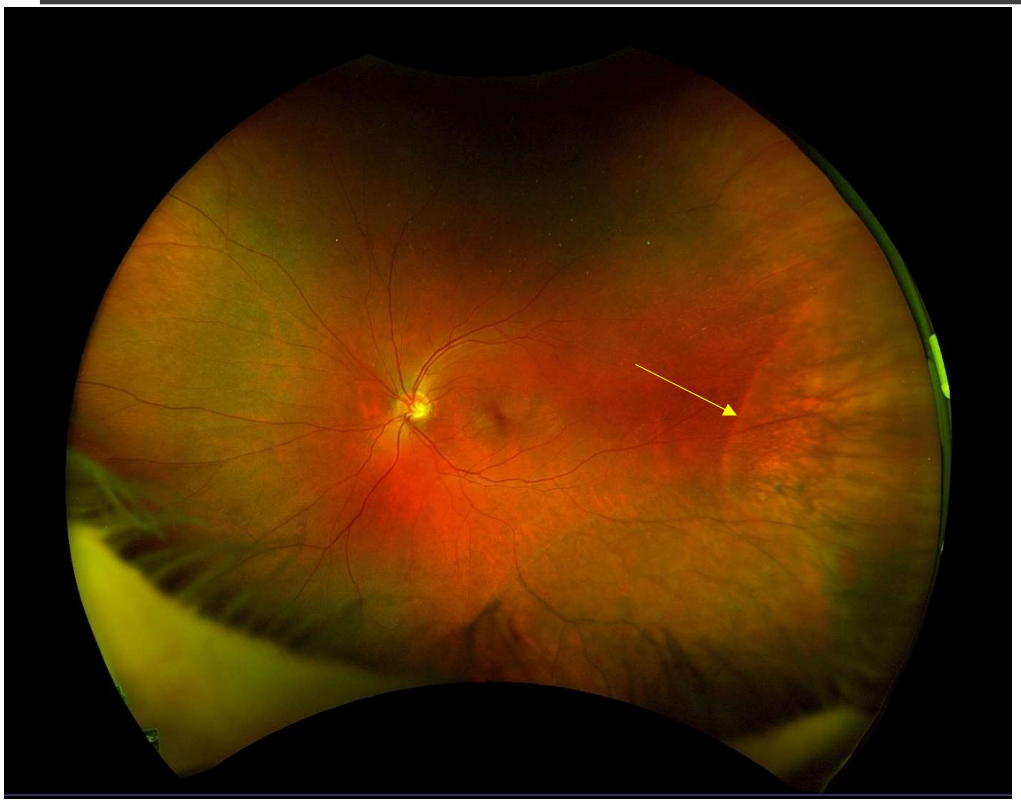
Mrs. B.K.

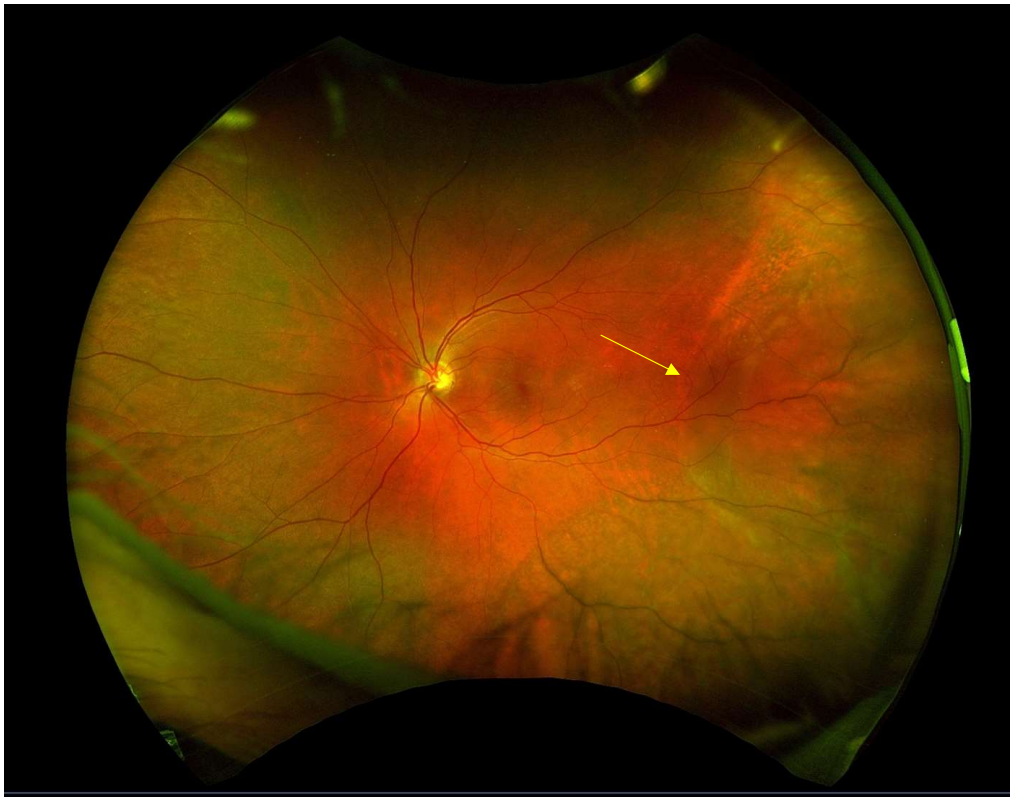
- Ophthal opinion 2 days later
- Completely resolved (sent image) VA 6/6 IOP 16 mmHg
- Differential Dx : Trauma, Valsalva, Rubeosis, Iris tumour,
- No PDR, no iris vasculature anomaly, no IN
- Action : not active, Trauma through sleep?

Ms G.H. aet 69

26-8-18 2 years later 13-08-20

RS has moved more posteriorly





Mrs. G.H.
asymptomatic

No obvious PVD either eye & has retinoschisis infero temporally OU…..There appears to be a full thickness elevation more temporally left eye confirmed with OCT & I suspect an area of outer leaf defect. An Inner leaf break was not obvious & these are difficult to detect……She appears to be mounting some RPE change at the border & this maybe sufficient to stabilise the situation & if there is progression she may require barrier retinopexy & she is aware that surgical intervention maybe required.

Complications of retinoschisis

- Posterior extension of schisis
- Inner leaf breaks = small, round like atrophic holes (Mr. S.P.)
- Outer leaf breaks = schisis detachment = schisis fluid accumulates in the subretinal space may not progress to rRD due to higher viscosity intraschisis fluid
- Rhegmatogenous retinal detachment where breaks in both inner then outer leaves allow liquefied vitreous into subretinal space up to 6% of eyes
  Barricade laser?
- Natural history stops 3 disc diameters from the macula

REVIEW of OPTOMETRY

Diagnosis is on the Line

Pigmented and nonpigmented demarcation lines in both eyes hold the clues to this patient's condition

Mark T. Dunbar, O.D.

To distinguish between a RD and a retinoschisis

peripheral retinoschisis more commonly appears inferotemporally, followed by superotemporally.

Retinoschisis is very clear and transparent, allowing for the choroidal detail below the schisis to be easily seen
Chronic retinal detachments, a pigmented demarcation line can be seen at the junction between attached and detached retina.

Retinoschisis is not associated with changes in RPE unless an outer tear change

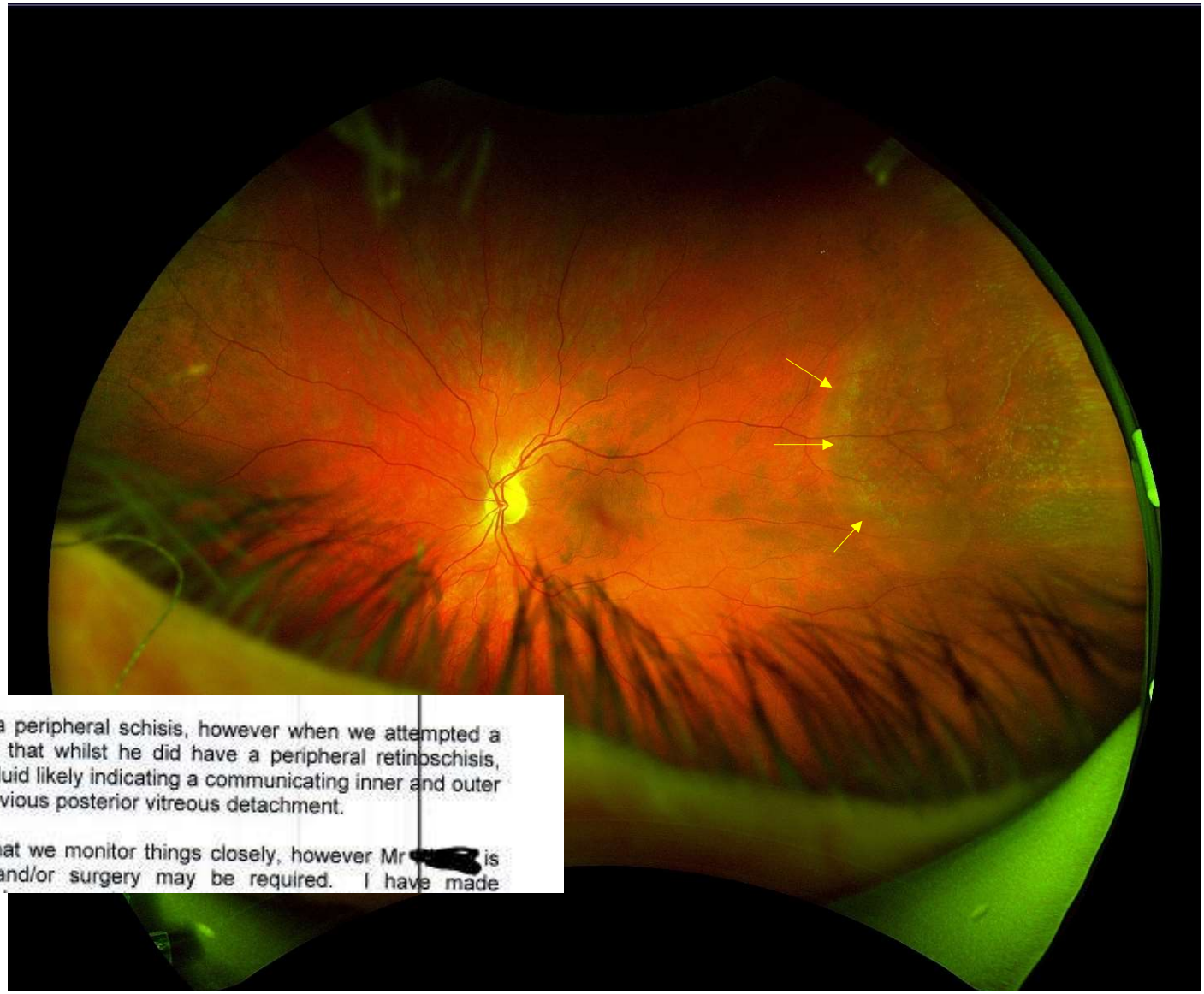
6% of retinoschisis has associated retinal detachment = outer wall break to allow fluid into SRS

Outer wall holes may be single or multiple, and they may be small or large. They appear as retinal holes with smooth, rounded margins and rolled edges. These rolled edges are the hallmark of outer wall holes. Inner holes are rare and harder to diagnose due to the variations in the thickness of the inner retinal layer.

Bilateral…… Tx? Barrier laser? Vitrectomy & scleral buckling?

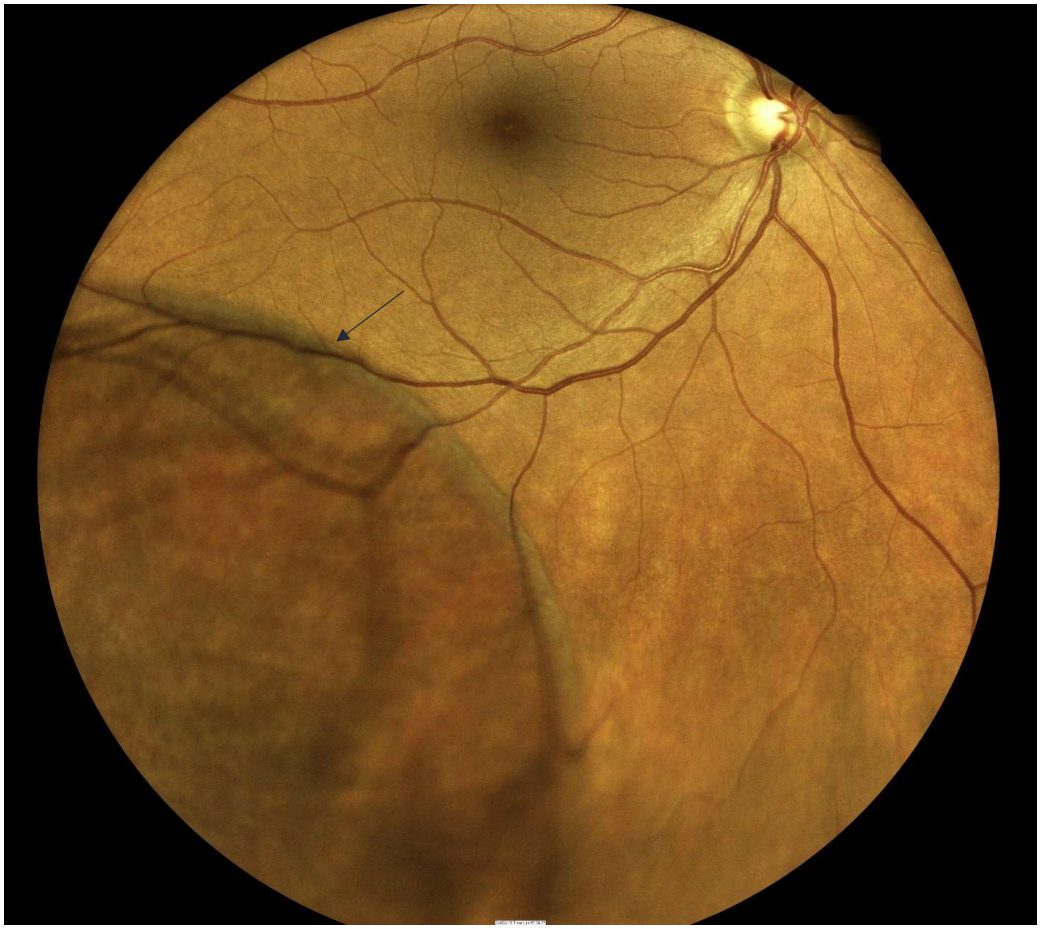
Mr. K.R. aet 65

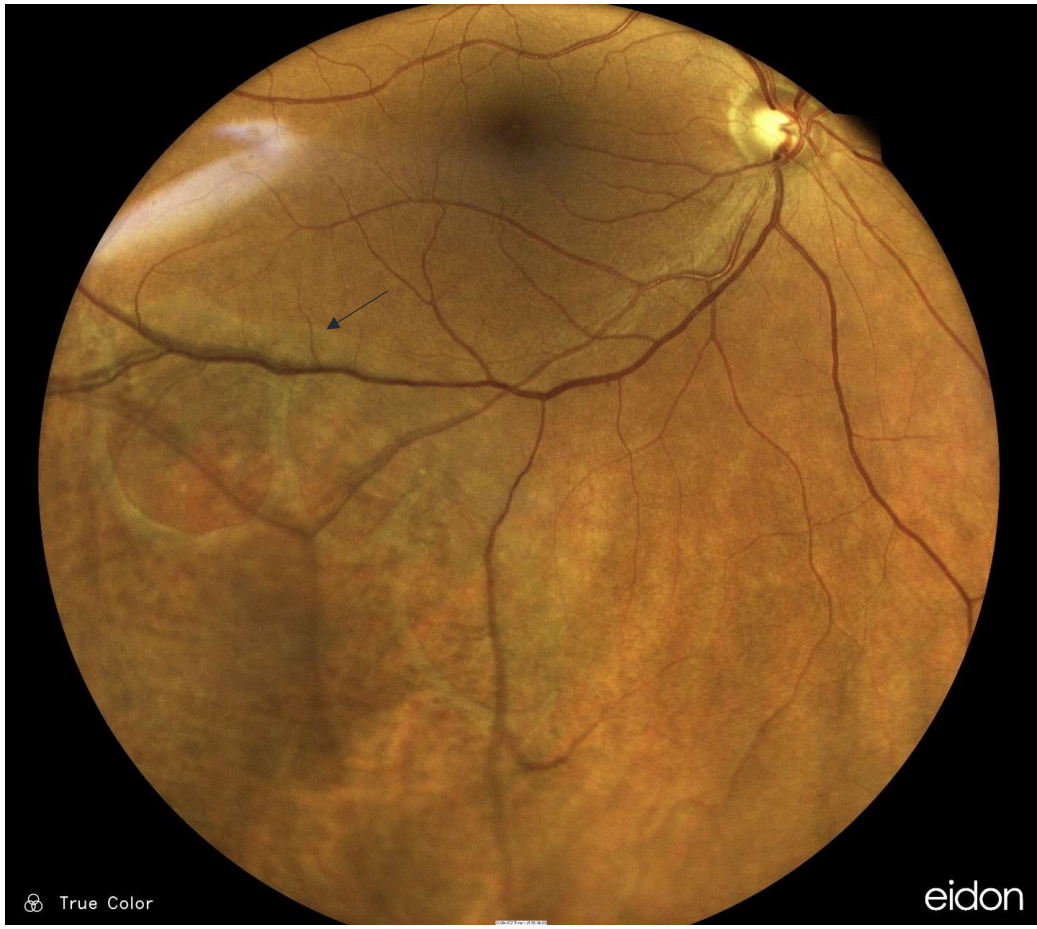
review new readers
Hyperopia R 6/7.5 L 6/6
asymptomatic



On examination I felt clinically he had a peripheral schisis, however when we attempted a confirmatory OCT scan, it was evident that whilst he did have a peripheral retinoschisis, there was a leading edge of sub-retinal fluid likely indicating a communicating inner and outer leaf break situation. I could detect no obvious posterior vitreous detachment.

I have suggested in the first instance that we monitor things closely, however Mr [redacted] is aware that some barrier retinopexy and/or surgery may be required. I have made



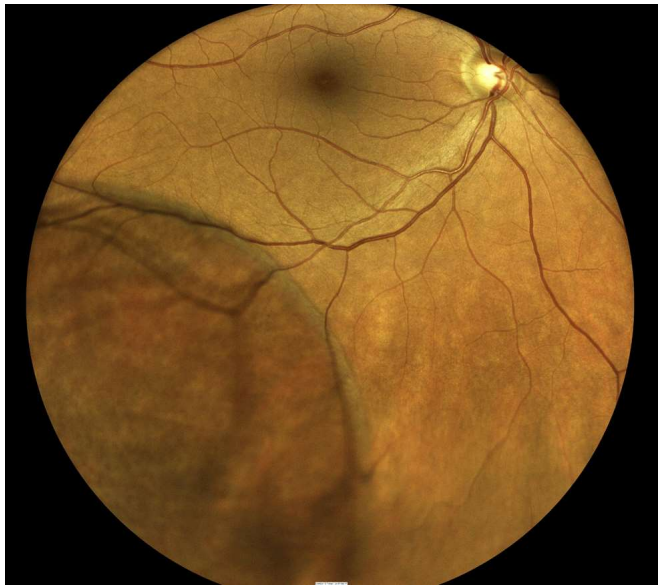


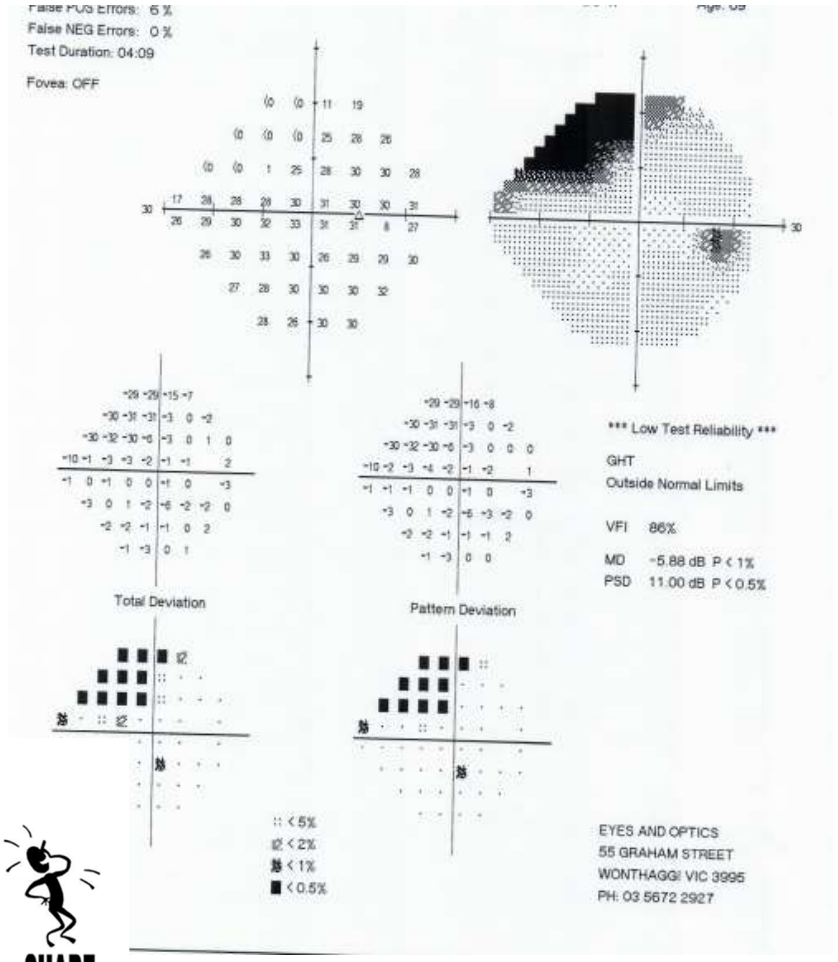
These images are 3 years apart 30-1-17 & 9-6-20
69 yo what's her Rx? What can you see?

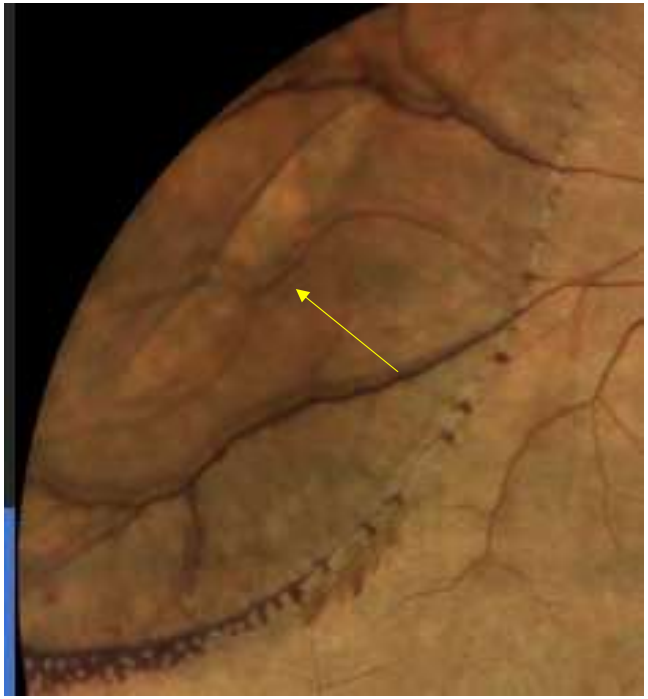
Round atrophic holes = inner leaf break?

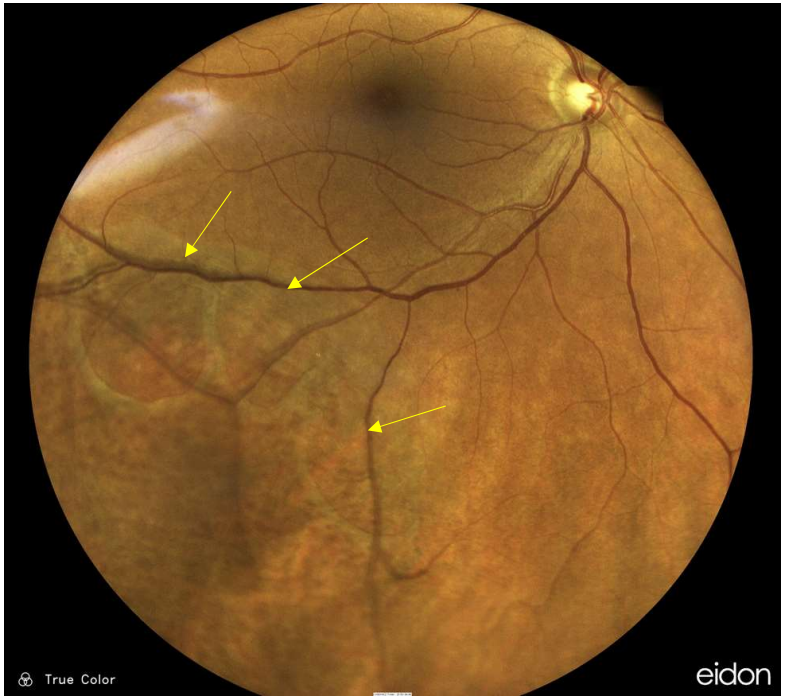
Where’s the scotoma?

- A infero temporal
- B supero temporal
- C Infero nasal
- D supero nasal ✓

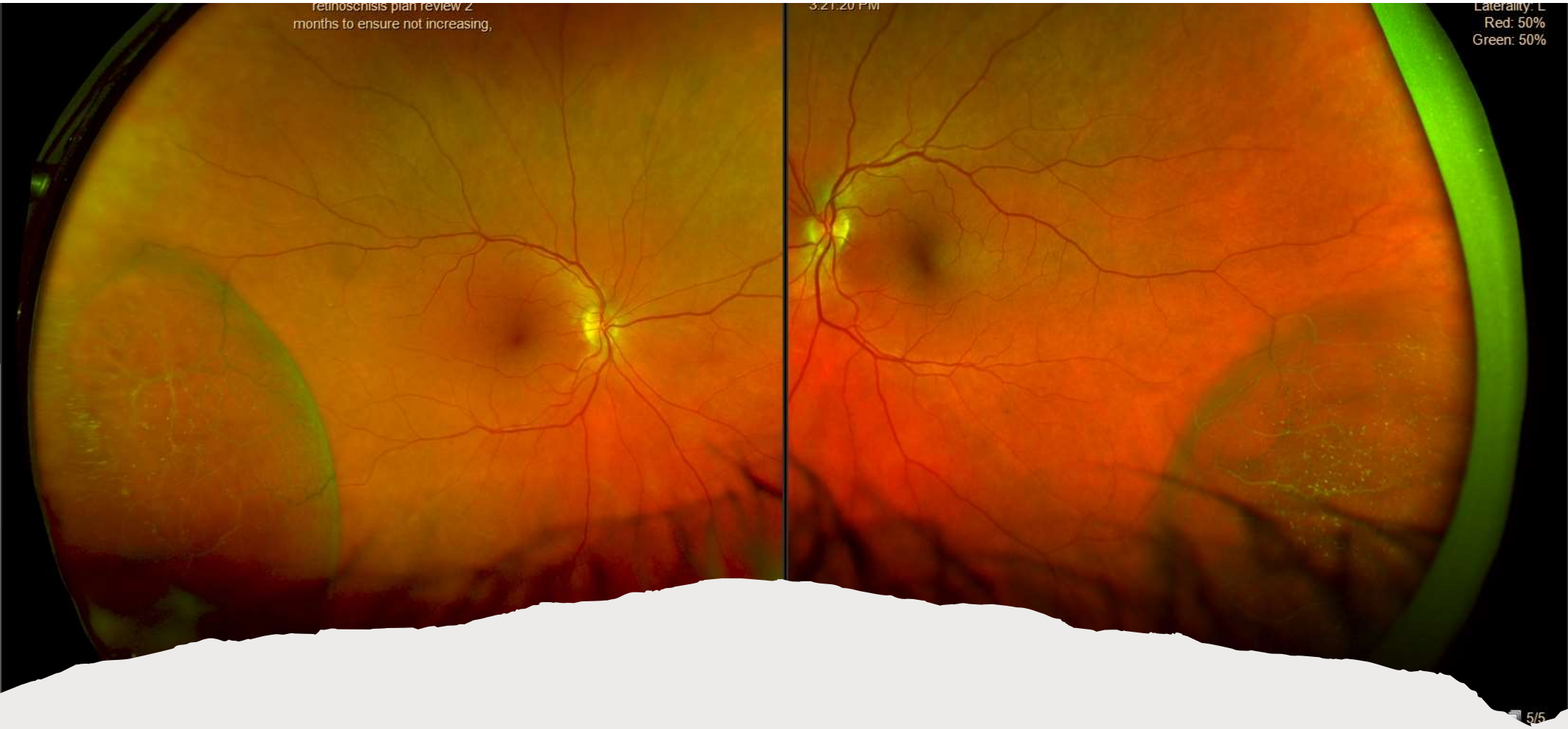




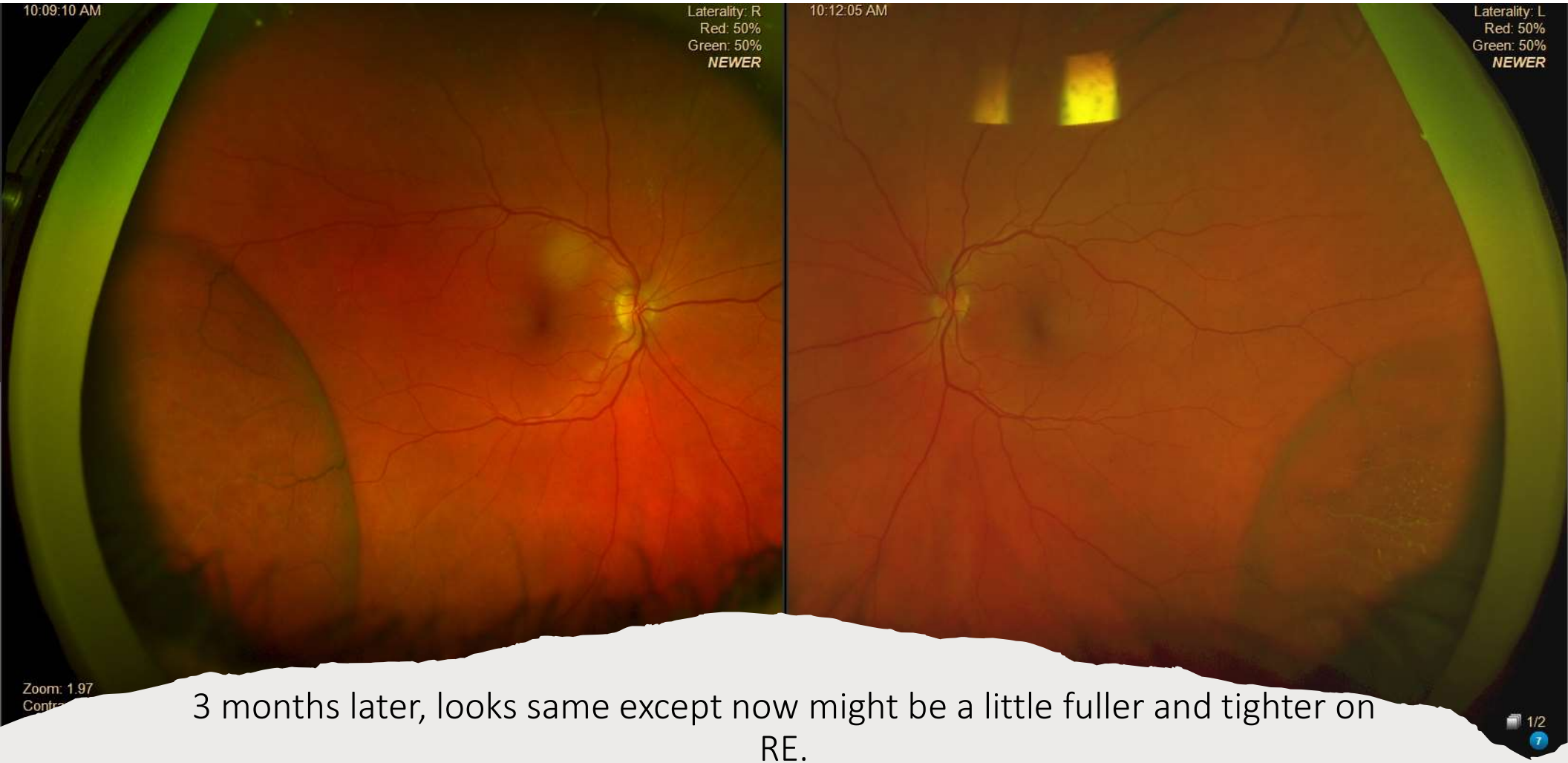




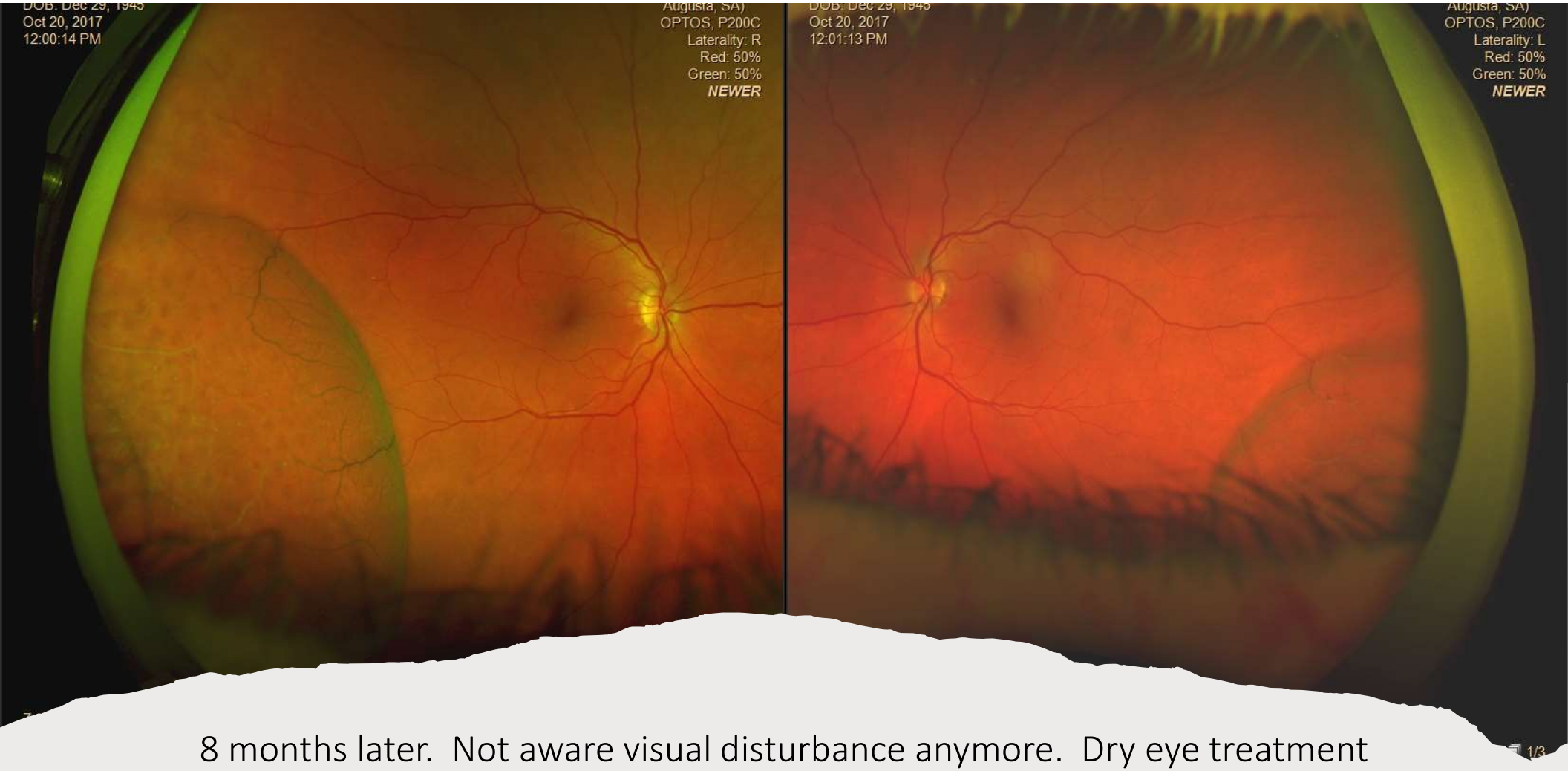
Inner leaf breaks? intraschisis fluid flows into the vitreous and schisis flattens



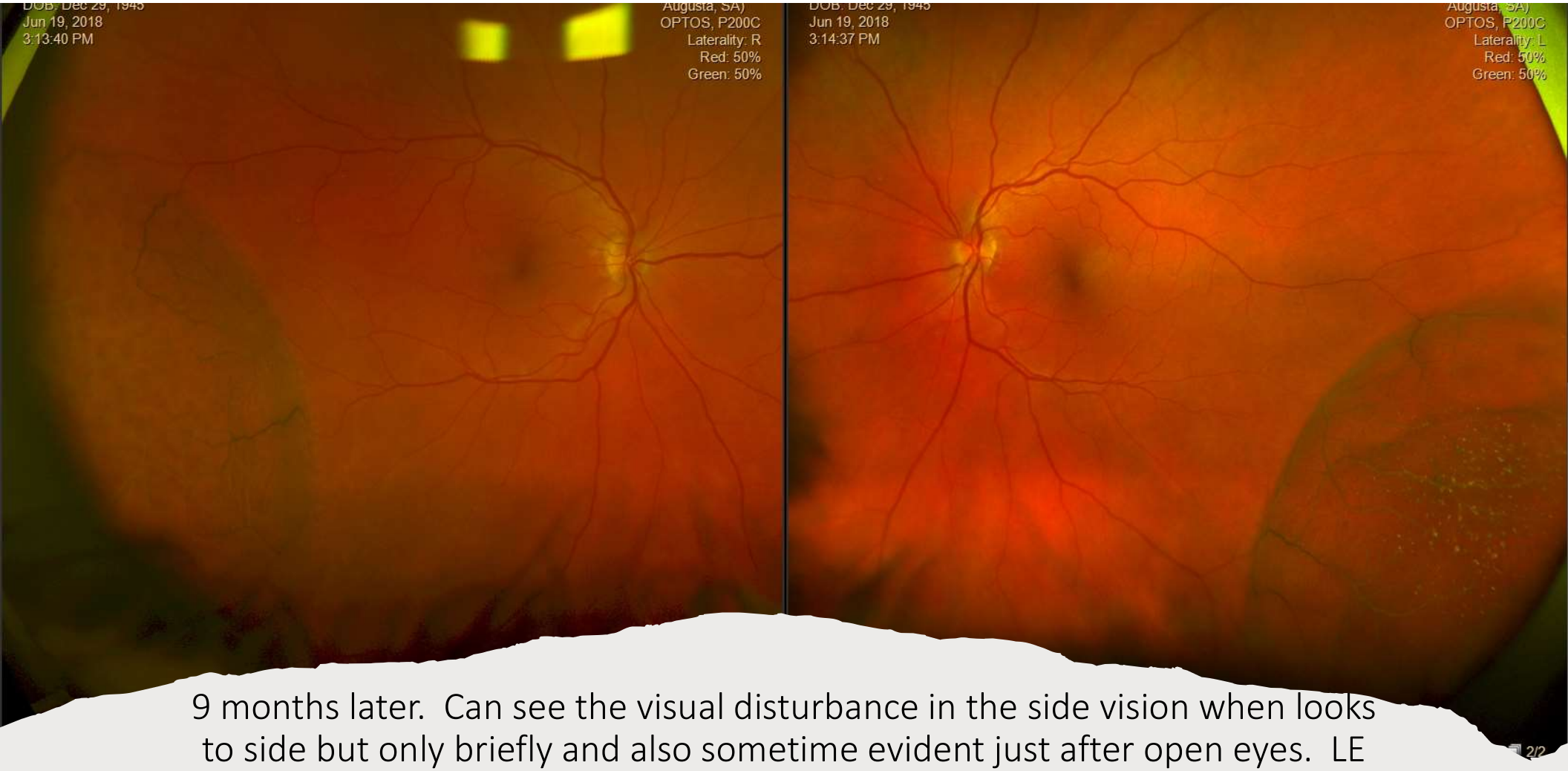
Mrs K, age 68, mild moderate hyperopia. Large retinoschisis bilateral. No symptoms and 2 years prior peripheral exam was clear. “How likely to become an issue?”



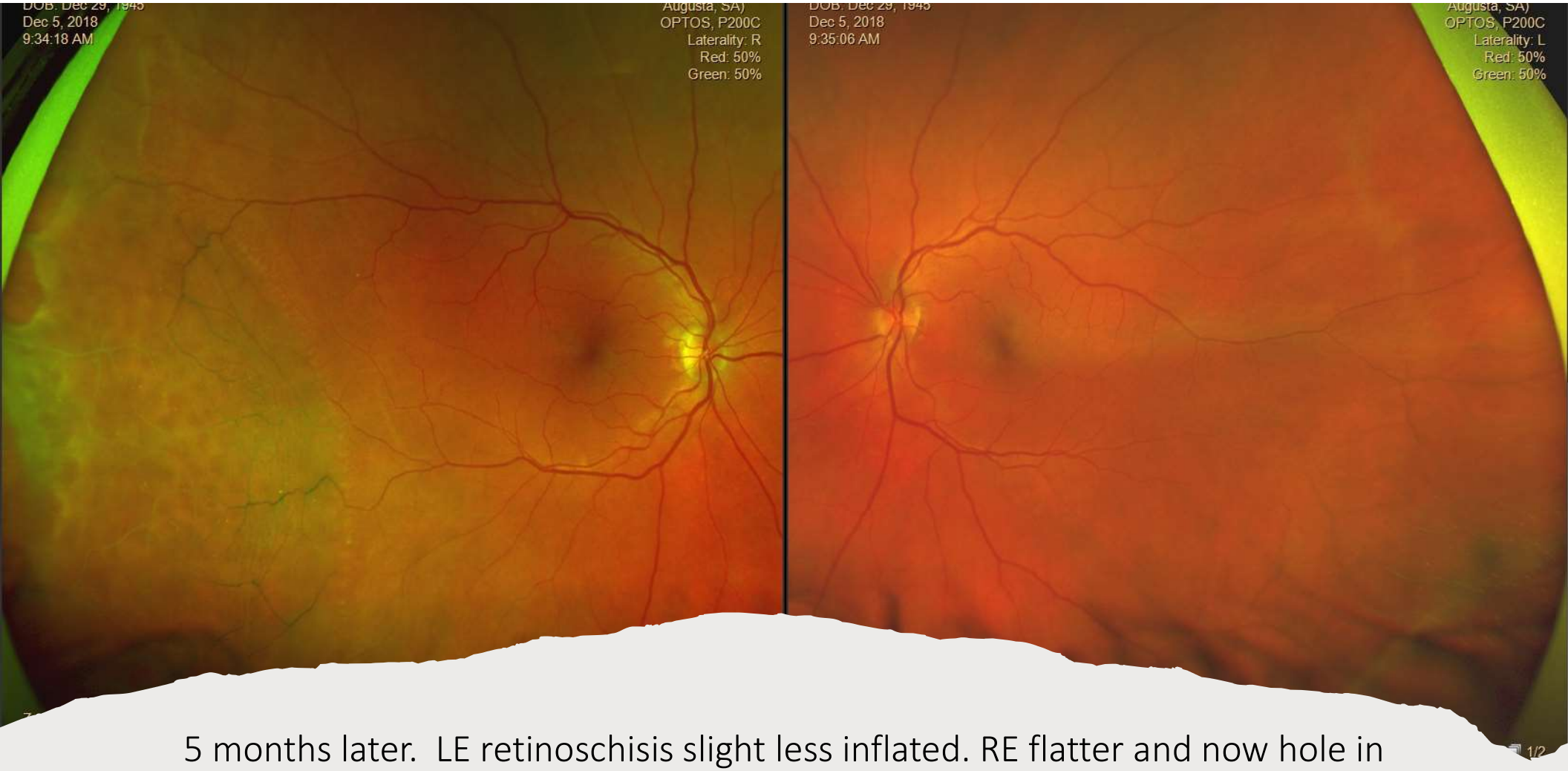
3 months later, looks same except now might be a little fuller and tighter on RE.



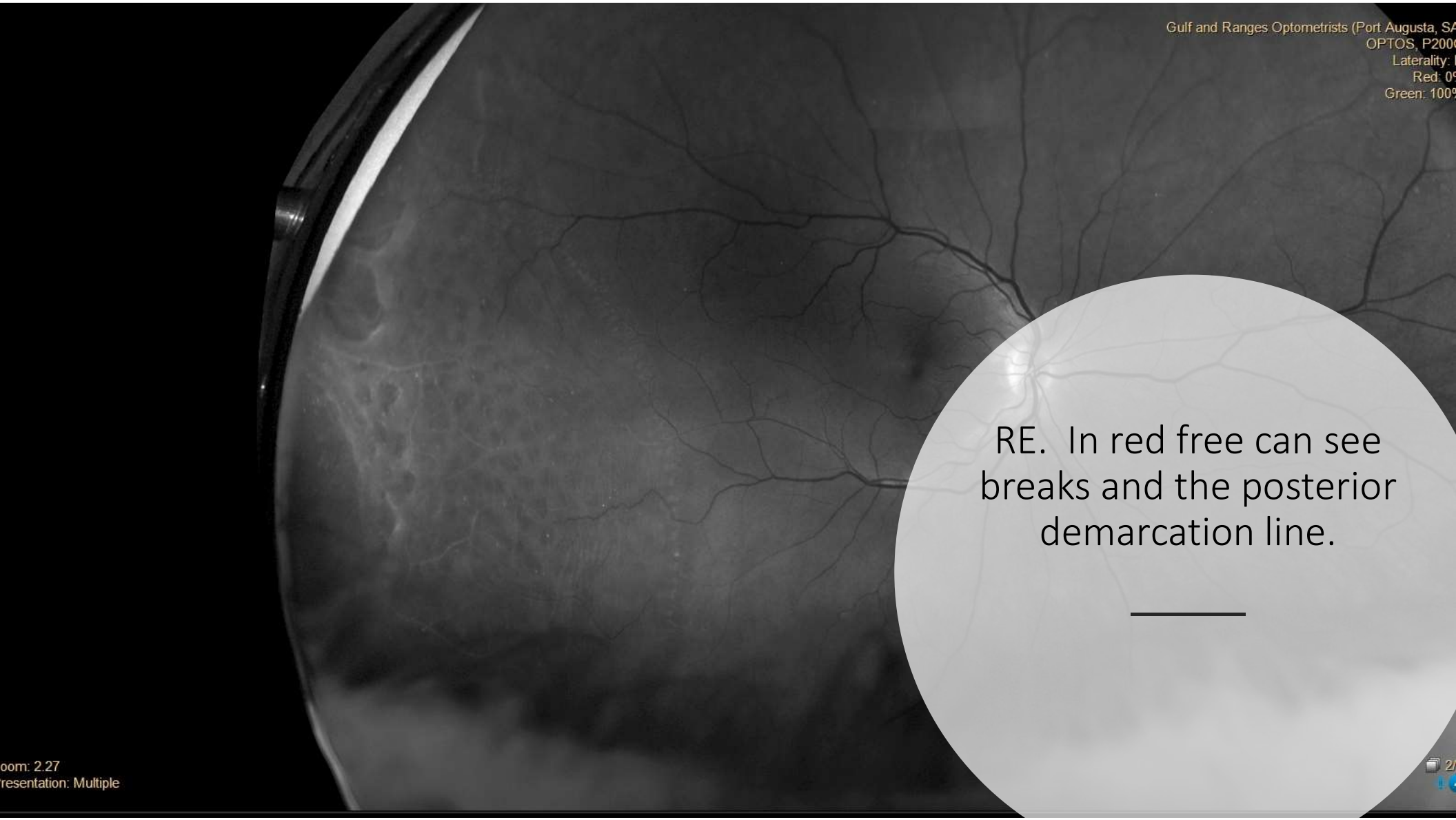
8 months later. Not aware visual disturbance anymore. Dry eye treatment completed. No signs tears or holes. RE slightly larger and is extending posteriorly



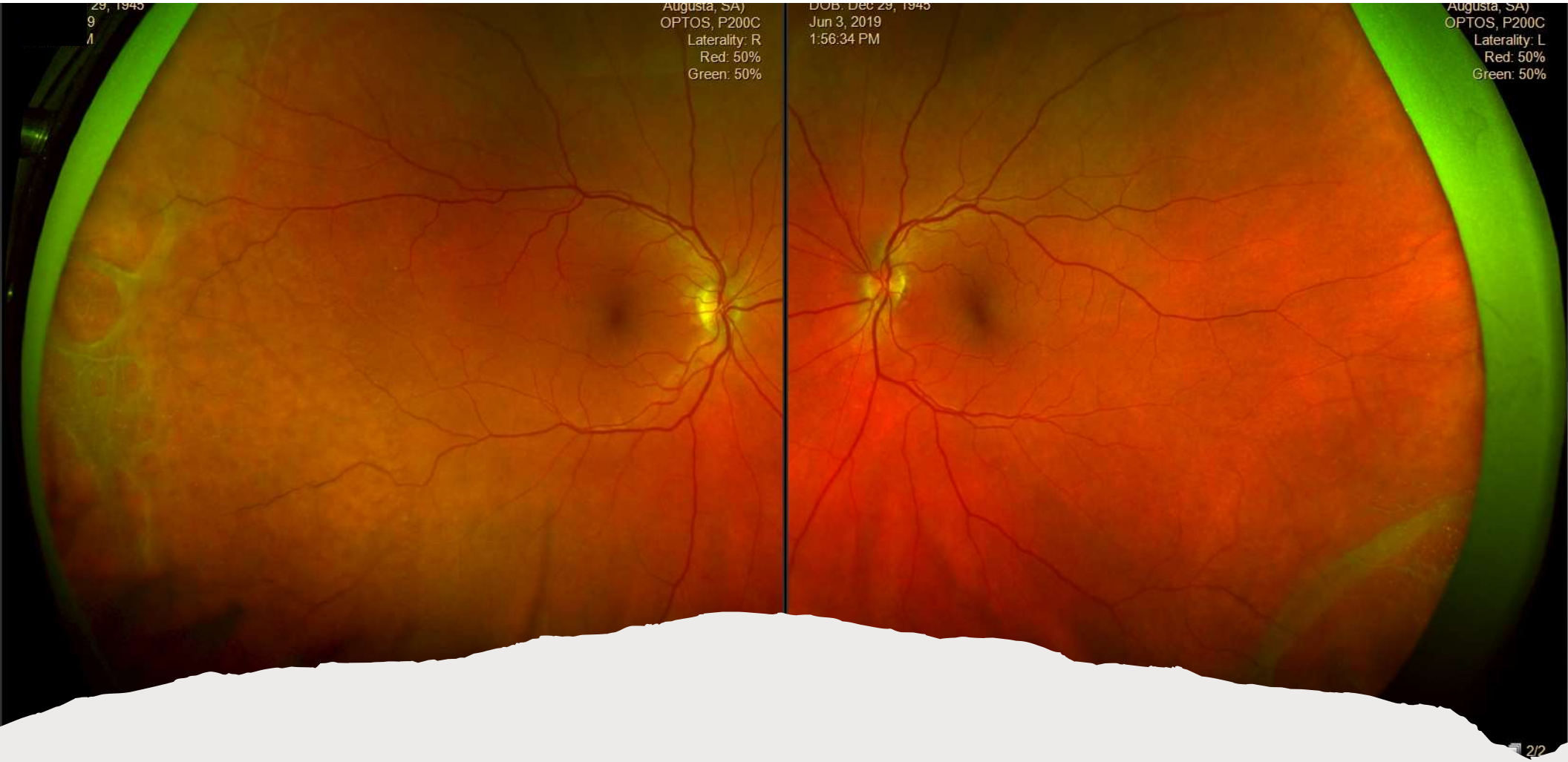
9 months later. Can see the visual disturbance in the side vision when looks to side but only briefly and also sometime evident just after open eyes. LE retinoschisis slight less inflated. No holes evident. RE tighter, larger and is extending posteriorly



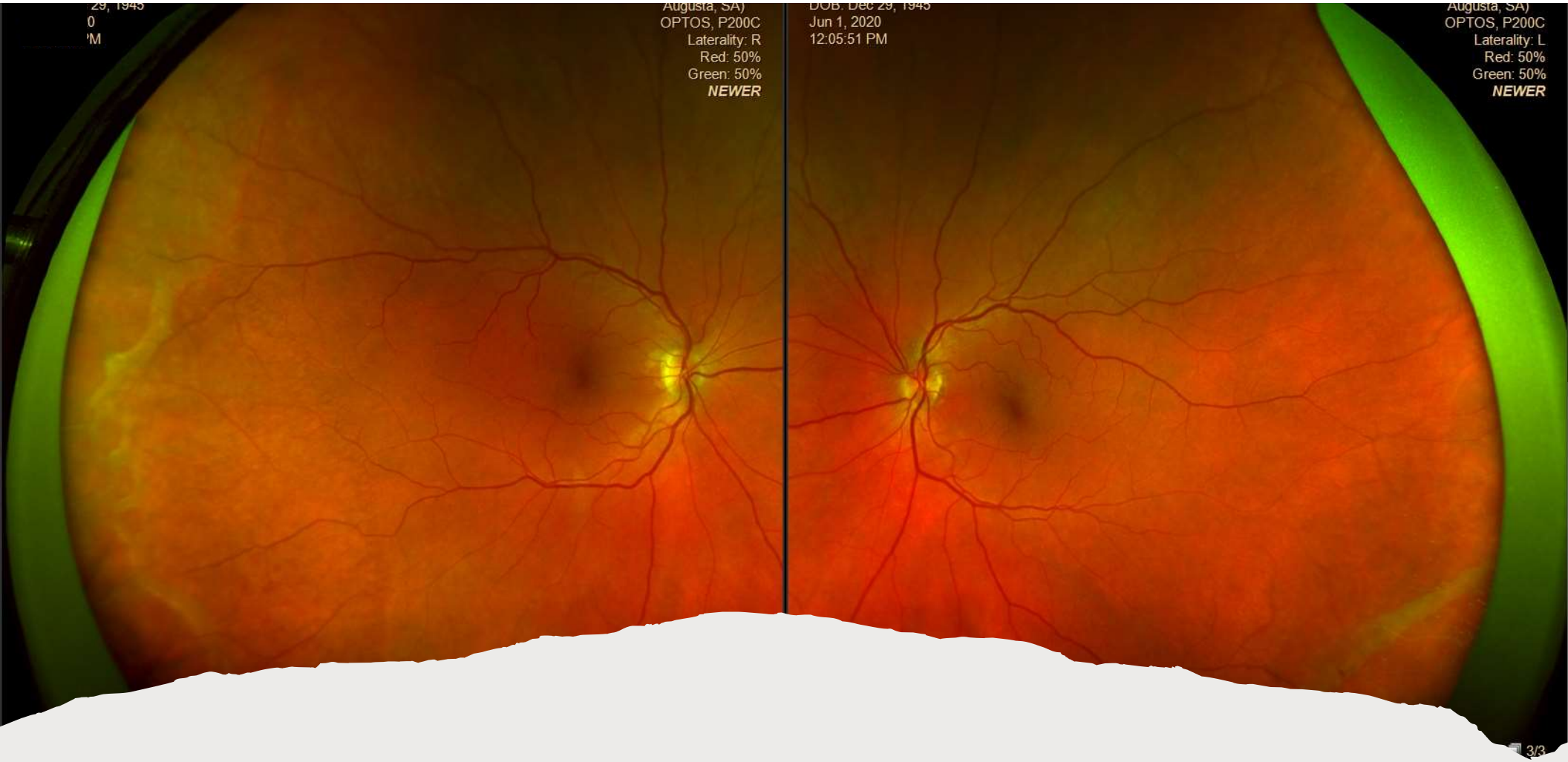
5 months later. LE retinoschisis slight less inflated. RE flatter and now hole in peripheral aspect of schisis and fluid loss.



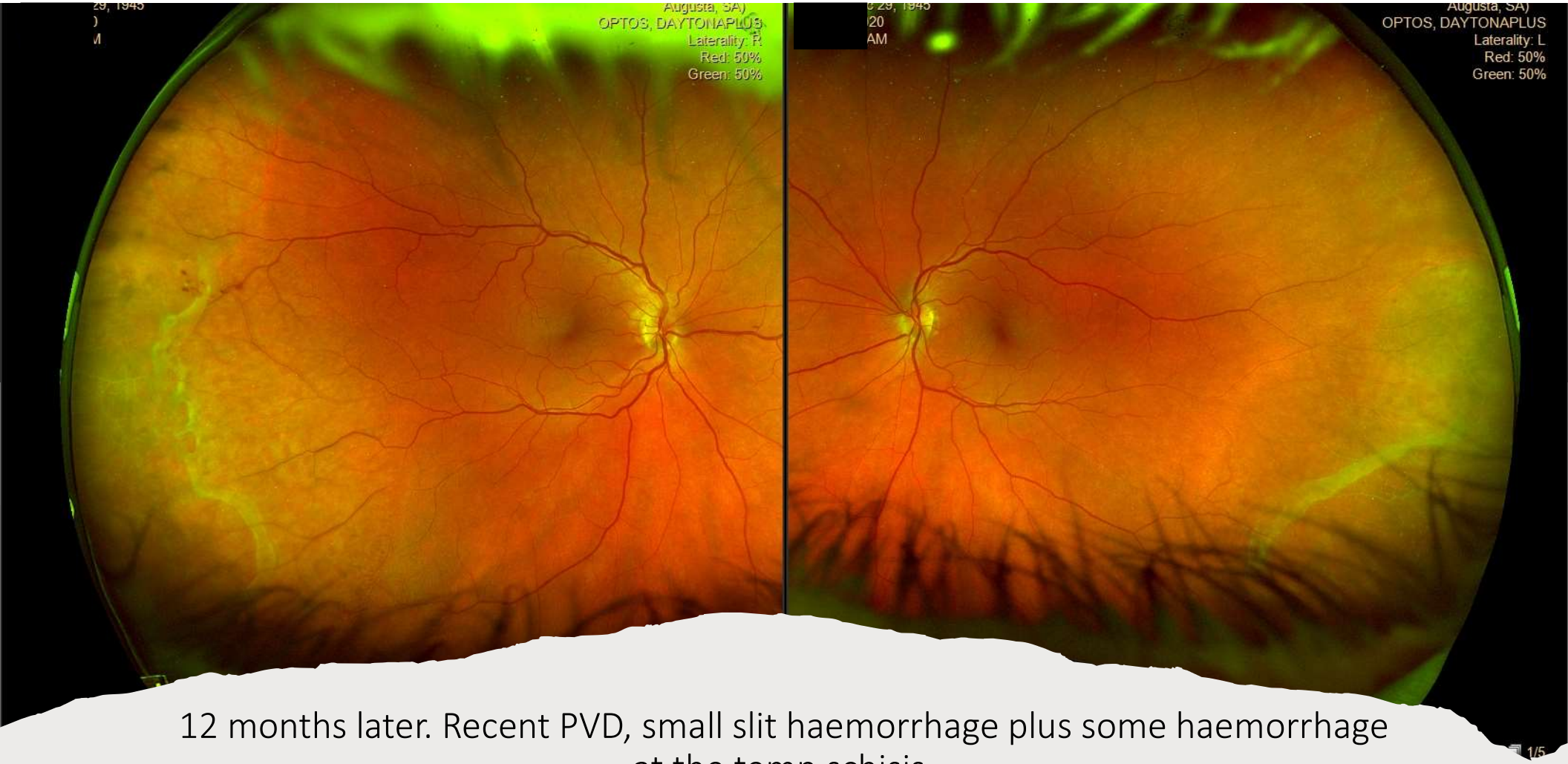
RE. In red free can see breaks and the posterior demarcation line.



7 months later yet again. Both flatter and the tear more evident.



12 months later. Both flatter yet again and the tear more evident.

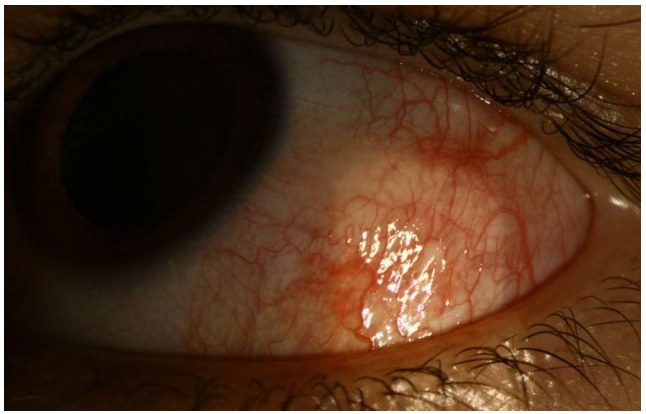


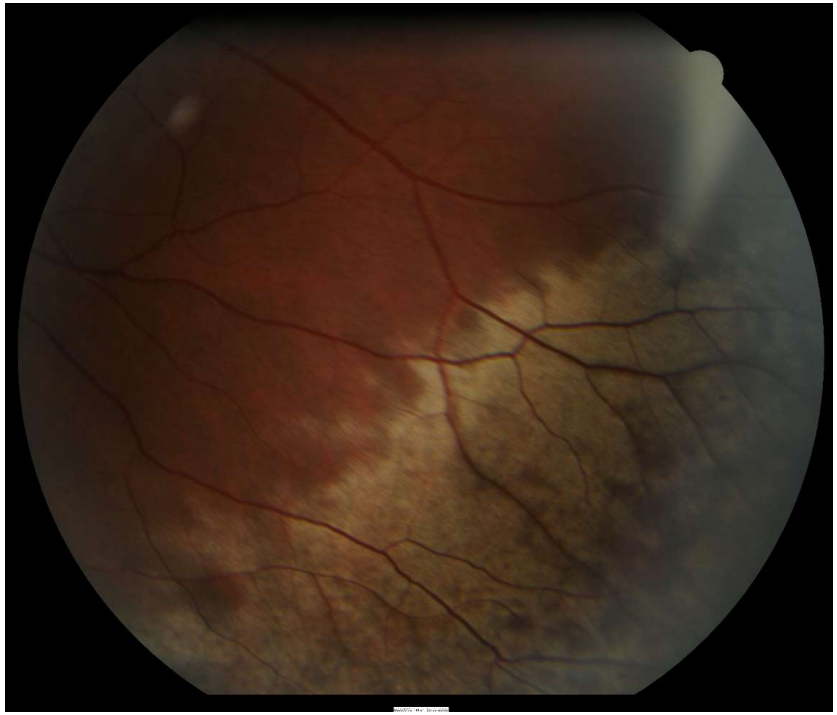
12 months later. Recent PVD, small slit haemorrhage plus some haemorrhage at the temp schisis.

Quiz Which is true regarding acquired retinoschisis

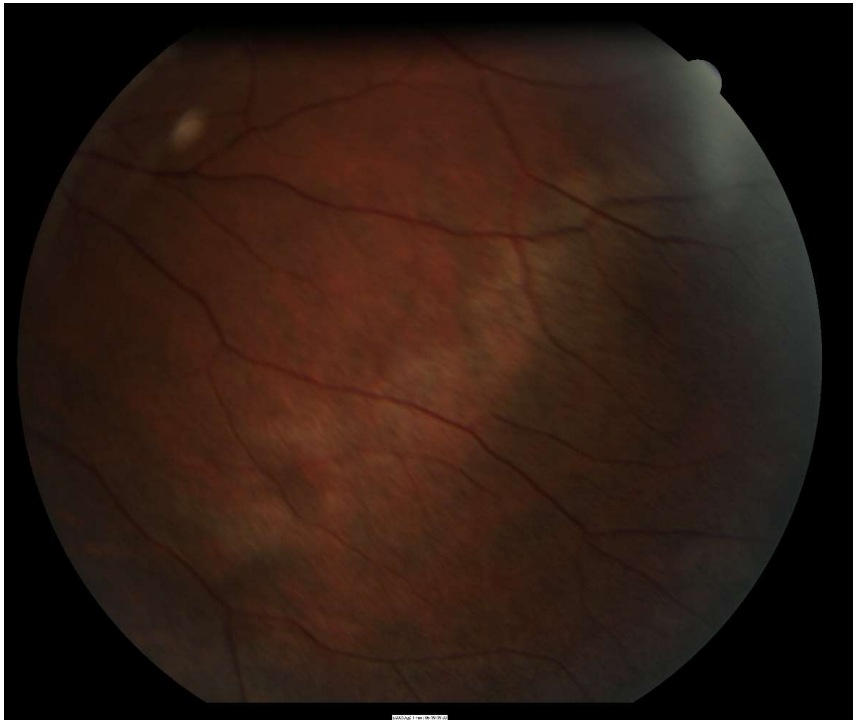
- 1 Inner leaf breaks have a pigmented edge
- 2 retinoschisis typically only occurs in one eye
- 3 Outer leaf breaks are mostly benign and do not need treatment
- 4 Retinoschisis are not uncommon amongst hyperopes & create positive scotomas
- 5 PVD and retinoschisis = rRD
- 6 Outer leaf breaks effectively create break that is a retinal detachment and always require treatment

Miss E.G. aet 14
Walking through playground
Struck LE with errant tennis ball
Vision sl blurred, Sore red eye,ache



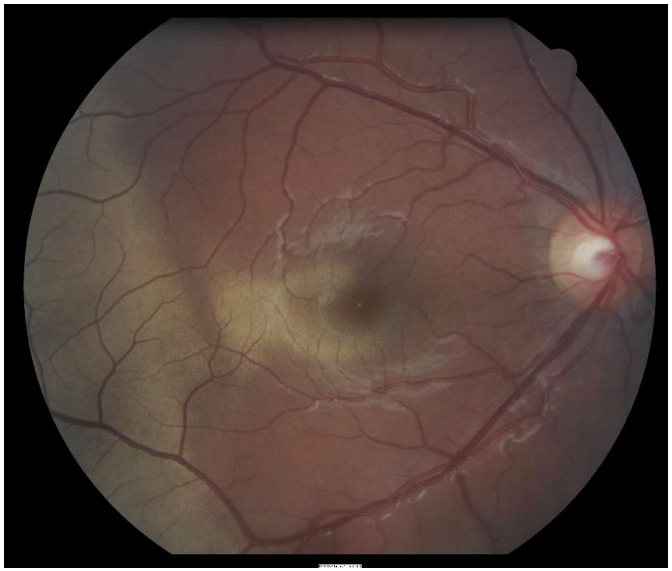


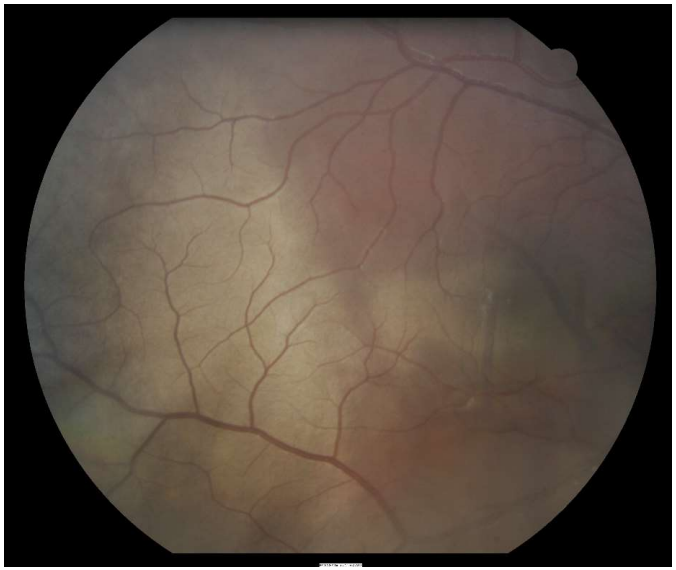
2
days
later



Commotio Retinae 'violent shaking retina'

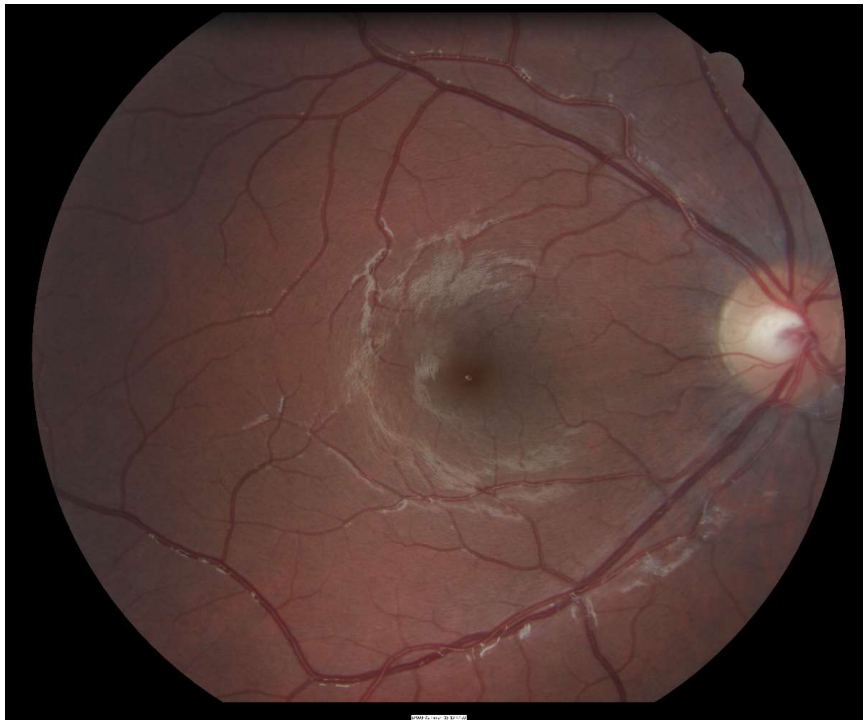
traumatic retinopathy secondary to direct or indirect trauma to the globe.
''Shockwave''
glistening grey-white opacification of the retina with or without intraretinal hemorrhages and RPE mottling.
blunt trauma has a compressive effect on the inner choroid, which produces outer retinal ischemia.
disruption of photoreceptor outer segments at IS/OS and begins to recover within 1 week
Tx nil, high dose steroids?
Patients with commotio retinae involving macula have poorer prognosis
6% of patients with macular involvement are permanently visually impaired with a VA of <6/9





Mr. B.P.
aet 14 ‘Pool toy

1 week
later



Quiz Which of the statements is false regarding Commotio retinae

a/ Angle recession is a possible sequalae to Commotio retinae
b/ No treatment is generally necessary following Commotio retinae. Observation and the retinal findings should start resolving within the week
c/ Anticoagulants PO should be initiated immediately to resolve the retinal ischaemia following Commotio retinae
d/ Traumatic Iritis often presents with a patient diagnosed with Commotio retinae

MCQ Which of these statements about lattice degeneration is false

- 1/ All lattice degenerations should be treated with retinal laser to prevent progression to rRD
- 2/ Approx 60% of eyes with rRD have lattice degeneration
- 3/ Lattice degeneration is more common amongst high myopes
- 4/ Lattice degeneration has stronger adhesion to the vitreal face and are more prone to retinal tears with PVD

Quiz A solo flat, pigmented lesion is found in the temporal periphery of one eye. It has both hypopigmented lacunae and a halo. Appropriate course of action is

a/ Image the lesion and review in 6 months
b/ Assess for FAF hypoautofluoresence with fluorescence of only the lacunae & halo
c/ Refer to GP on suspicion of Gardner's syndrome
d/ Refer to retinal Ophthalmologist as they are difficult to tell between typical & atypical
e/ all of the above

Quiz Which is true regarding acquired retinoschisis

- 1 Inner leaf breaks have a pigmented edge
- 2 retinoschisis typically only occurs in one eye
- 3 Outer leaf breaks are mostly benign and do not need treatment
- 4 Retinoschisis are not uncommon amongst hyperopes & create positive scotomas
- 5 PVD and retinoschisis = rRD

Next : Last breakout group.

Quiz Which of the statements is false regarding Commotio retinae

a/ Angle recession is a possible sequalae to Commotio retinae
b/ No treatment is generally necessary following Commotio retinae. Observation and the retinal findings should start resolving within the week
c/ Anticoagulants PO should be initiated immediately to resolve the retinal ischaemia following Commotio retinae
d/ Traumatic Iritis often presents with a patient diagnosed with Commotio retinae

MCQ Which of these statements about lattice degeneration is false

- 1/ All lattice degenerations should be treated with retinal laser to prevent progression to rRD
- 2/ Approx 60% of eyes with rRD have lattice degeneration
- 3/ Lattice degeneration is more common amongst high myopes
- 4/ Lattice degeneration has stronger adhesion to the vitreal face and are more prone to retinal tears with PVD

Quiz A solo flat, pigmented lesion is found in the temporal periphery of one eye. It has both hypopigmented lacunae and a halo. Appropriate course of action is

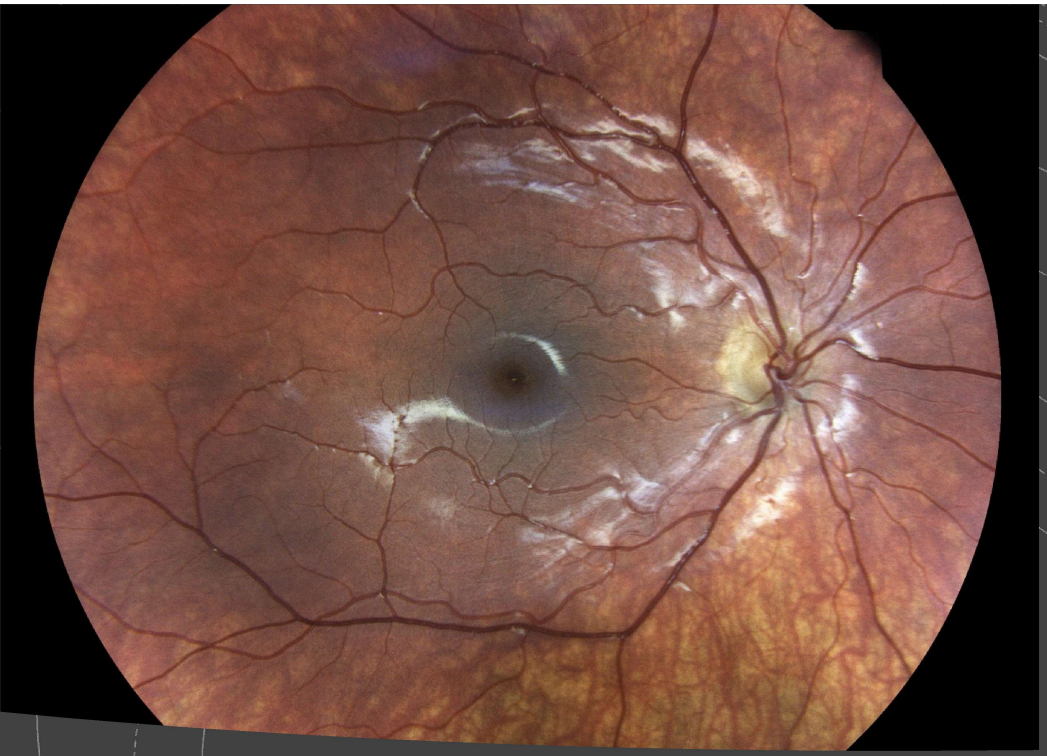
a/ Image the lesion and review in 6 months
b/ Assess for FAF hypoautofluoresence with fluorescence of only the lacunae & halo
c/ Refer to GP on suspicion of Gardner's syndrome
d/ Refer to retinal Ophthalmologist as they are difficult to tell between typical & atypical
e/ all of the above ✓

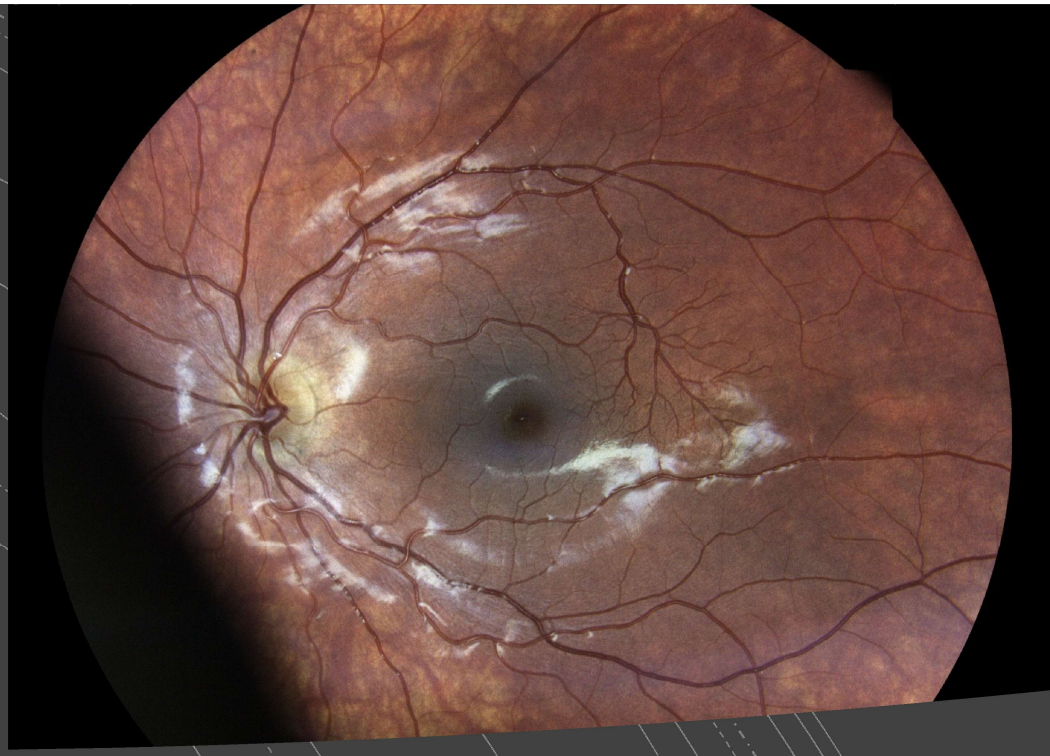
Quiz Which is true regarding acquired retinoschisis

- 1 Inner leaf breaks have a pigmented edge
- 2 retinoschisis typically only occurs in one eye
- 3 Outer leaf breaks are mostly benign and do not need treatment
- 4 Retinoschisis are not uncommon amongst hyperopes & create positive scotomas
- 5 PVD and retinoschisis = rRD

Next : Section 4 if need one more weird case.

Congenital retinal macrovessel

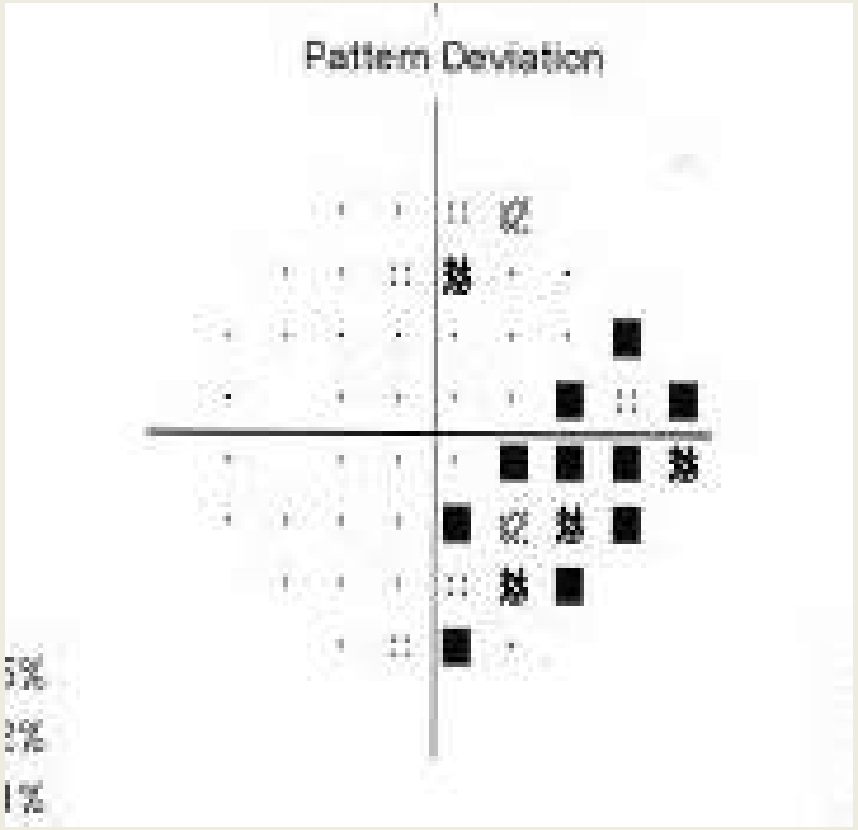


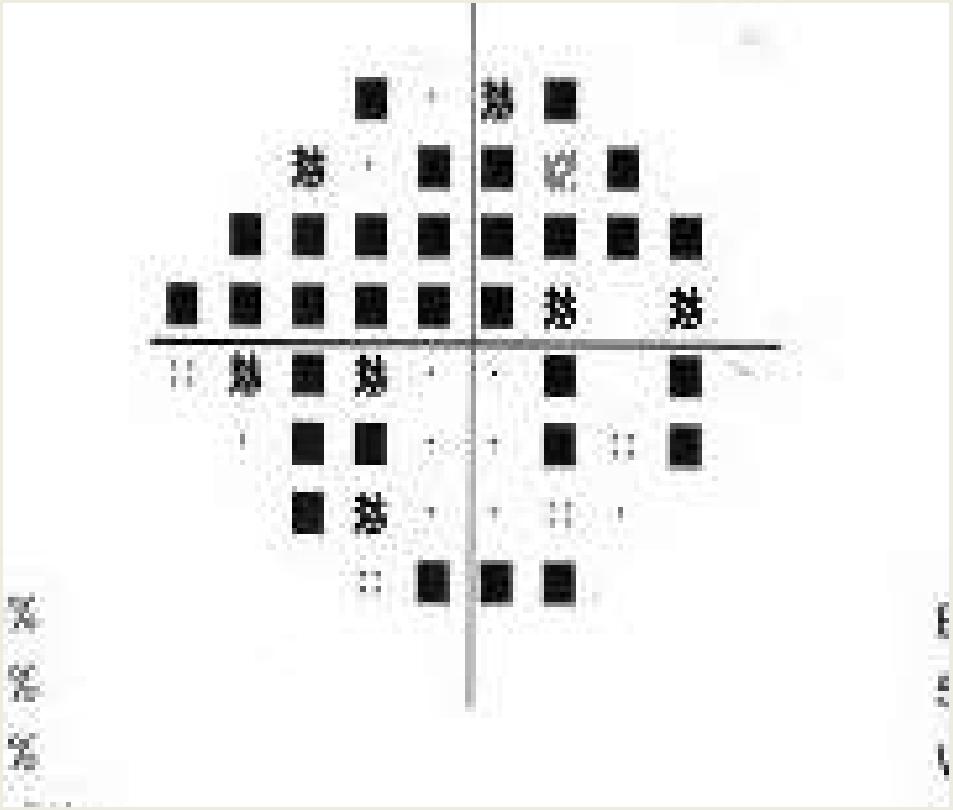


Miss ZS aet 14

- 11/8/20 reduced D vision N seems okay & eyes hurt
- -0.25/-1.25 x 180 6/24 -0.25/-0.25 x 35 6/24
- Pupils normal, CV not tested, External NAD
- Referred to neuroophthalmologist

Infero nasal step LE R supero nasal step?
BCVA 6/24 OU





REVIEW of OPTOMETRY

Is This Big Vessel a Big Problem?

Our patient presented with evidence of a congenital retinal macrovessel. How significant is this finding, and how should he be managed?

By Richard Zimbalist, OD

- 1869 and were later defined as a large aberrant artery or vein that crosses the horizontal raphe, with minimal to no effect on vision or color perception
- 1 in 200000
- Venous more common than arteriolar
- No CV effect & may affect vision

CASE REPORT

Year : 2016 | Volume : 2 | Issue : 3 | Page : 146-148

Congenital retinal macrovessel with macular edema: An unusual presentation

Geetika Khurana, Poninder Kumar

Department of Ophthalmology, Army College of Medical Sciences, Base Hospital, New Delhi, India

Congenital retinal macrovessel (CRM) is a rare developmental condition in which a large vein, artery, or occasionally an artery and a vein together, cross the macular region. CRMs do not affect vision unless there are associated foveolar cysts, foveal ectopia, pigmentary changes at fovea, macular hemorrhage, exudates, serous macular detachment, or the macrovessel crosses the fovea

CRM are usually benign, however, you must rule out Wyburn-Mason syndrome in the presence of neurologic symptoms.
Wyburn-Mason syndrome is characterized by the subsequent finding of an Arteriovenous malformation of the midbrain in the presence of an ipsilateral AVM of the retina.
Wyburn-Mason syndrome is a systemic condition that can cause hamartomas of the brain with severe neurologic deficit