The FLIO in AMD challange – what we know and what we don‘t understeand

Martin Hammer

Fluorescence lifetime imaging ophthalmoscopy (FLIO) measures the time constants of fluorescence decay which are characteristic for fluorescent molecules and their environment. We know that fundus autofluorescence (FAF) lifetimes in AMD patients are longer than in age-matched controls and an annular lifetime pattern was described[1, 2]. FAF lifetimes of drusen are heterogeneous, but hyperfluorescent drusen as well as subretinal drusenoid deposits (SDD) tend to longer lifetimes[2, 3]. Hyperpigmentations show clearly longer lifetimes than the intact RPE in the same patient[3]. Areas of geographic atrophy (GA) show long lifetimes[4].The lifetimes of drusenoid RPE detachments are long whereas hemorrhagic RPE detachments have short lifetimes[5]. This is what we know from published studies. In the following, we present individual cases who show conflicting findings or pose other questions. Limitations of this presentation are i. the quality of the OCT (besides the quality of the scans, their allocation to the fundus photography was difficult because of the poor quality of the en face images provided by the Zeiss Cirrus OCT) and ii. a limited accuracy of image registration (had to be done manually, I didn't find a software doing that reliably). Nevertheless, the findings presented here will teach us about pathomechanisms of AMD as well as the diagnostic potential of FLIO – if we will find answers to the questions (printed in bold in the brief description of each case), these cases pose. Thus, all answers, suggestions – even speculations – and more questions are welcome! Please address them to martin.hammer@med.uni-jena.de.

References

1. Sauer L, Gensure RH, Andersen KM, et al. Patterns of Fundus Autofluorescence Lifetimes In Eyes of Individuals With Nonexudative Age-Related Macular Degeneration. Invest Ophthalmol Vis Sci 2018;59:AMD65-AMD77.

2. Dysli C, Fink R, Wolf S, Zinkernagel MS. Fluorescence Lifetimes of Drusen in Age-Related Macular Degeneration. Invest Ophthalmol Vis Sci 2017;58:4856-4862.

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4. Dysli C, Wolf S, Zinkernagel MS. Autofluorescence Lifetimes in Geographic Atrophy in Patients With Age-Related Macular Degeneration. Invest Ophthalmol Vis Sci 2016;57:2479-2487.

5. Sauer L, Komanski CB, Vitale AS, Hansen ED, Bernstein PS. Fluorescence Lifetime Imaging Ophthalmoscopy (FLIO) in Eyes With Pigment Epithelial Detachments Due to Age-Related Macular Degeneration. Invest Ophthalmol Vis Sci 2019;60:3054-3063.

Contents

AMD_46	FAF of different lifetimes within geographic atrophy	134
AMD_54, baseline	neovascular AMD…	145
AMD_54, follow up 20 month	turning into atrophy surrounded by the “floodplain” phenomenon	156
AMD_67, baseline	Intermediate AMD…	166
AMD_67, follow up 52 month	developing GA as well as hypofluorescent outer retinal atrophy	175
AMD_68, baseline	areas of massive hyperpigmentation…	183
AMD_68, follow up 47 month	turning into atrophy which is hyperfluorescent at short wavelengths	193
FLIO-33	intermediate AMD, long lifetimes in the macula	202
FLIO-37	subretinal material in atrophy causing hyperfluorescence	211
GA_19, baseline	material inside GA is hyperfluorescent with short lifetimes…	220
GA_19, follow up 55 month	and persists for 4 ½ years	229

AMD_02, 53 years at baseline

The arrowhead shows an unknown structure of long FAF lifetimes at baseline (01/26/2015) which was slightly hyperfluorescent in LSC but unremarkabel in color fundus photography (CFP) and infrared (IR). OCT reveals a large but shallow druse. The patient developed CNV (showing short lifetimes) which is not dry after multiple injections of Ranibizumab and Aflibercept (09/24/2020). Although the druse seems to grow in z-direction, hyperfluorescence disappeared and the long lifetimes are not seen any more in the follow up. **What fluorophore/anatomical structure might have caused the long-living fluorescence and why is it gone while the druse was growing?**

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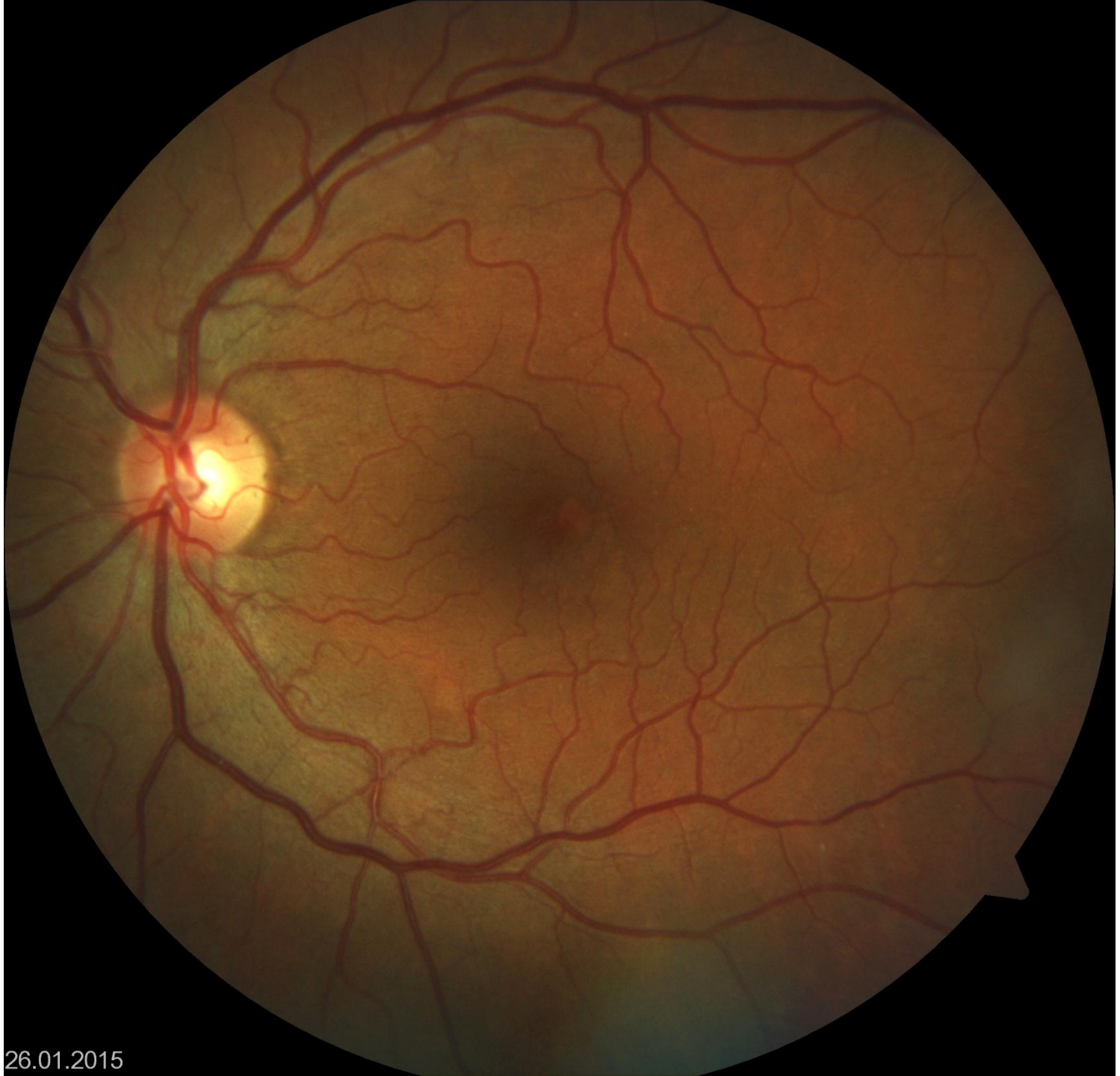

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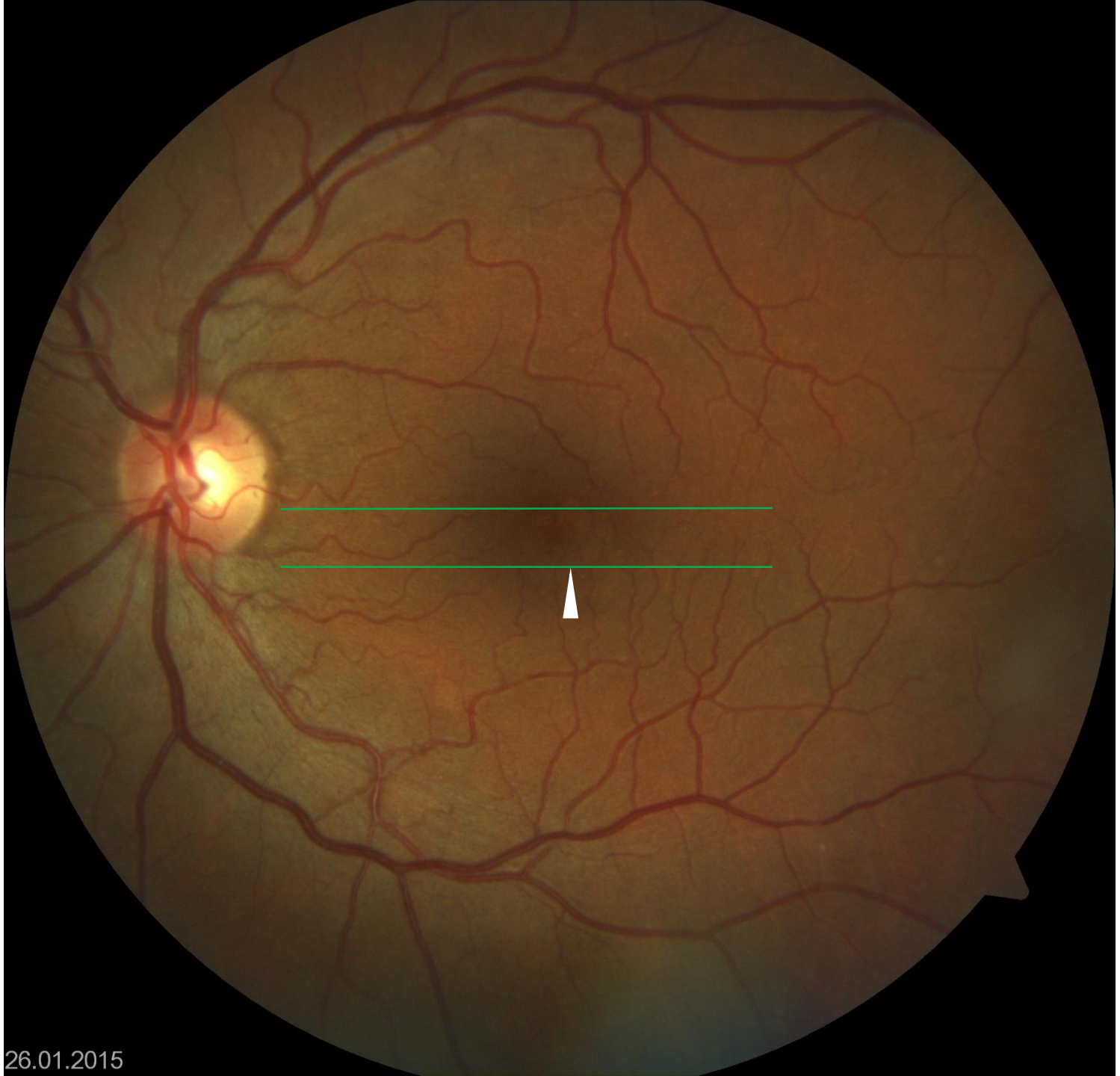
01/26/2015



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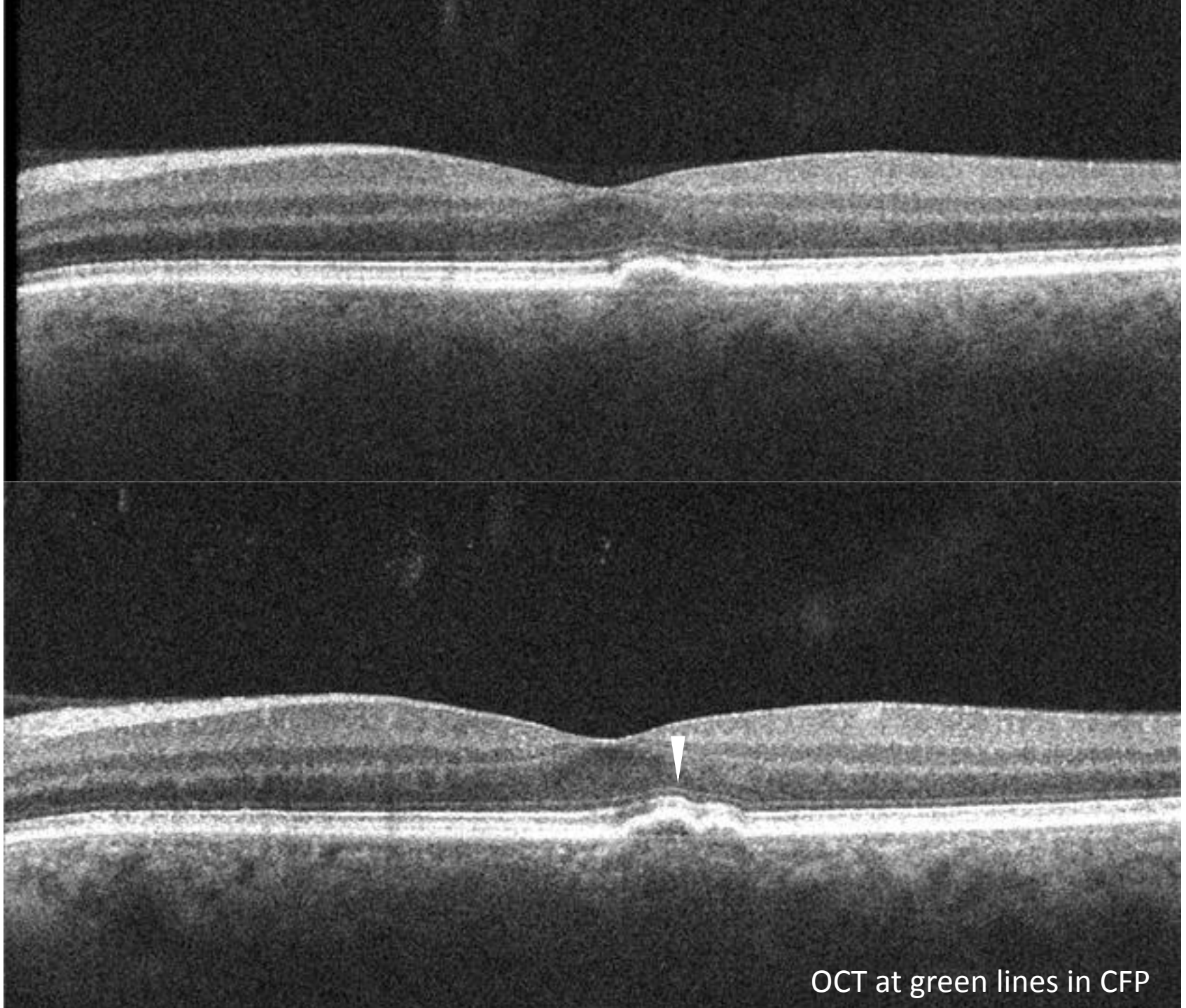
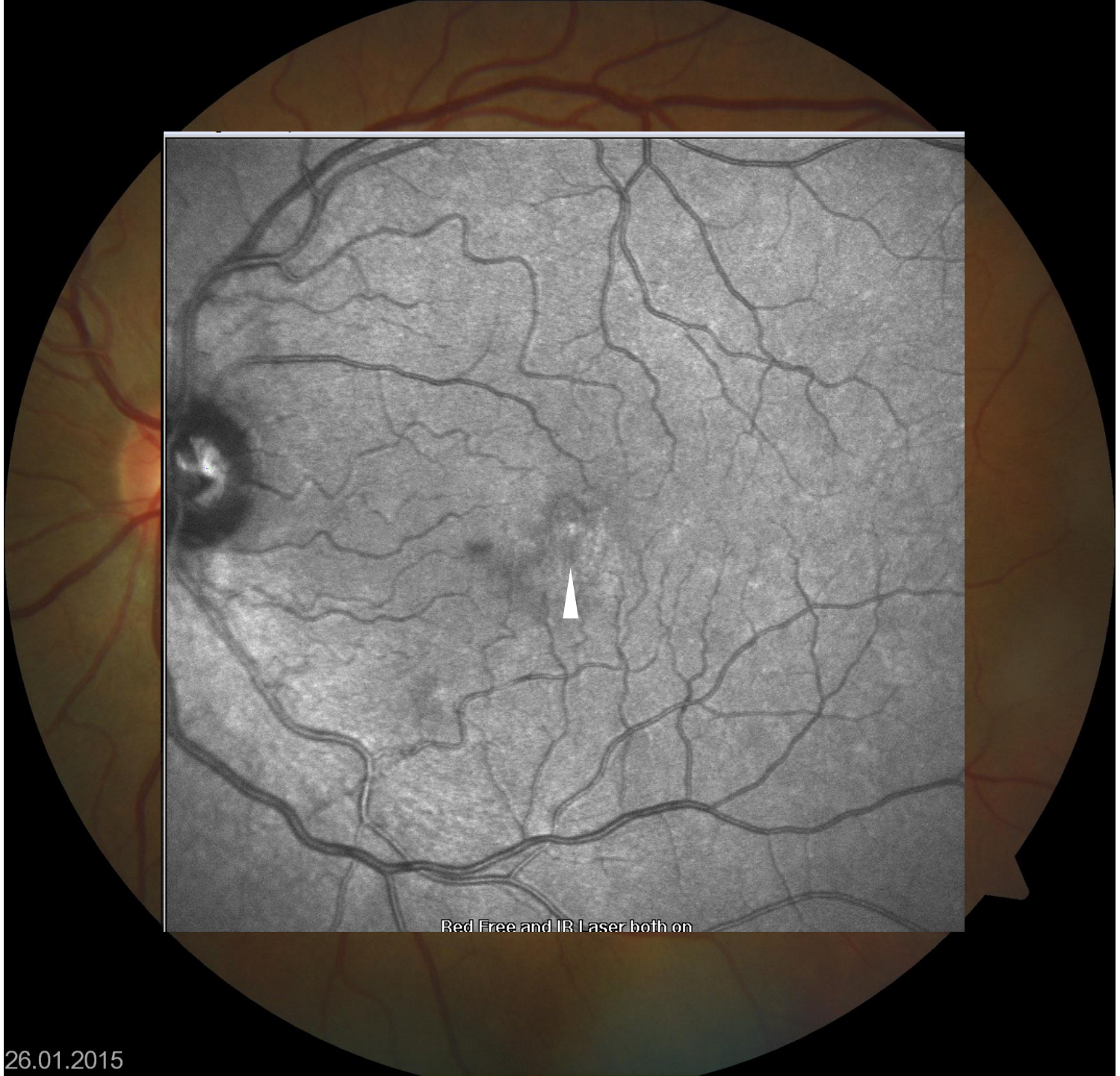


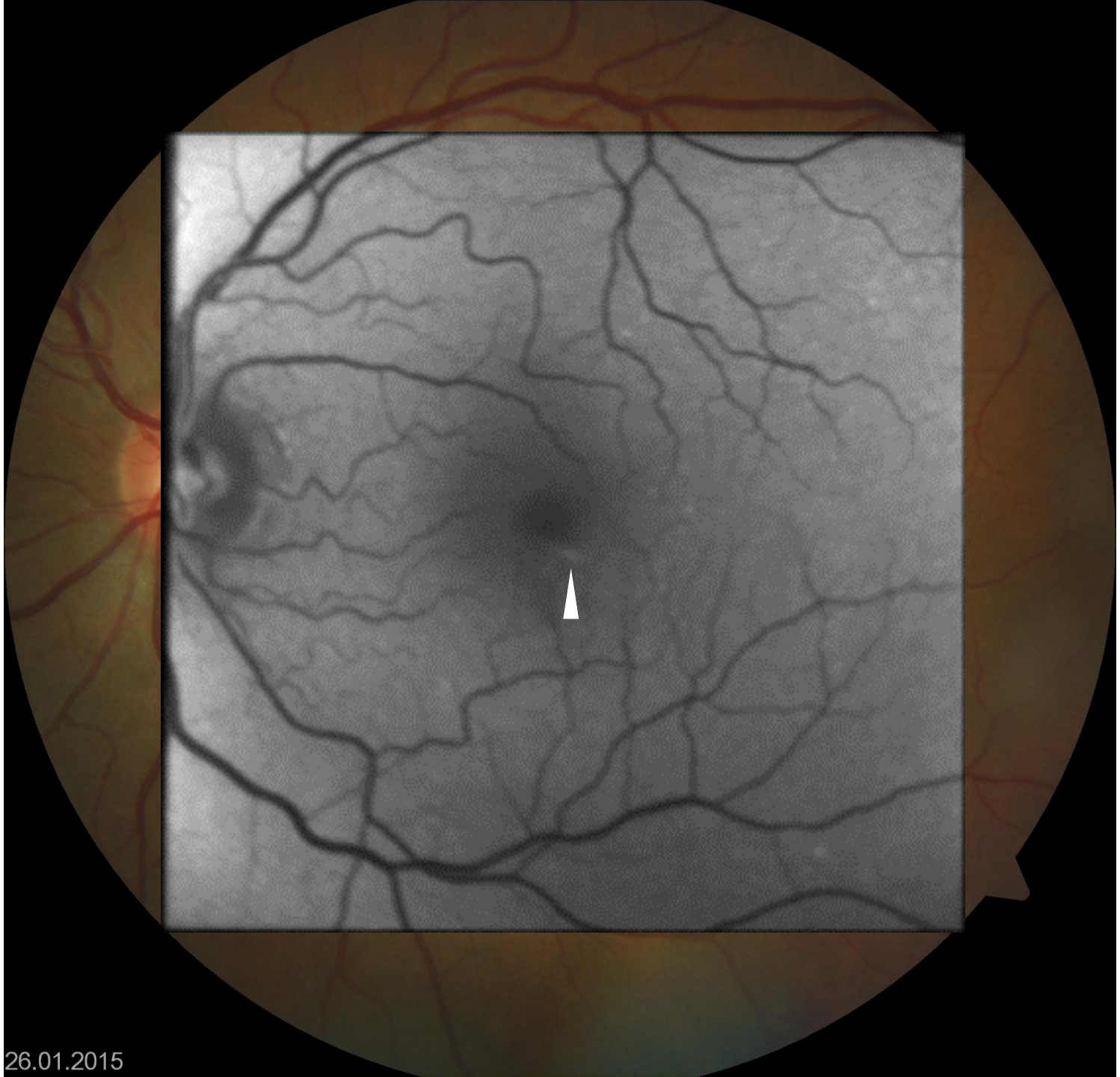
OCT at green lines in CFP

01/26/2015



IR

01/26/2015



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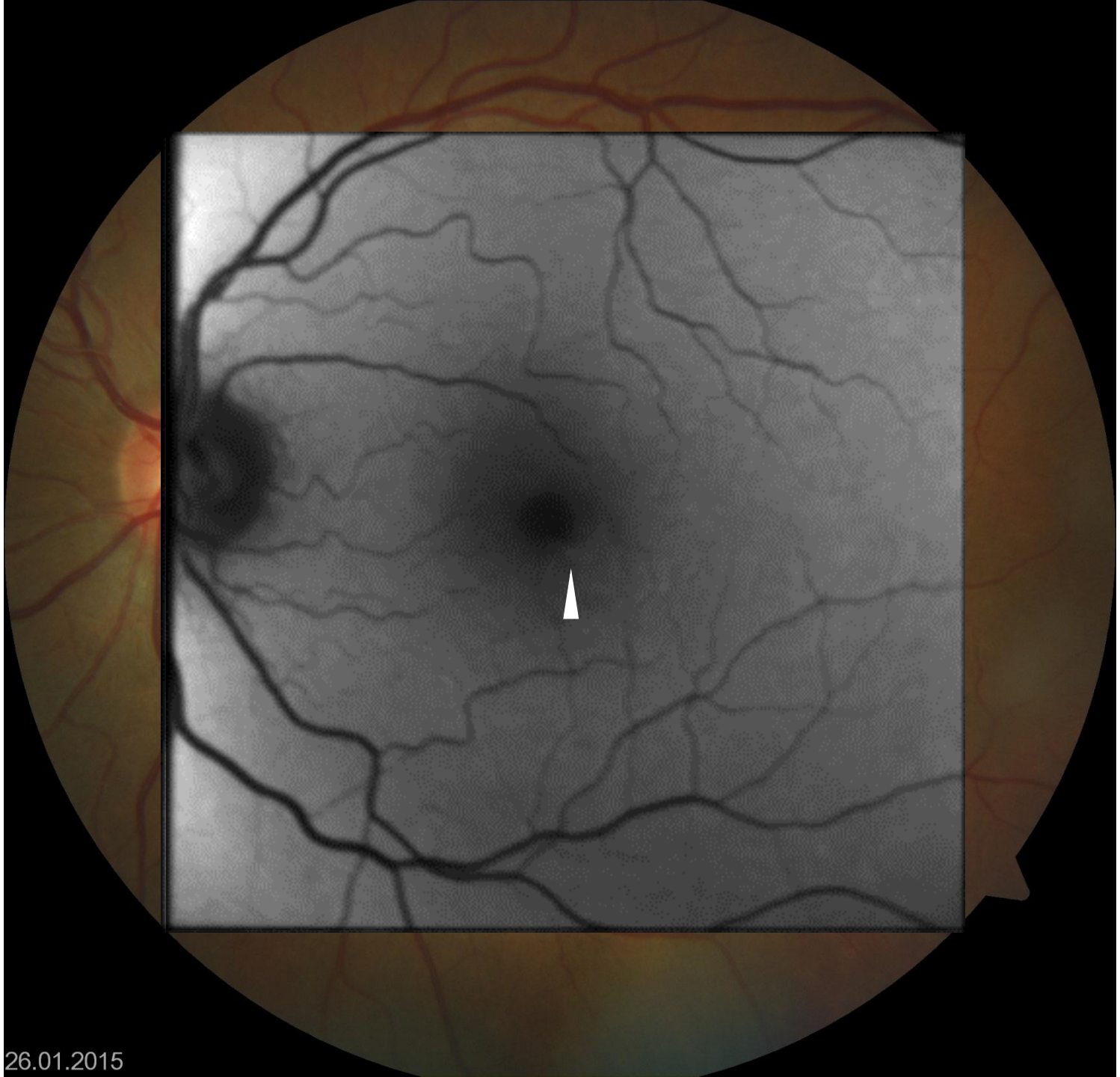

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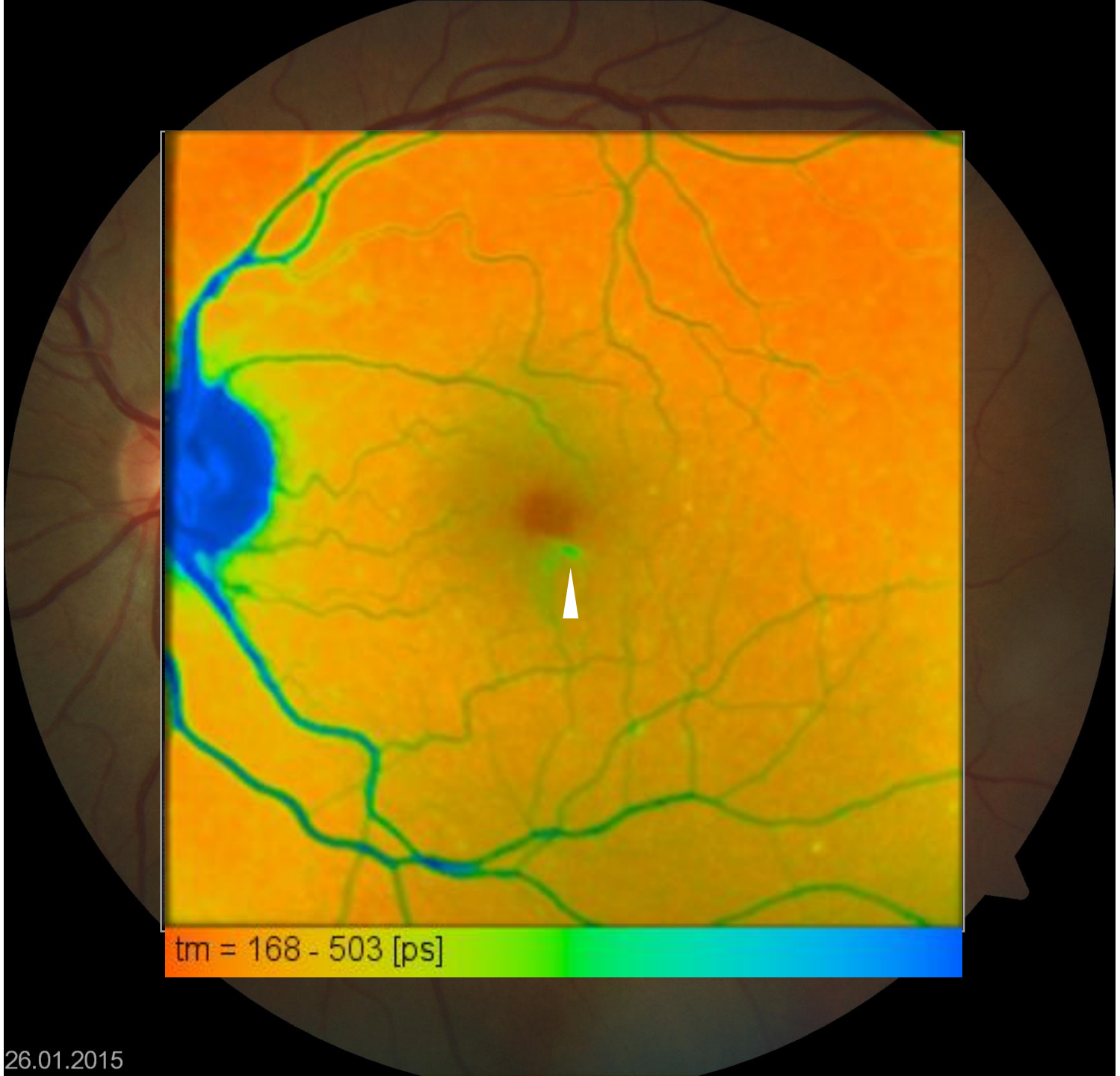
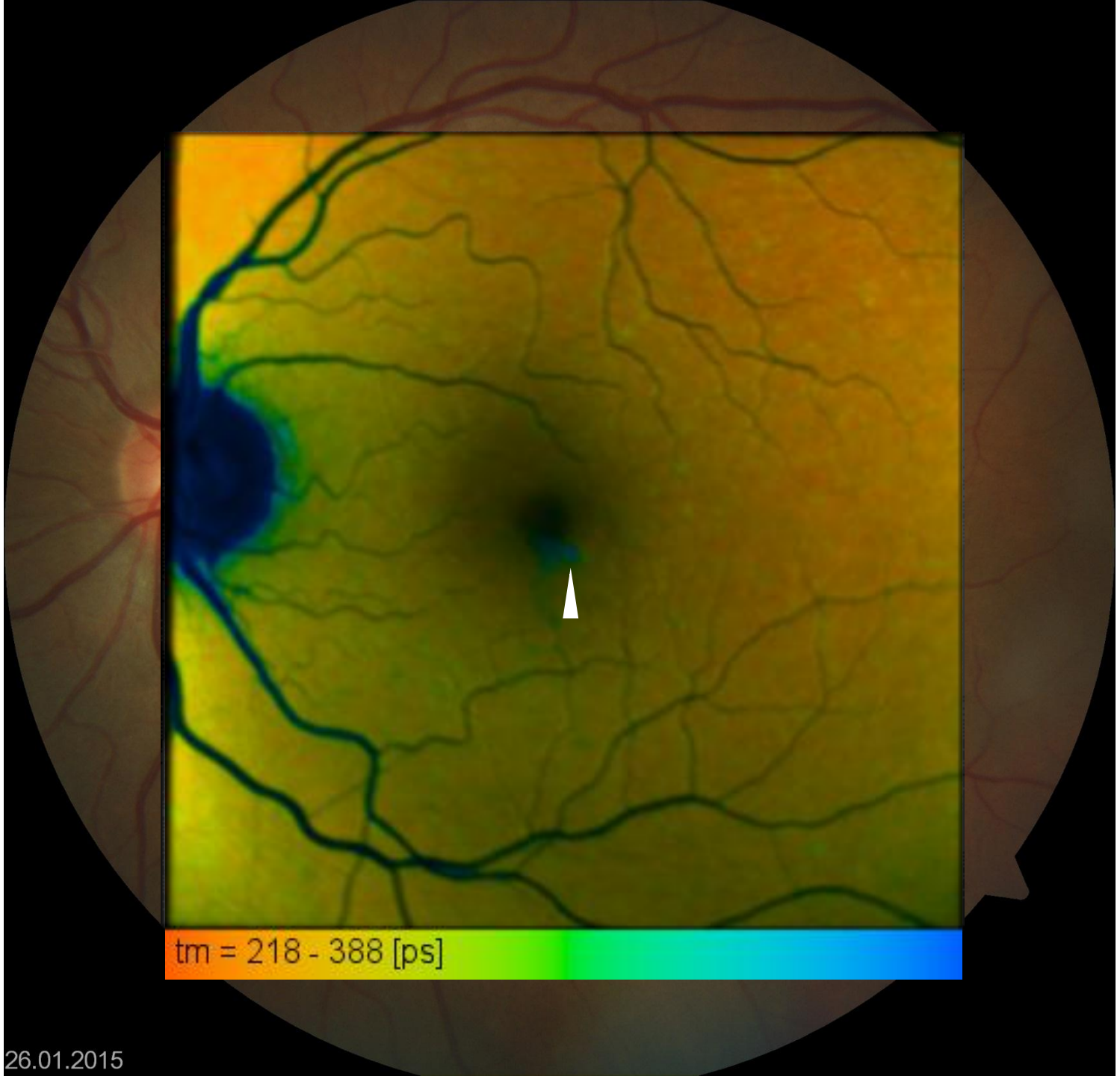


FLIO SSC

01/26/2015



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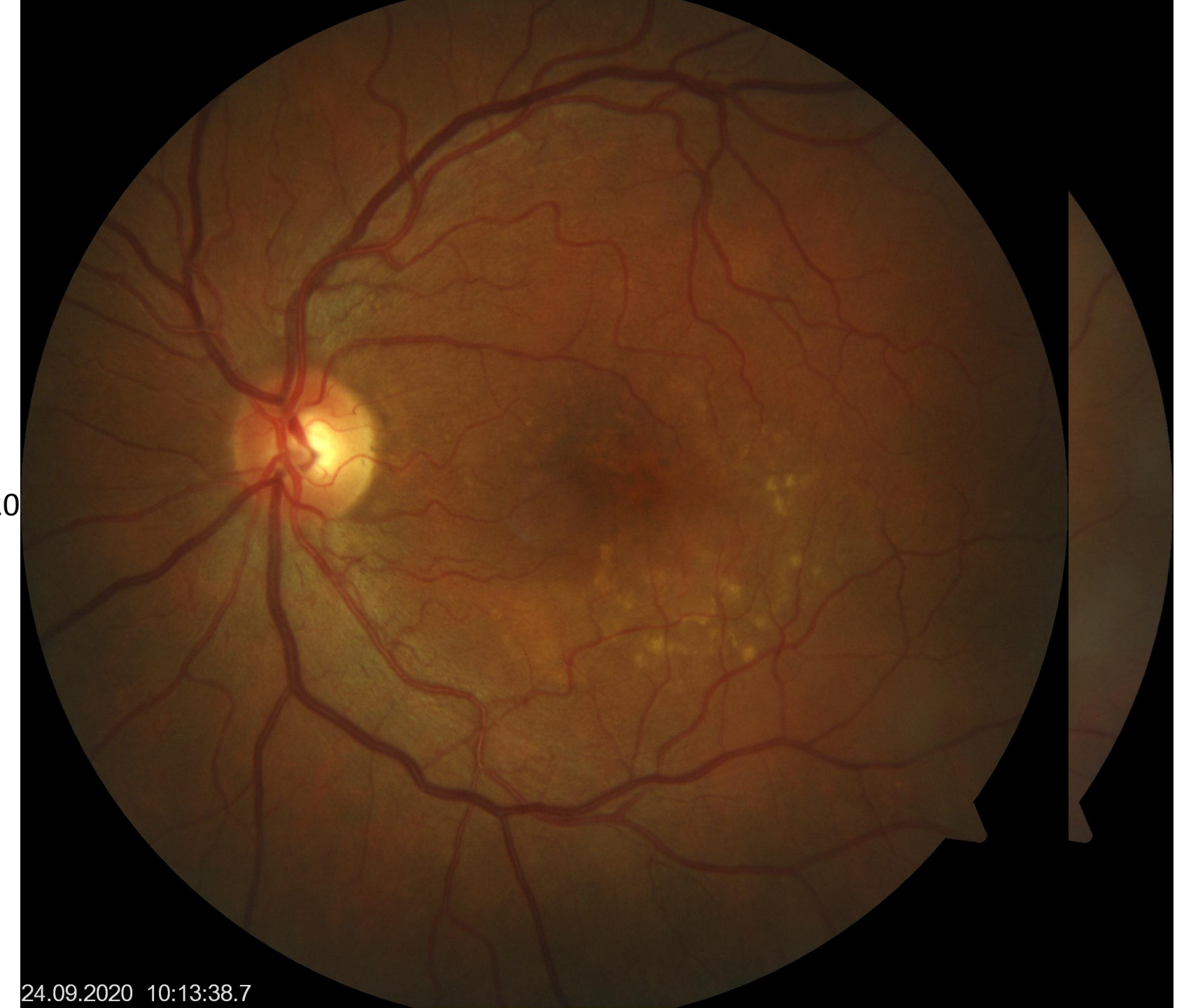

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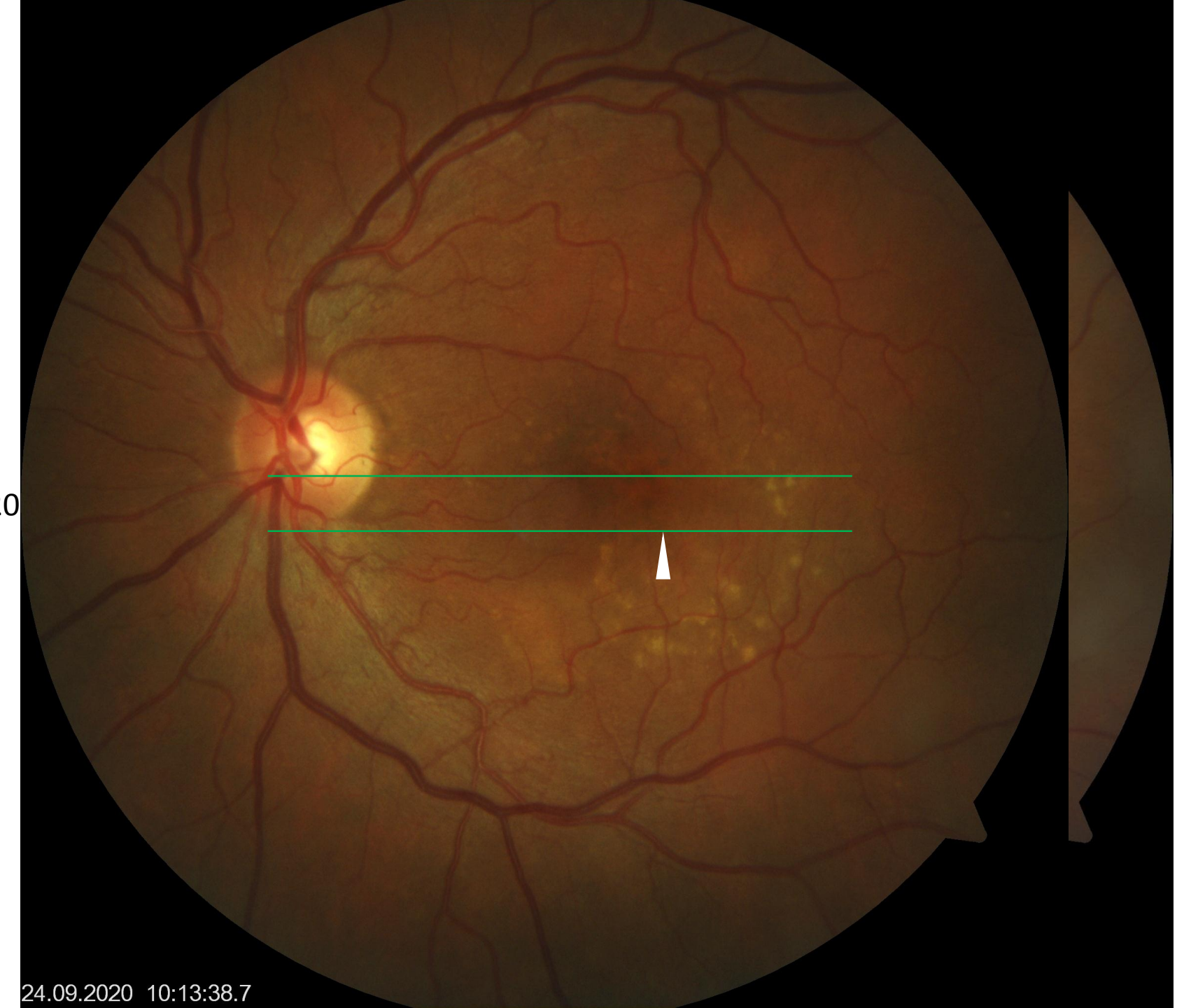

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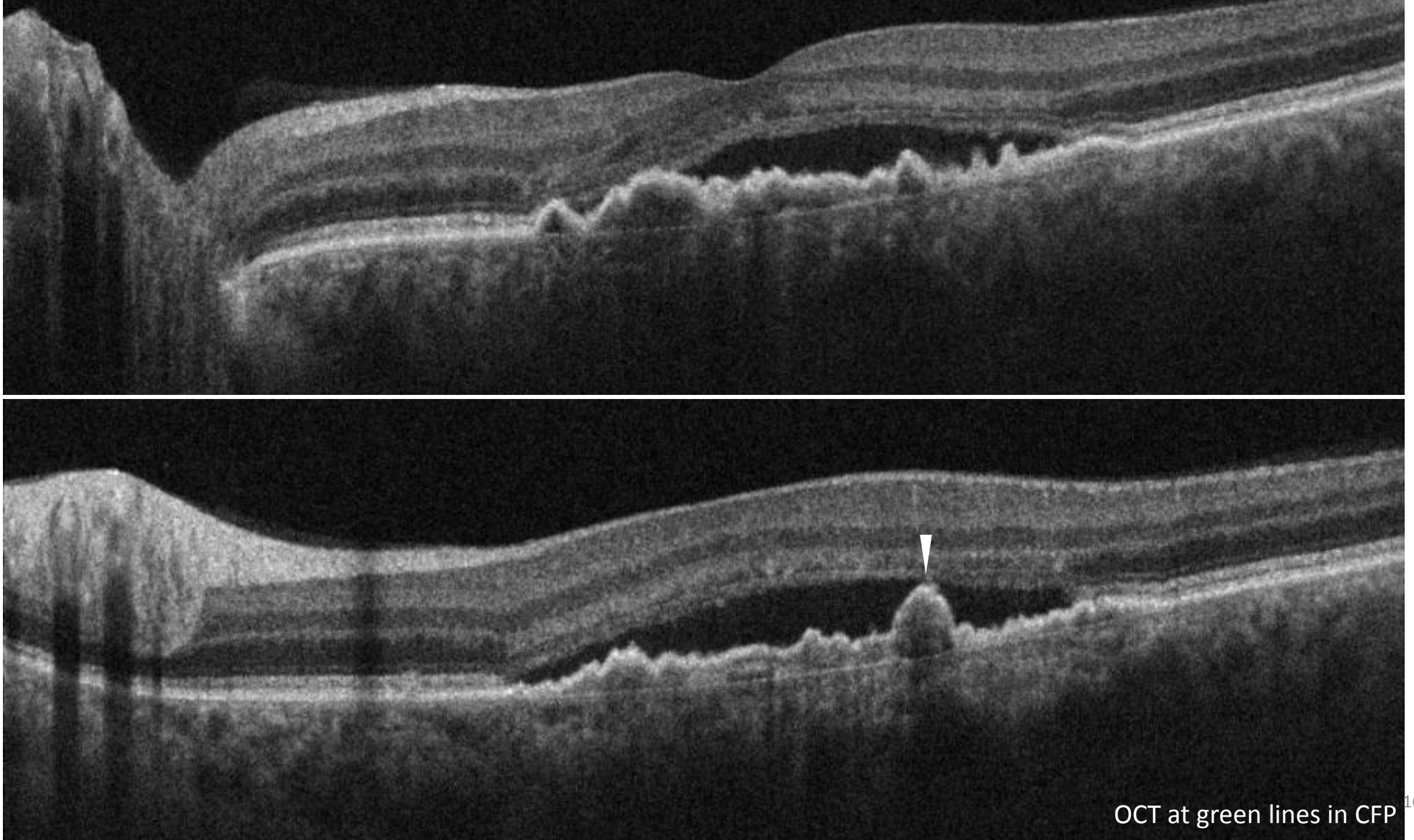

OCT at green lines in CFP

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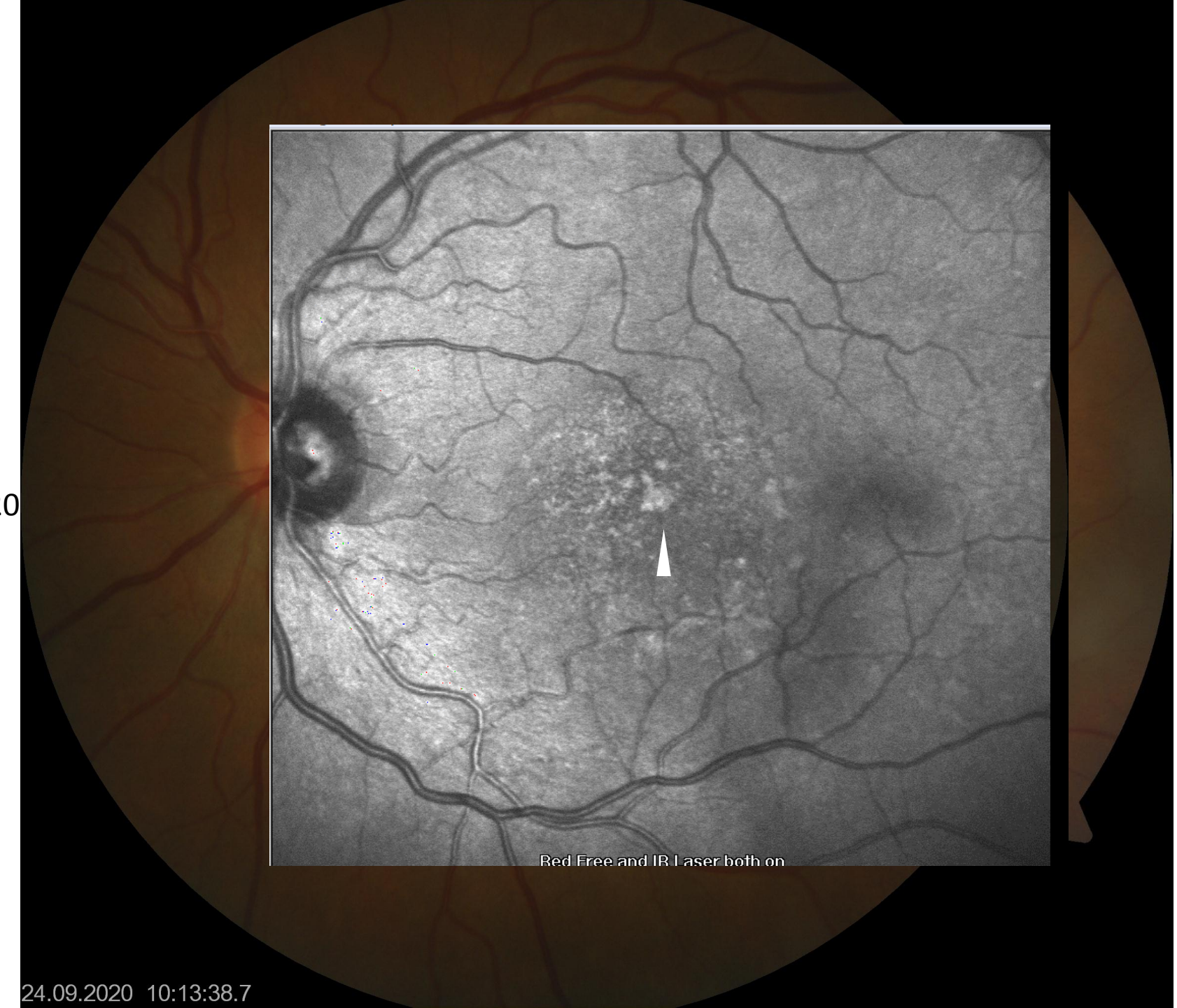

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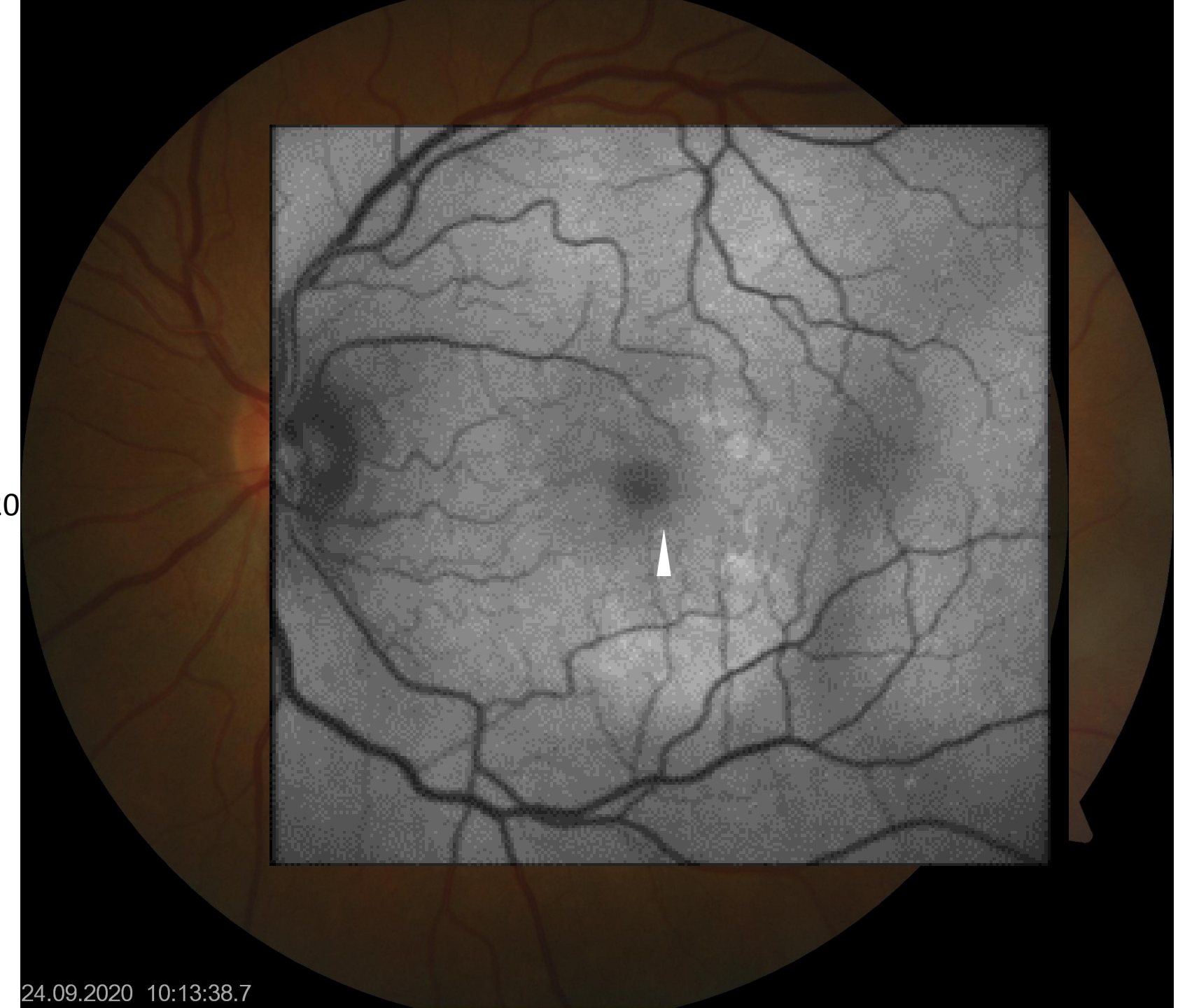

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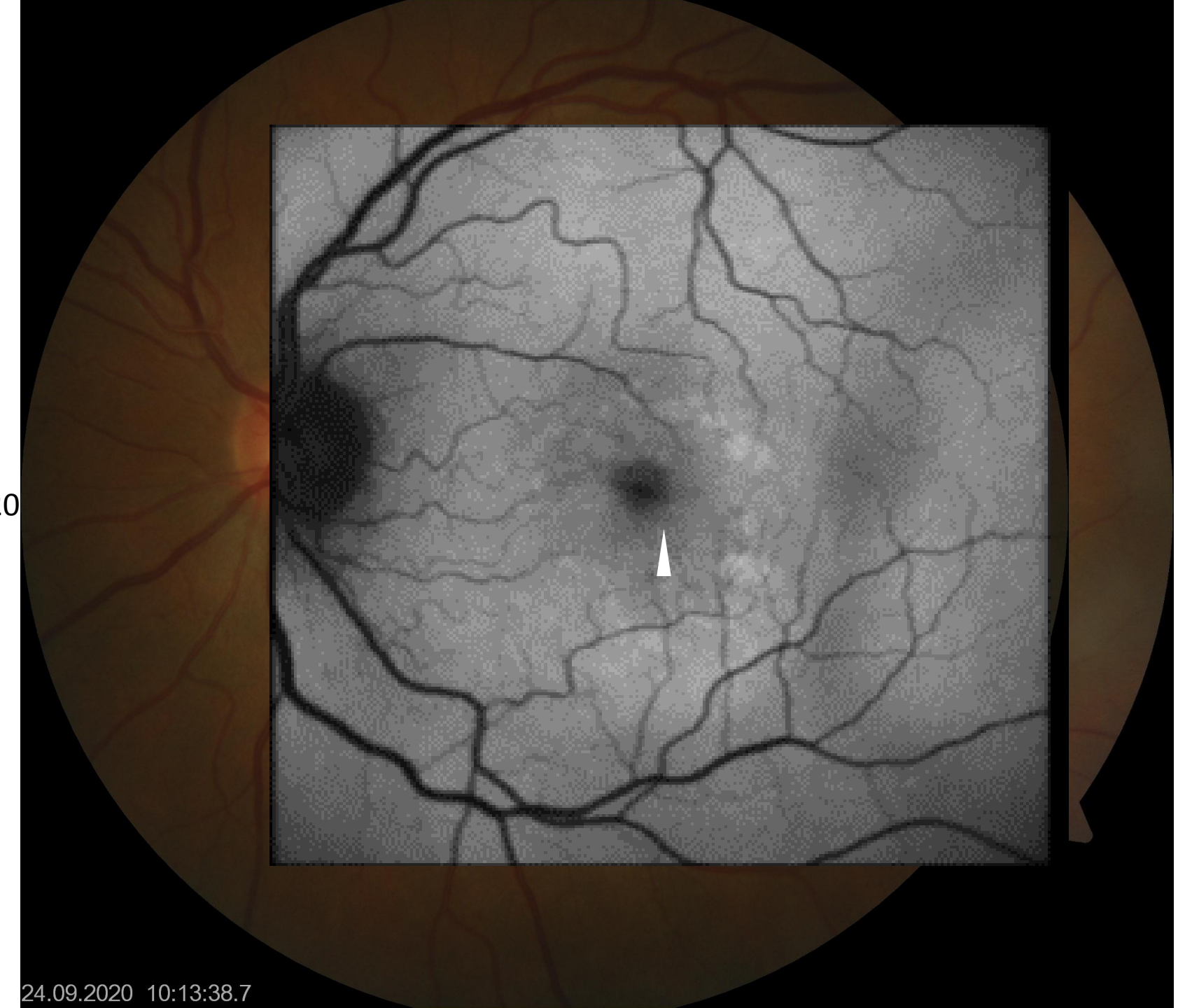

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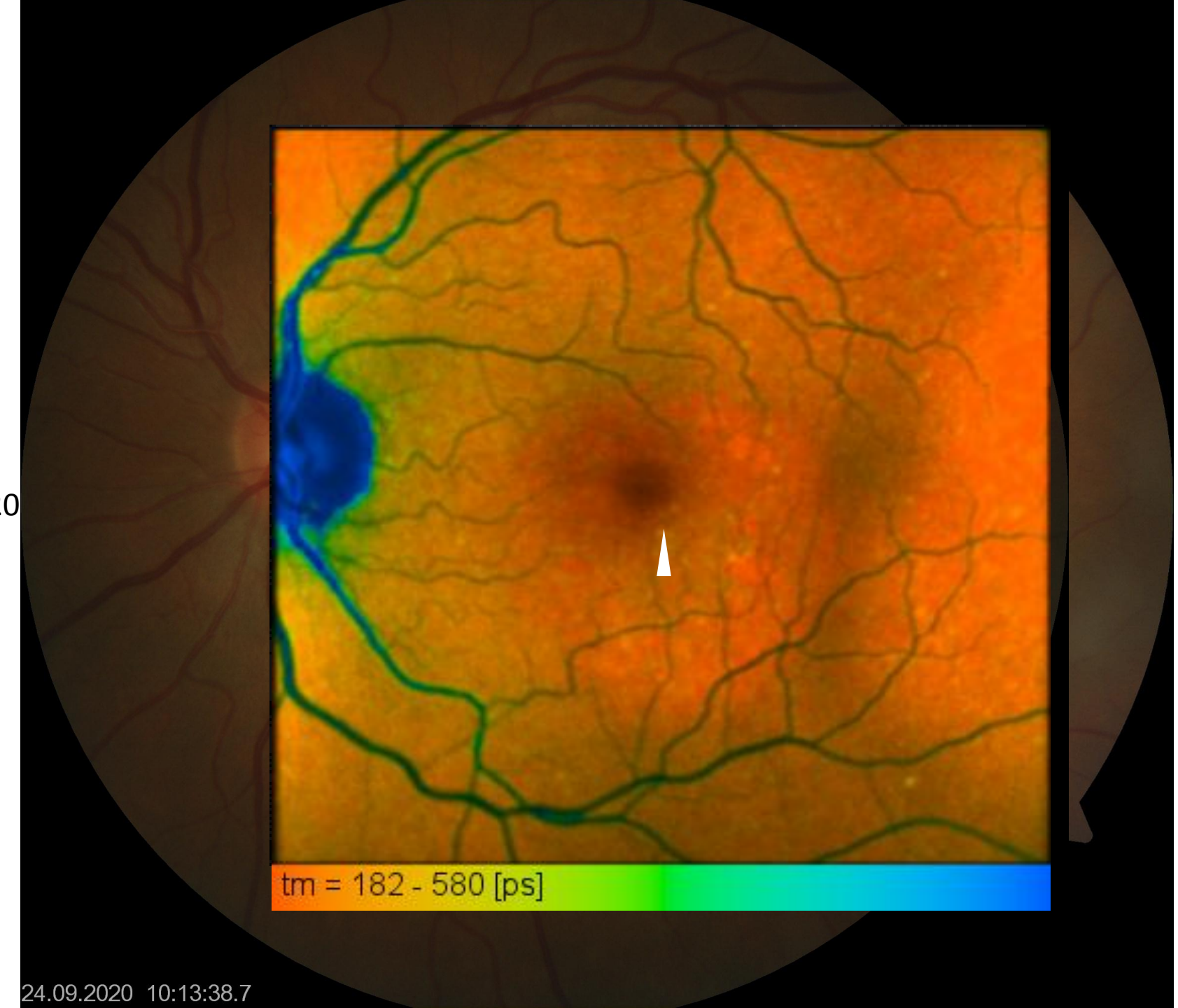

FLIO SSC

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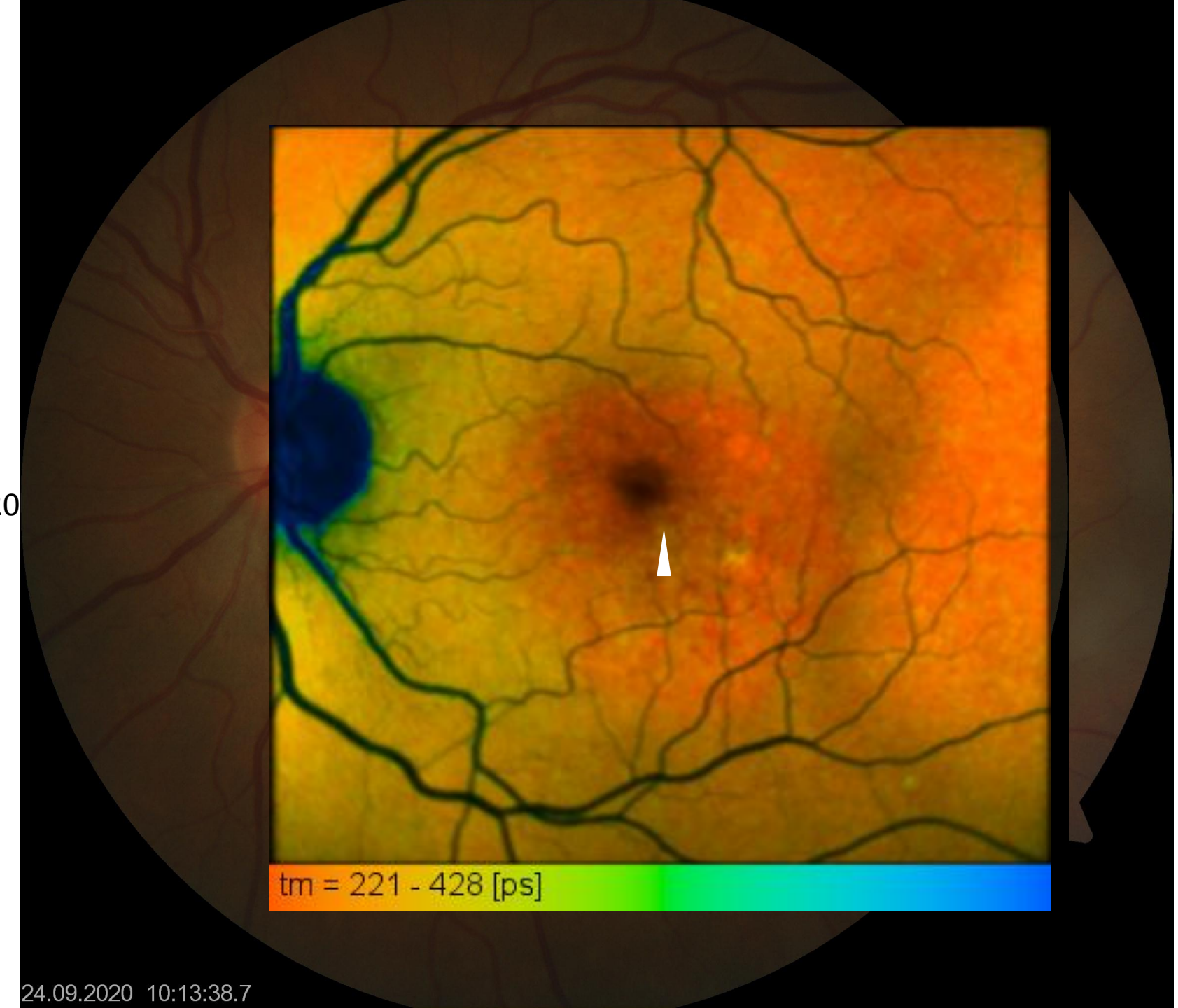
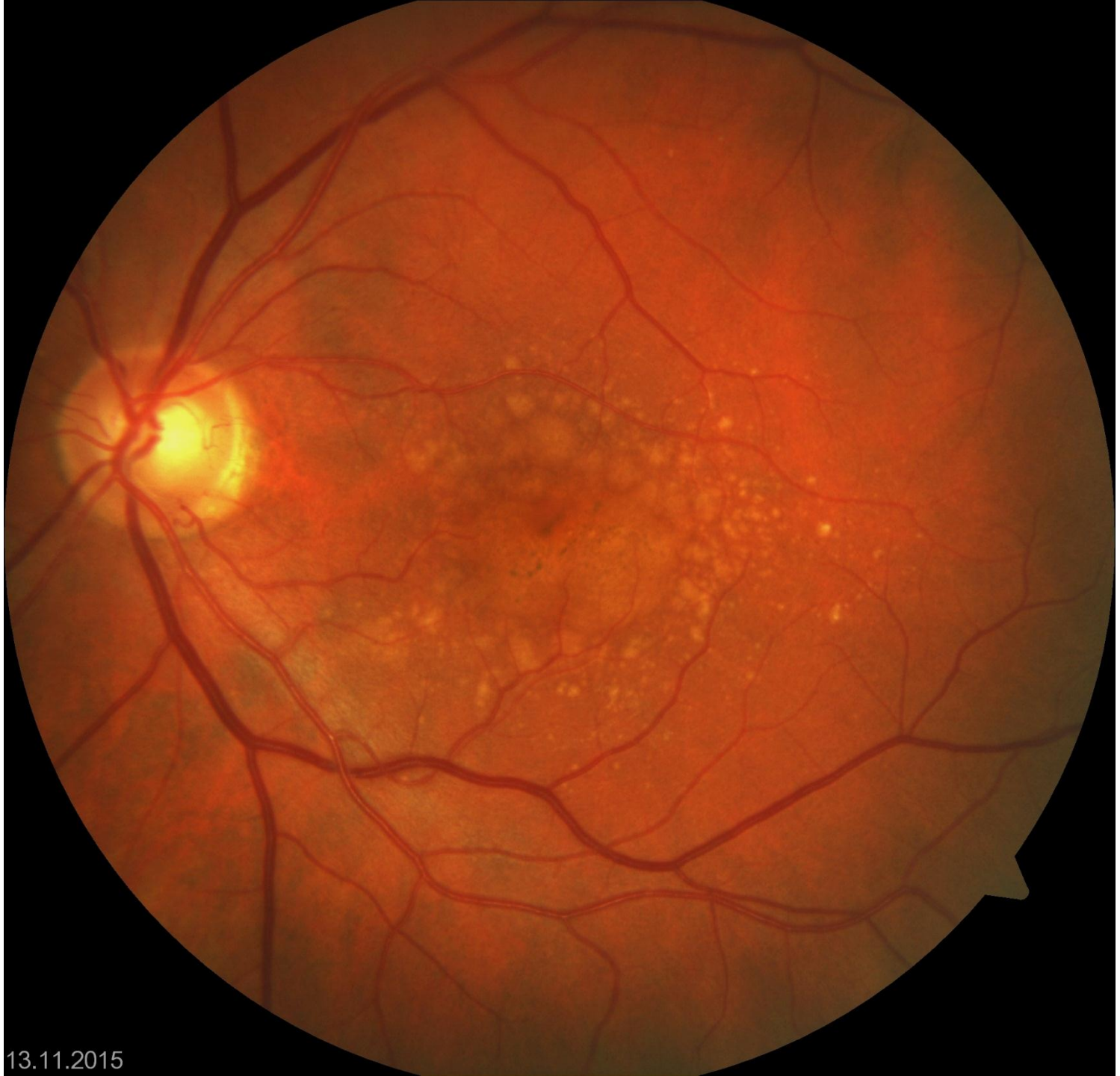


FLIO LSC

AMD_05, 72 years at baseline

The arrowhead points to an RPE detachment which was hyperfluorescent and showed short lifetimes. At follow up 2 years later, the detachment was resorbed and hyperfluorescence as well as short lifetime disappeared. However, drusen with short lifetimes remained (normally, drusen FAF tends to longer FAF lifetimes than RPE). **What could be the origin of the short-living fluorescence?**

11/13/2015



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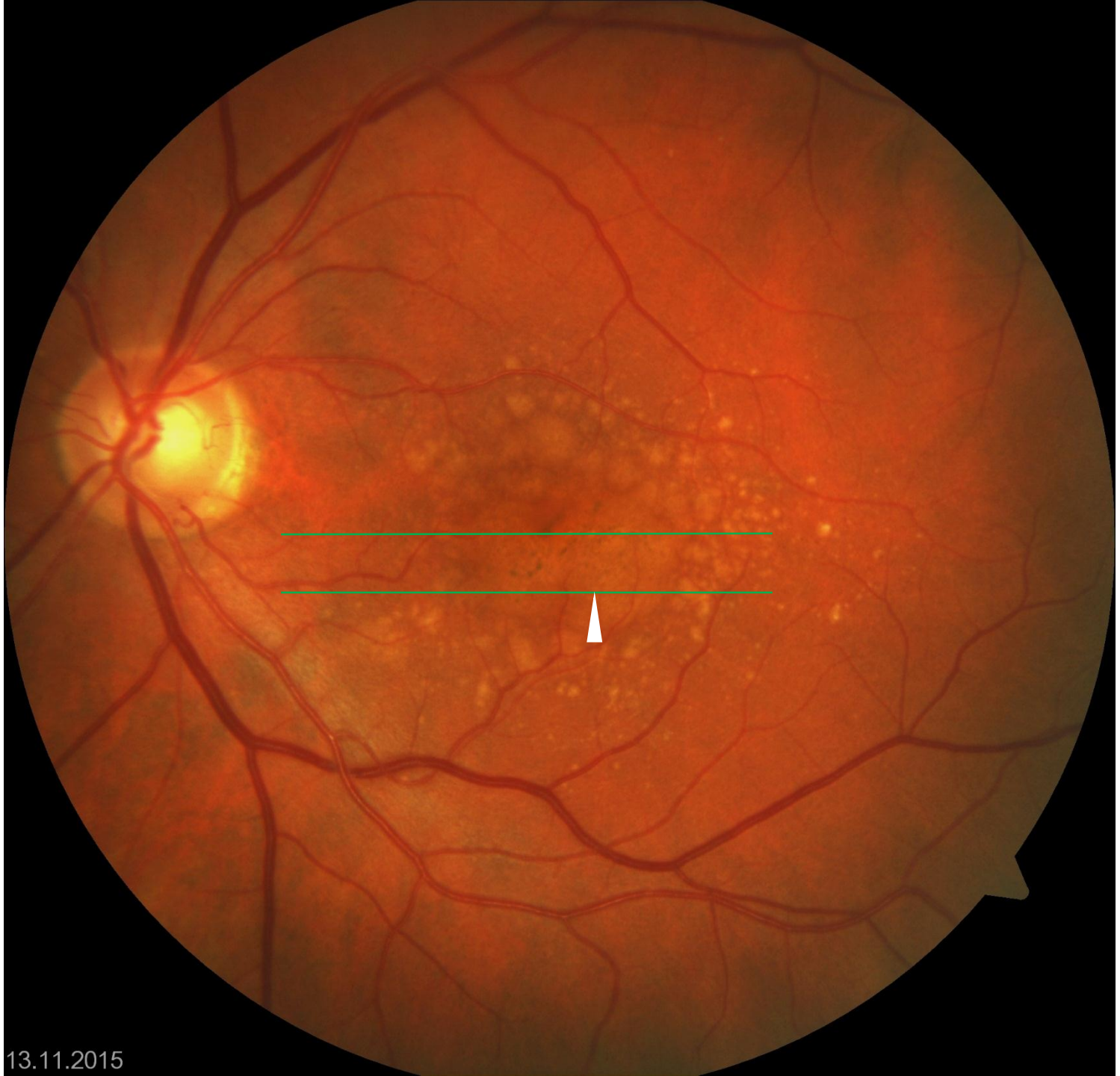

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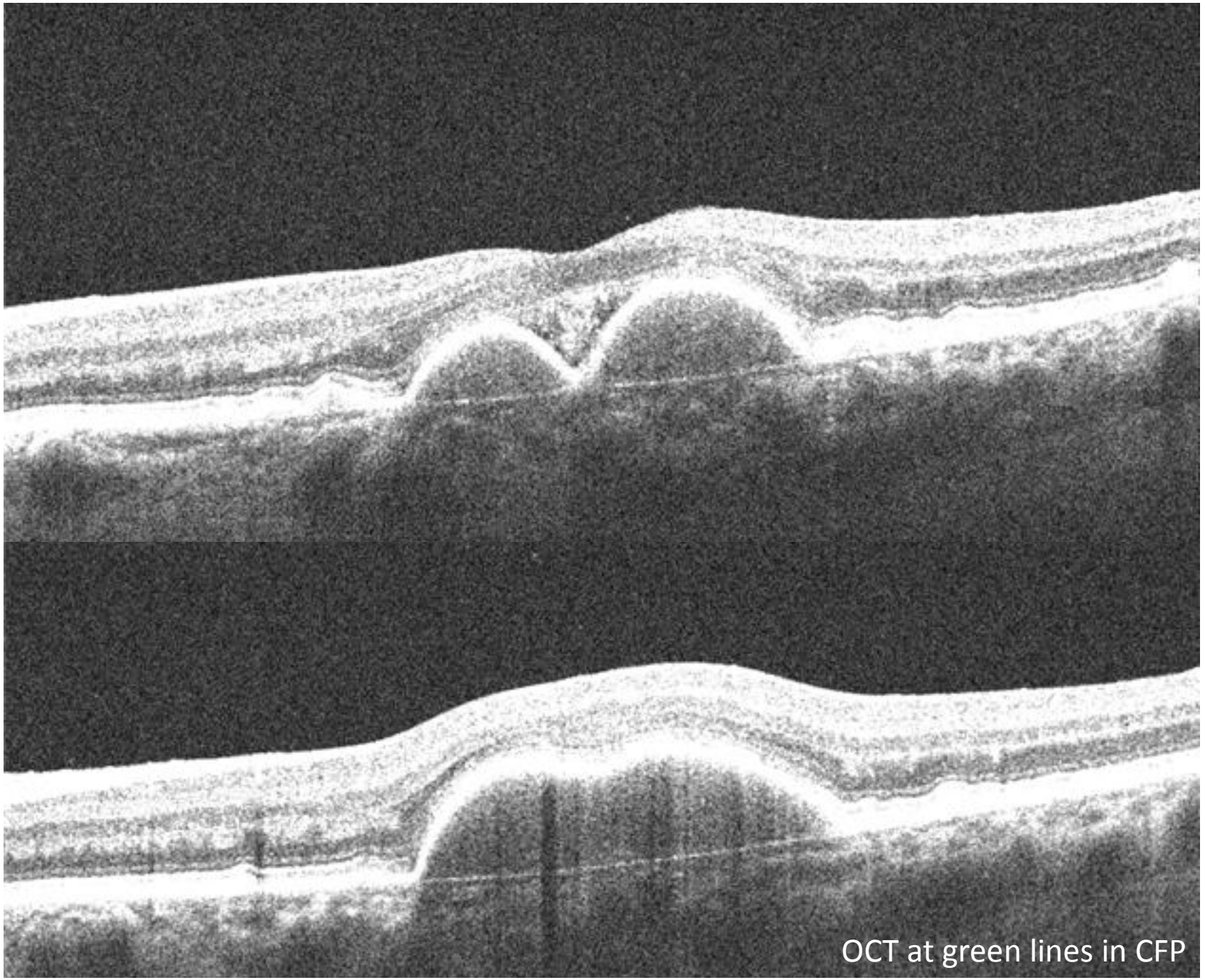

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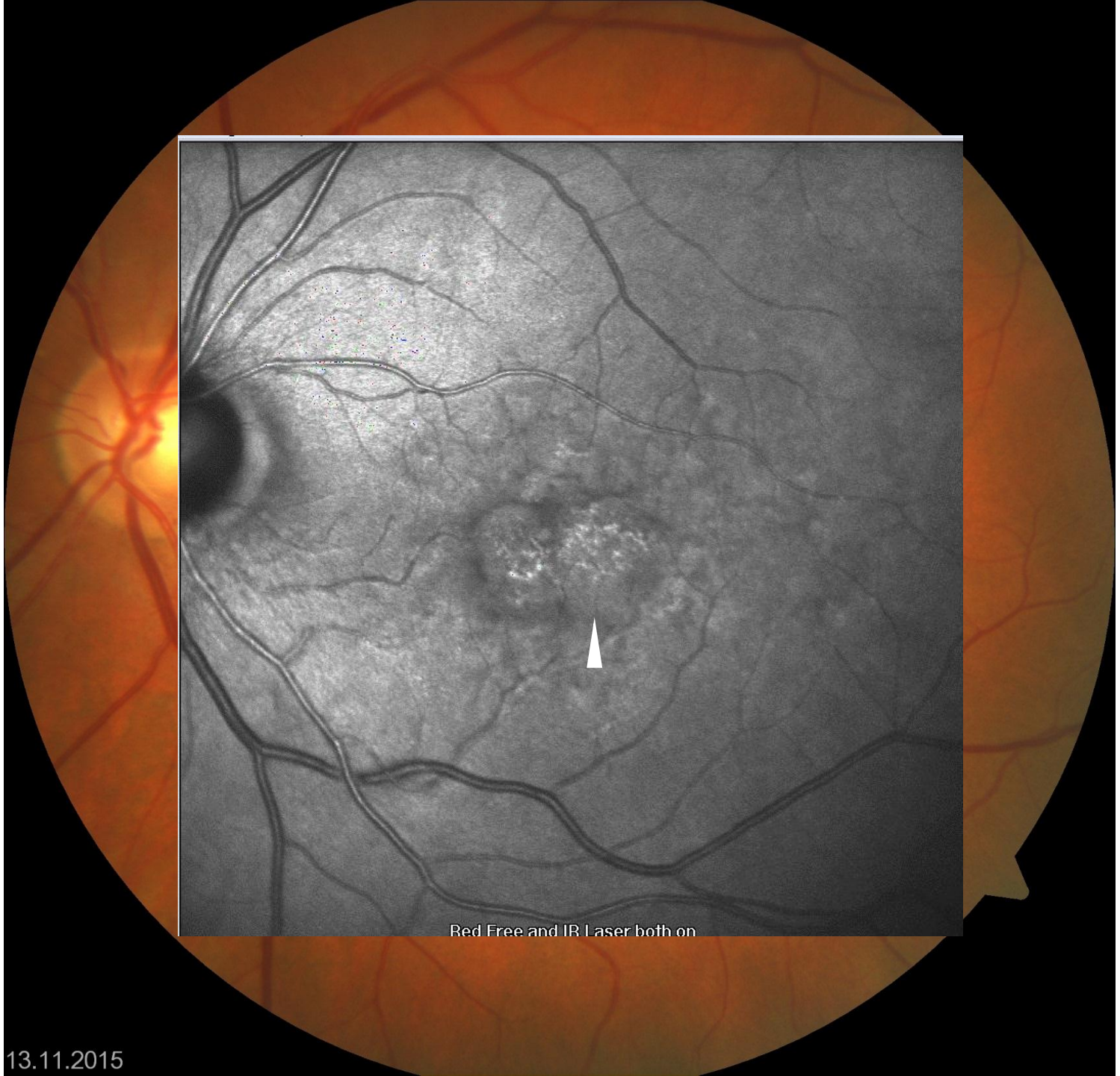

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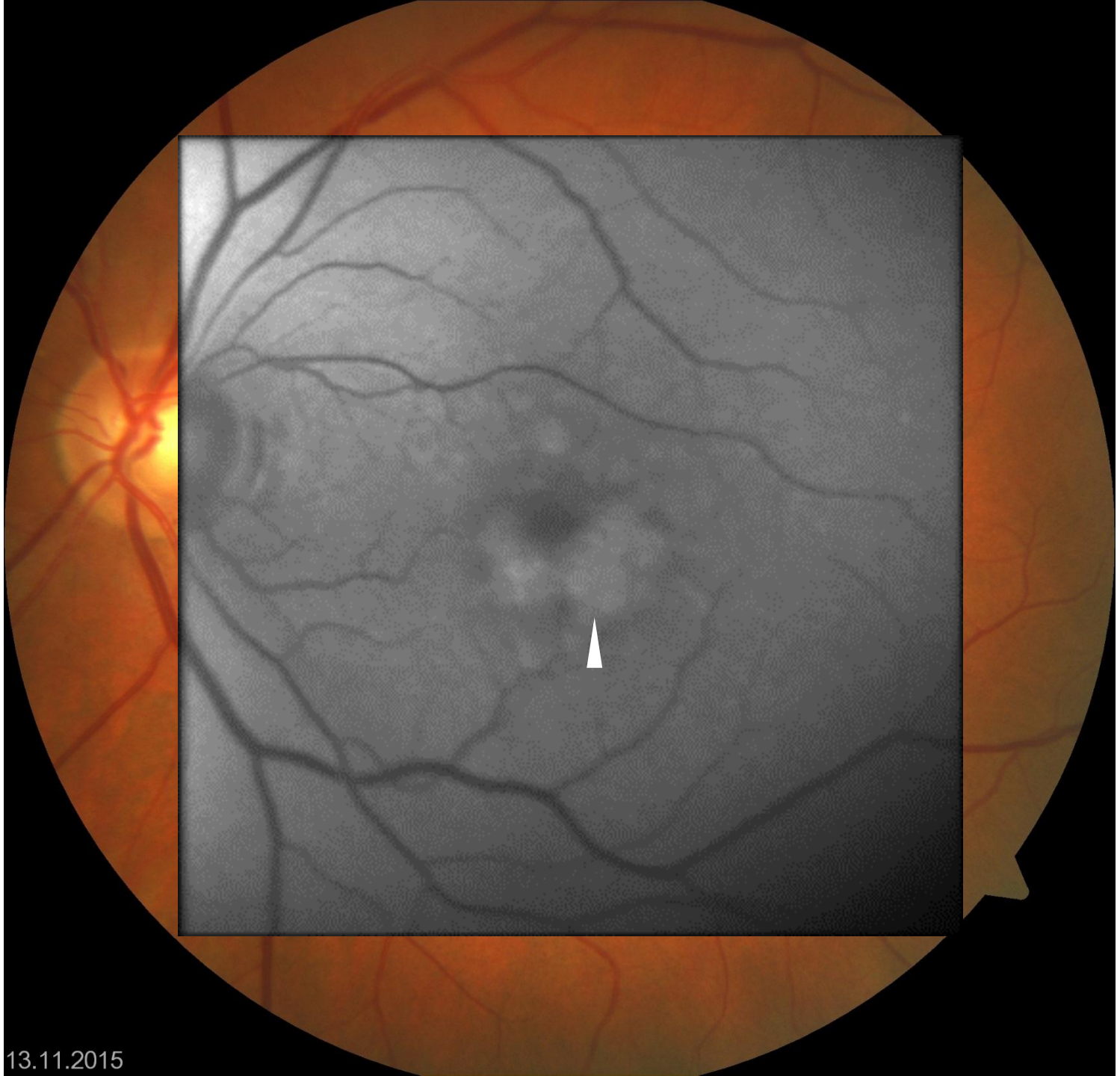

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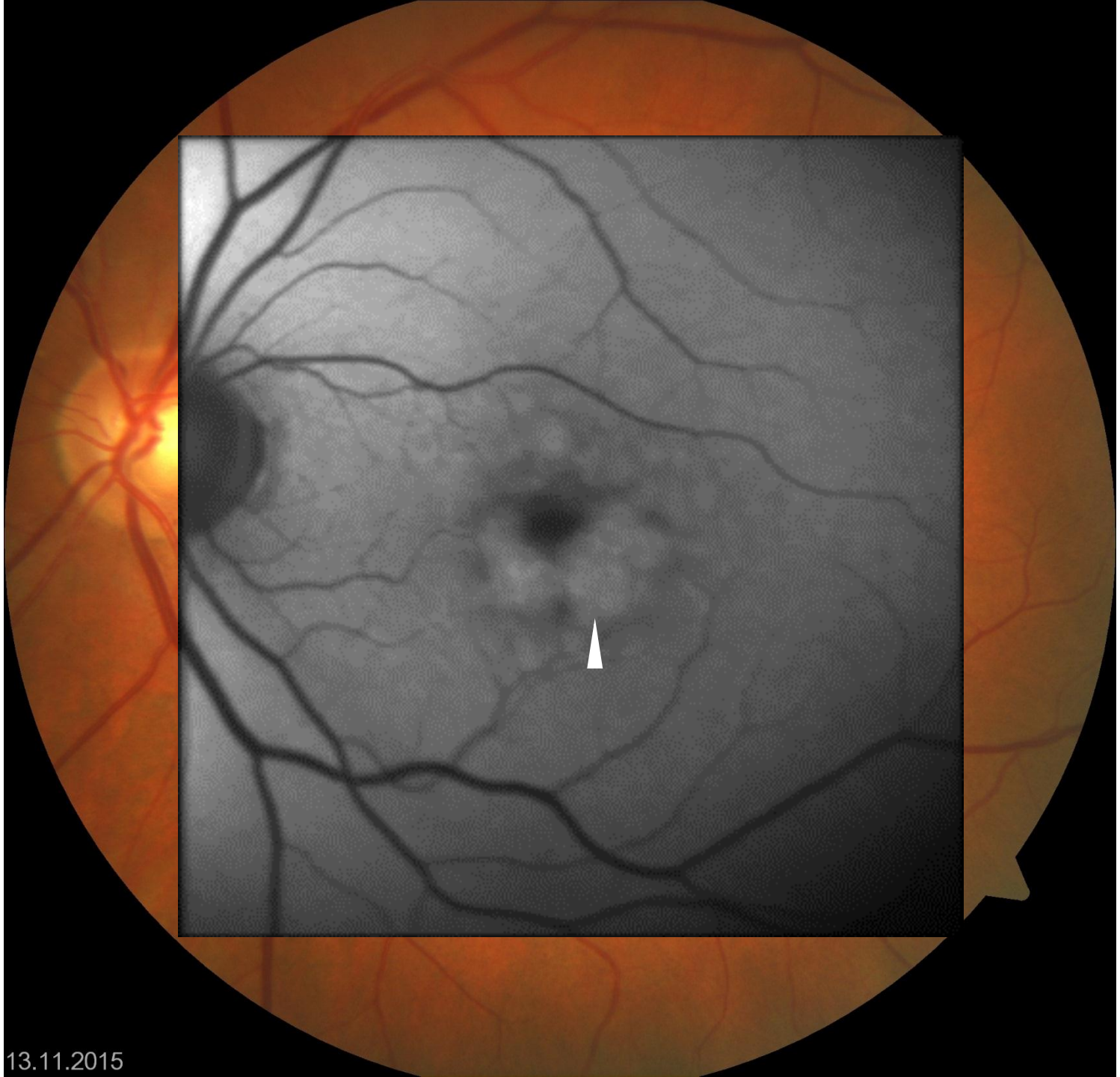

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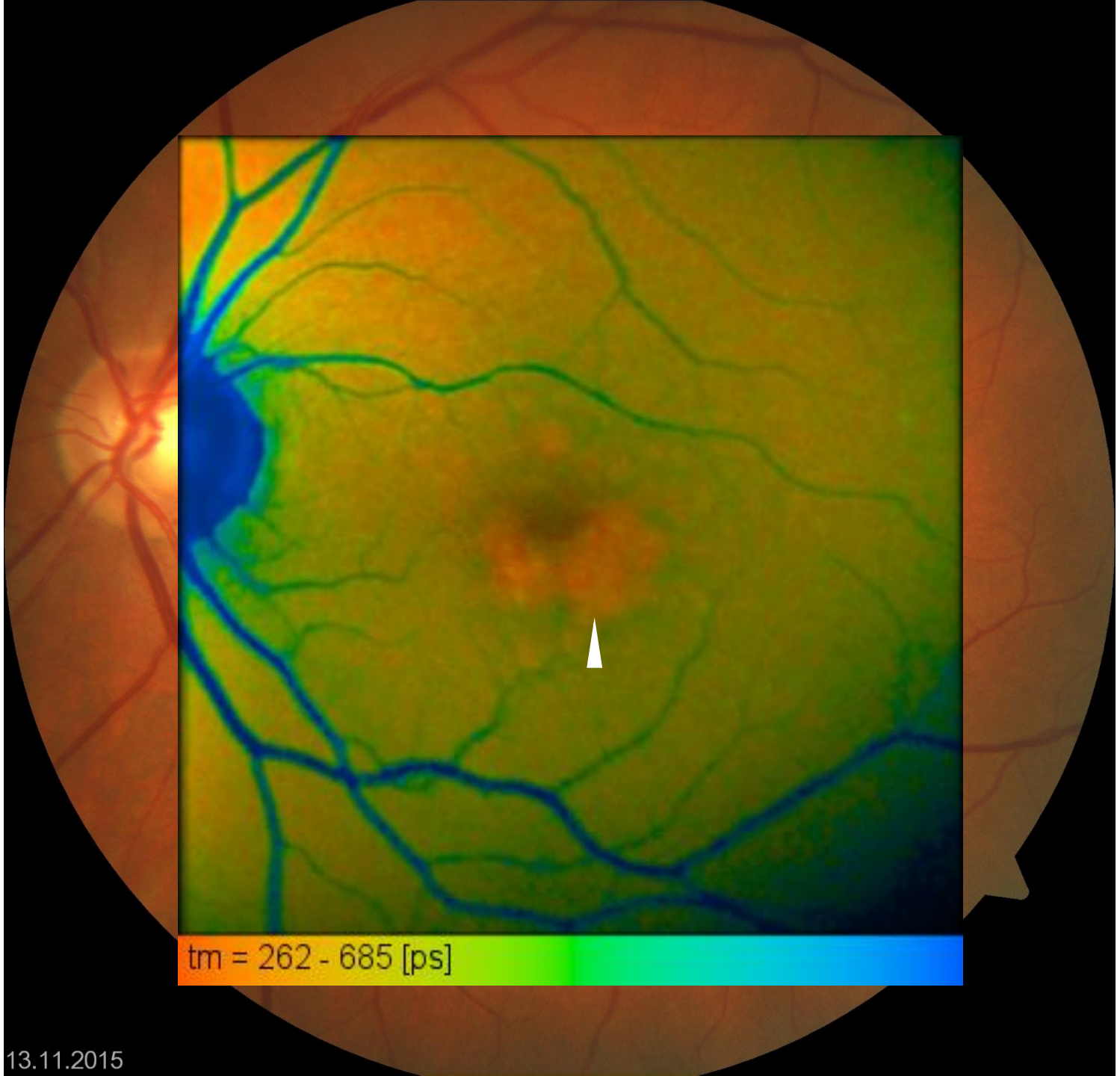

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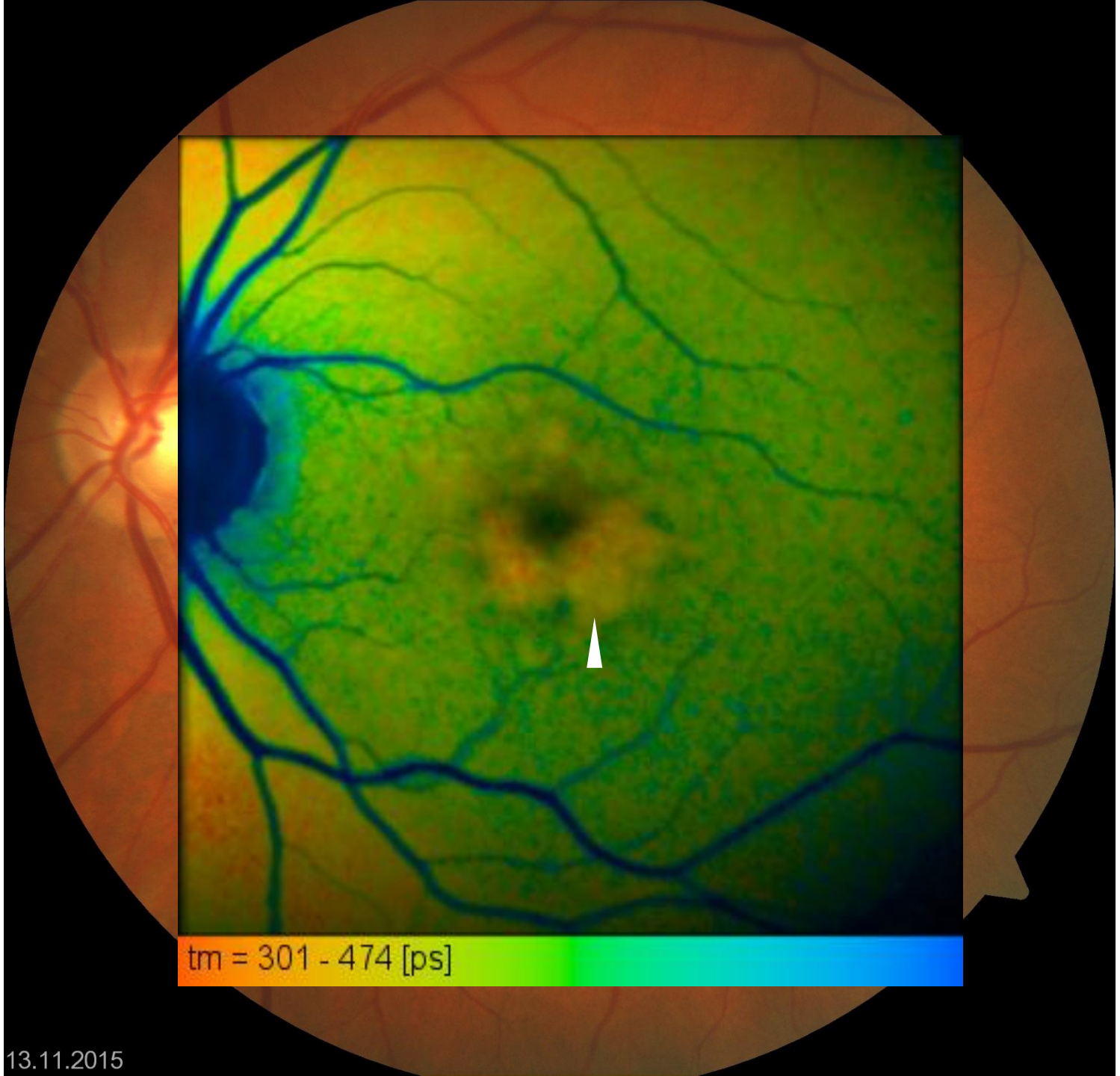
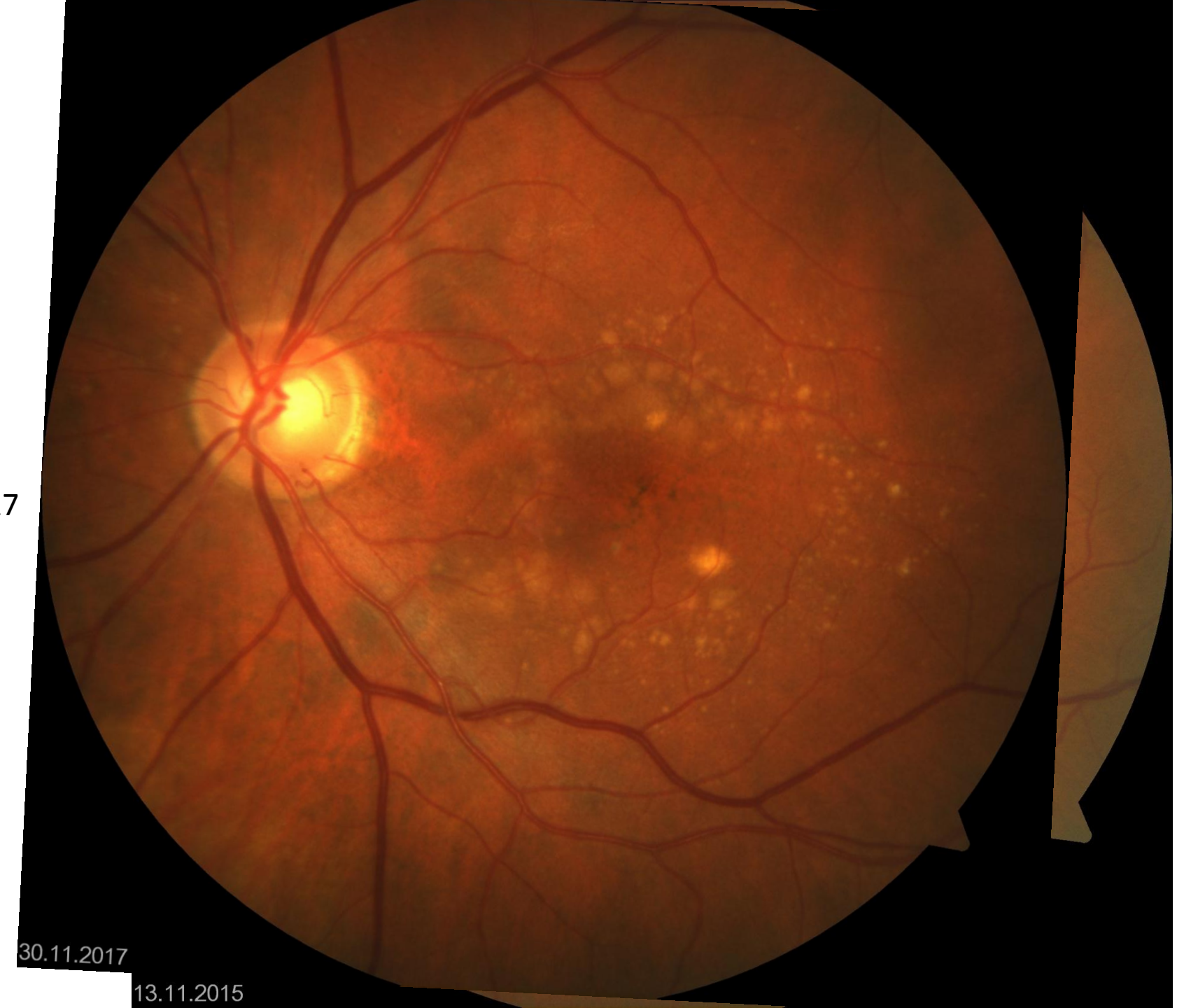


FLIO LSC

11/30/2017



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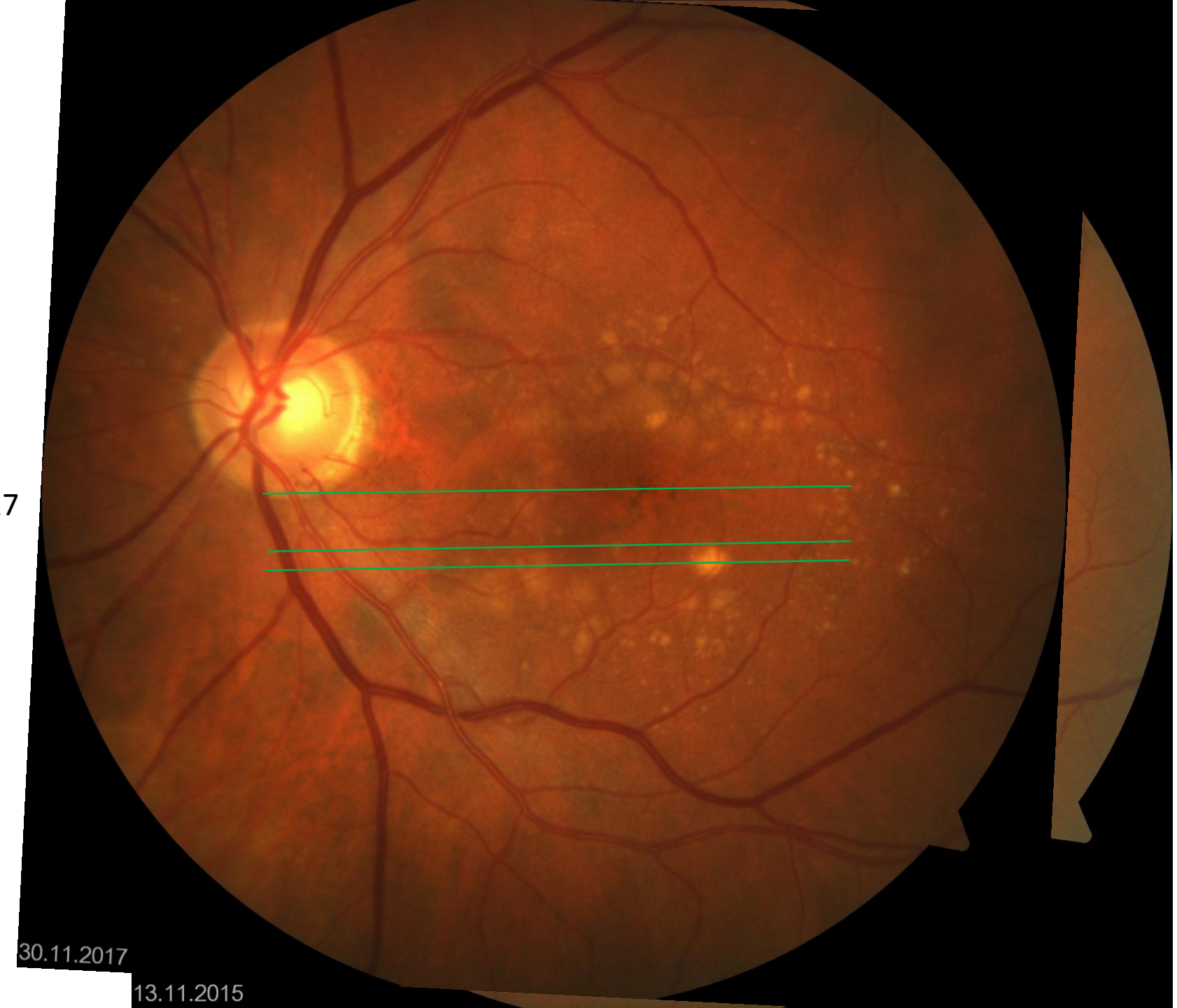

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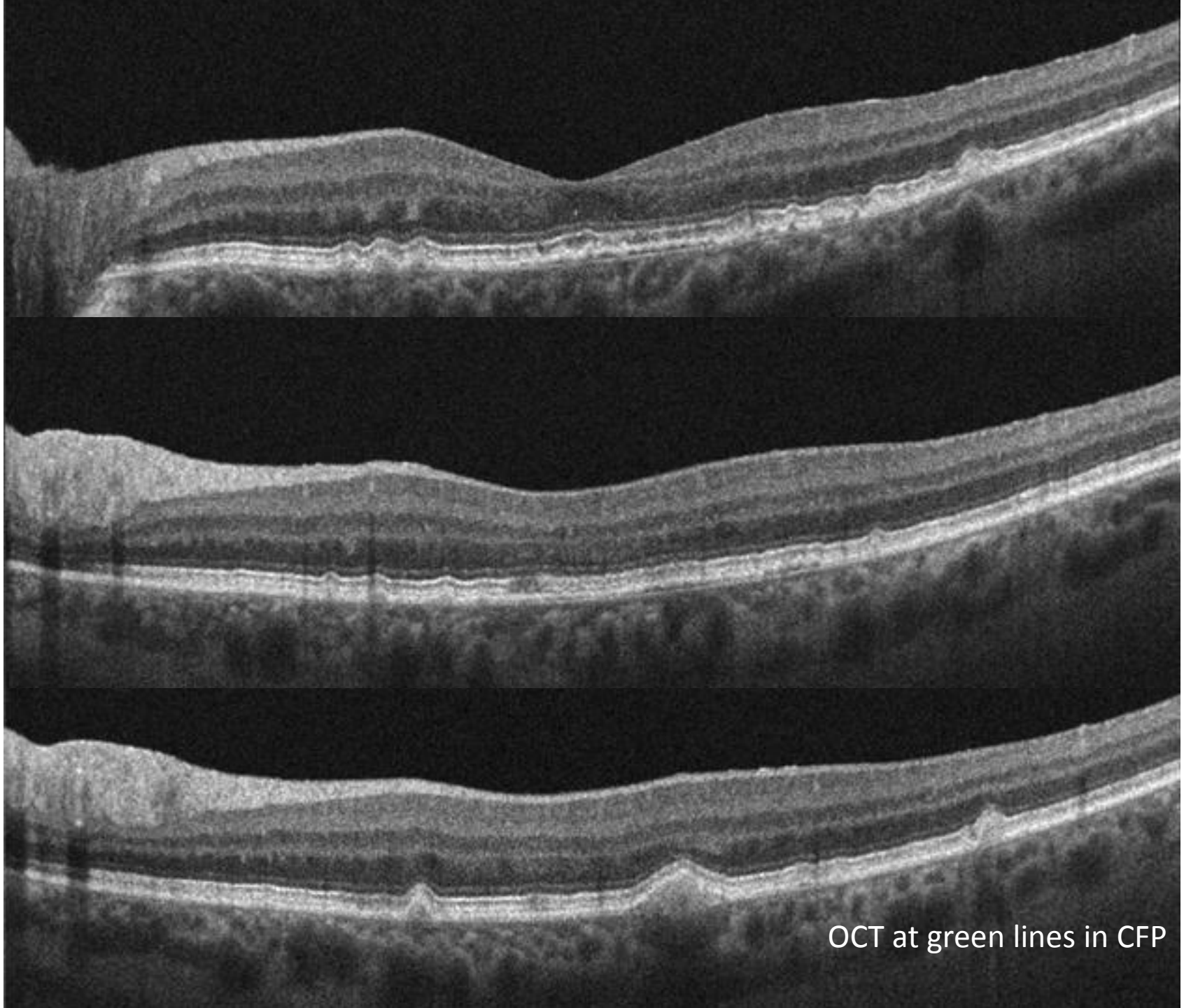
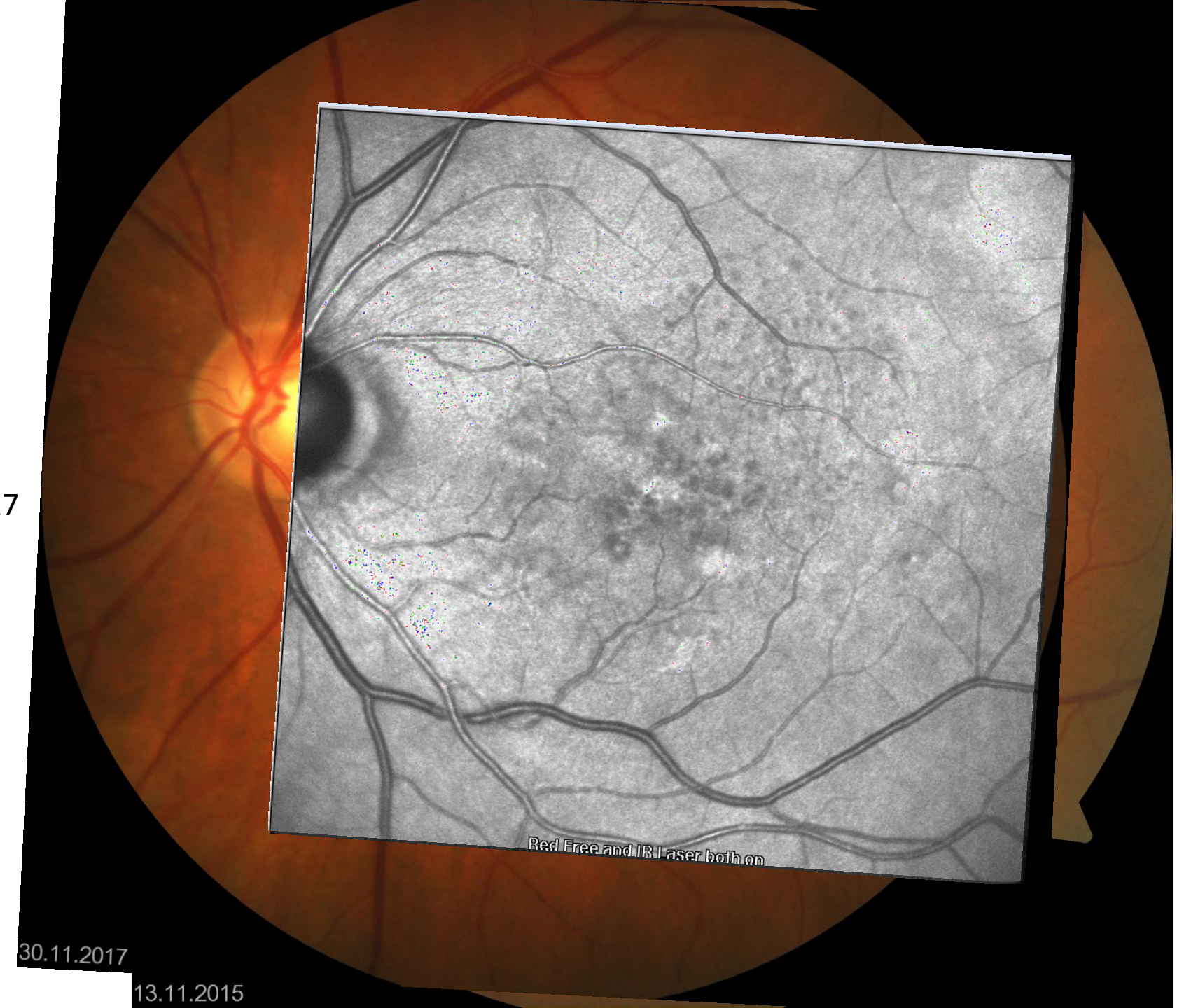


OCT at green lines in CFP

11/30/2017



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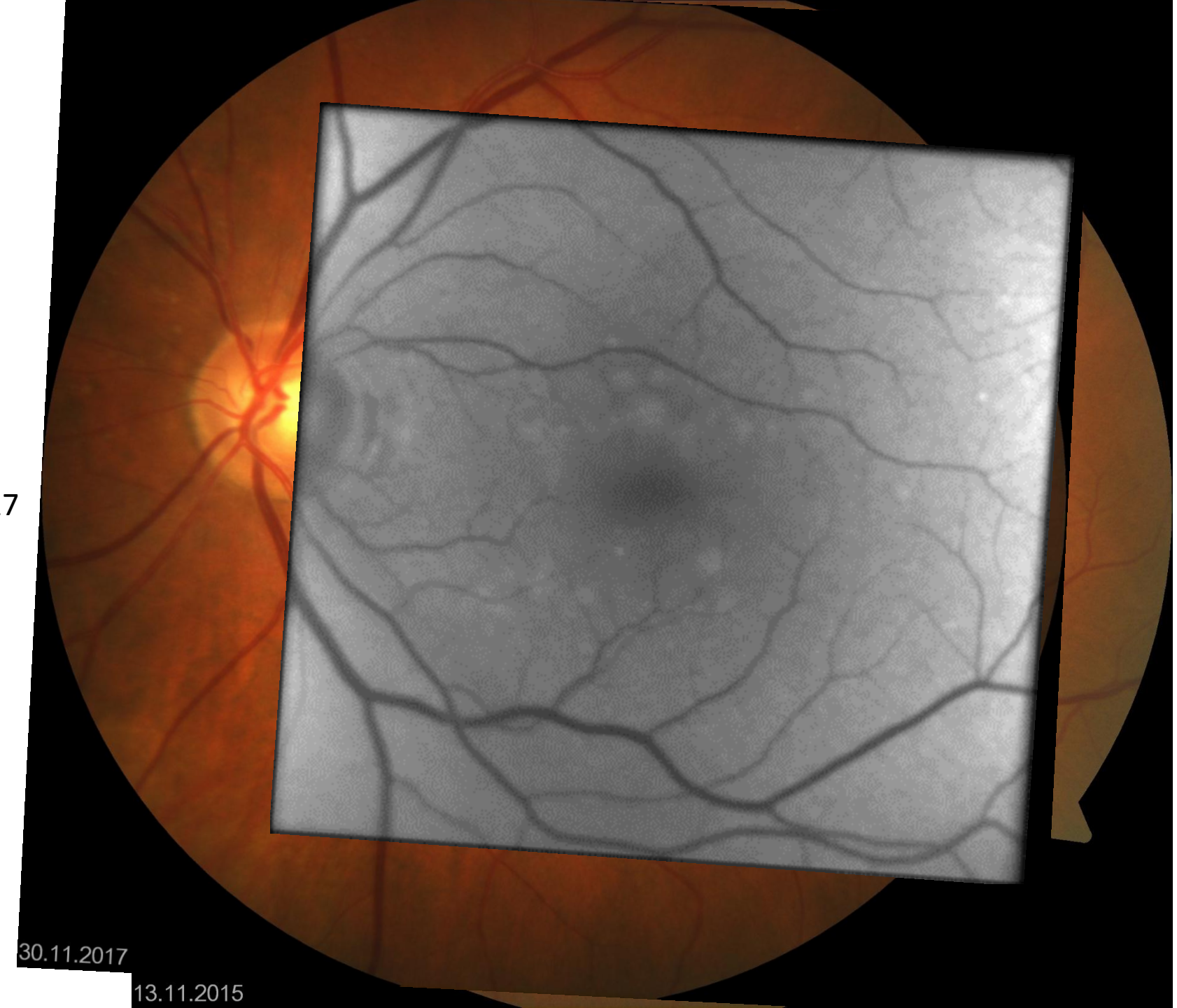

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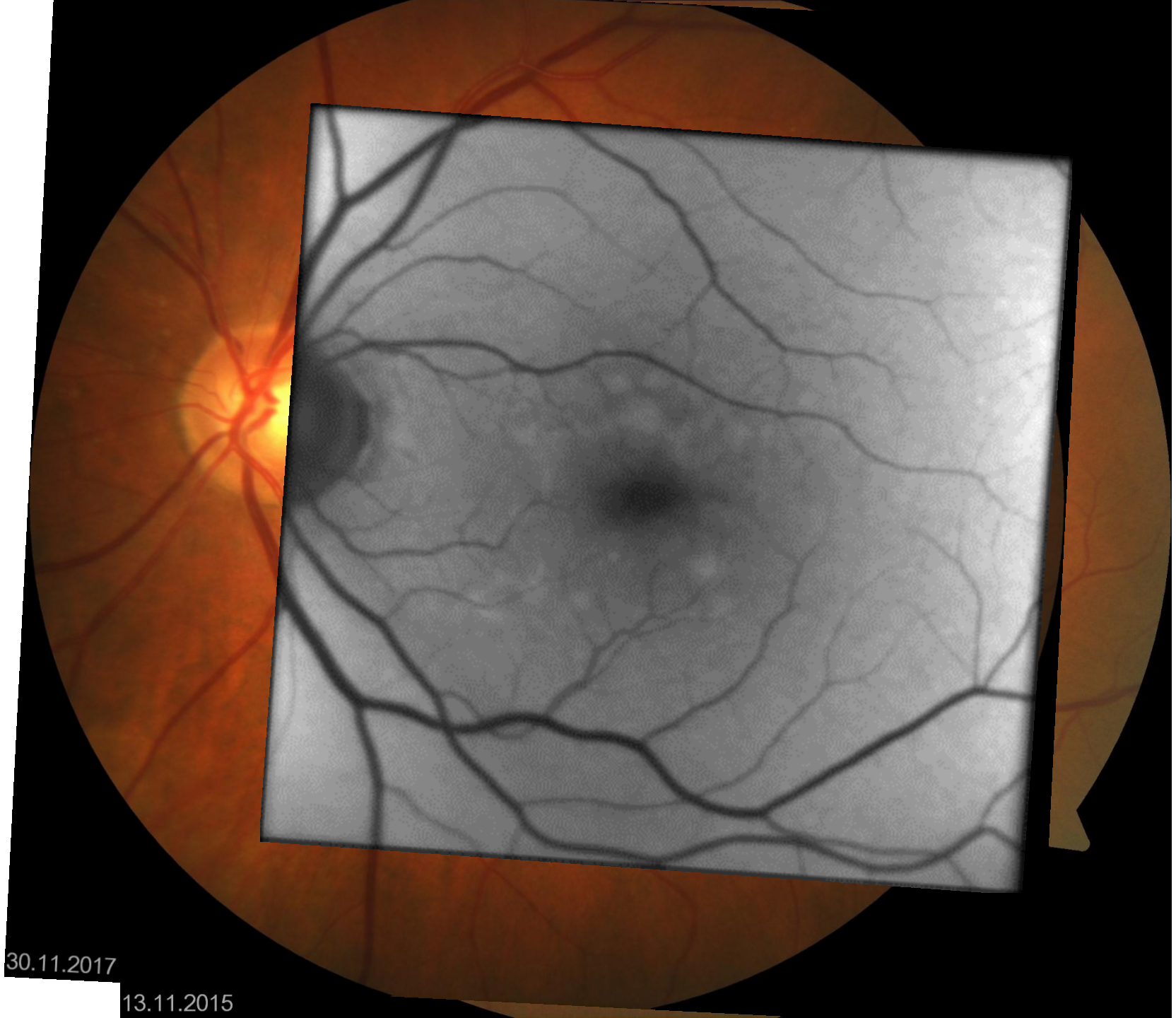

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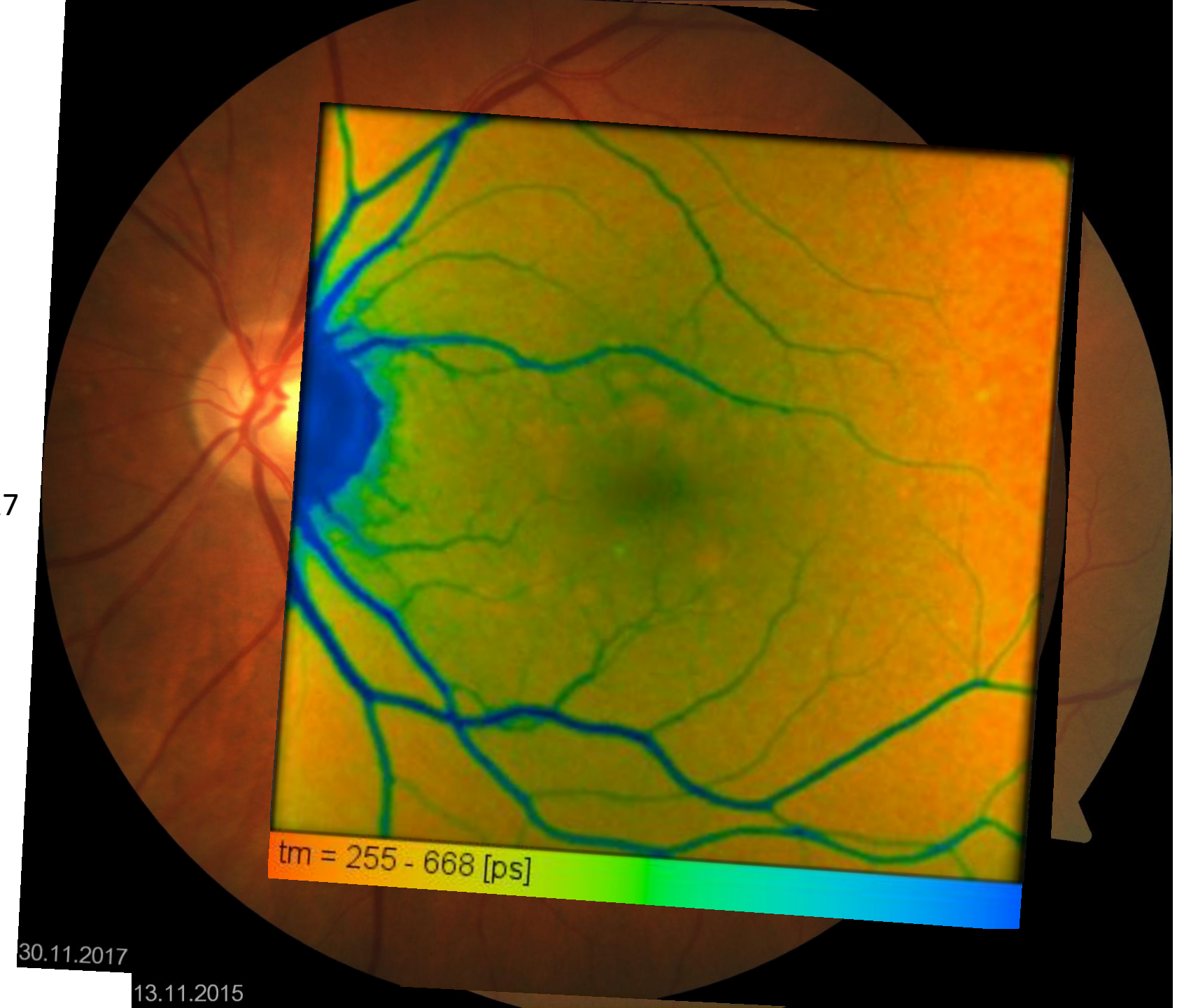

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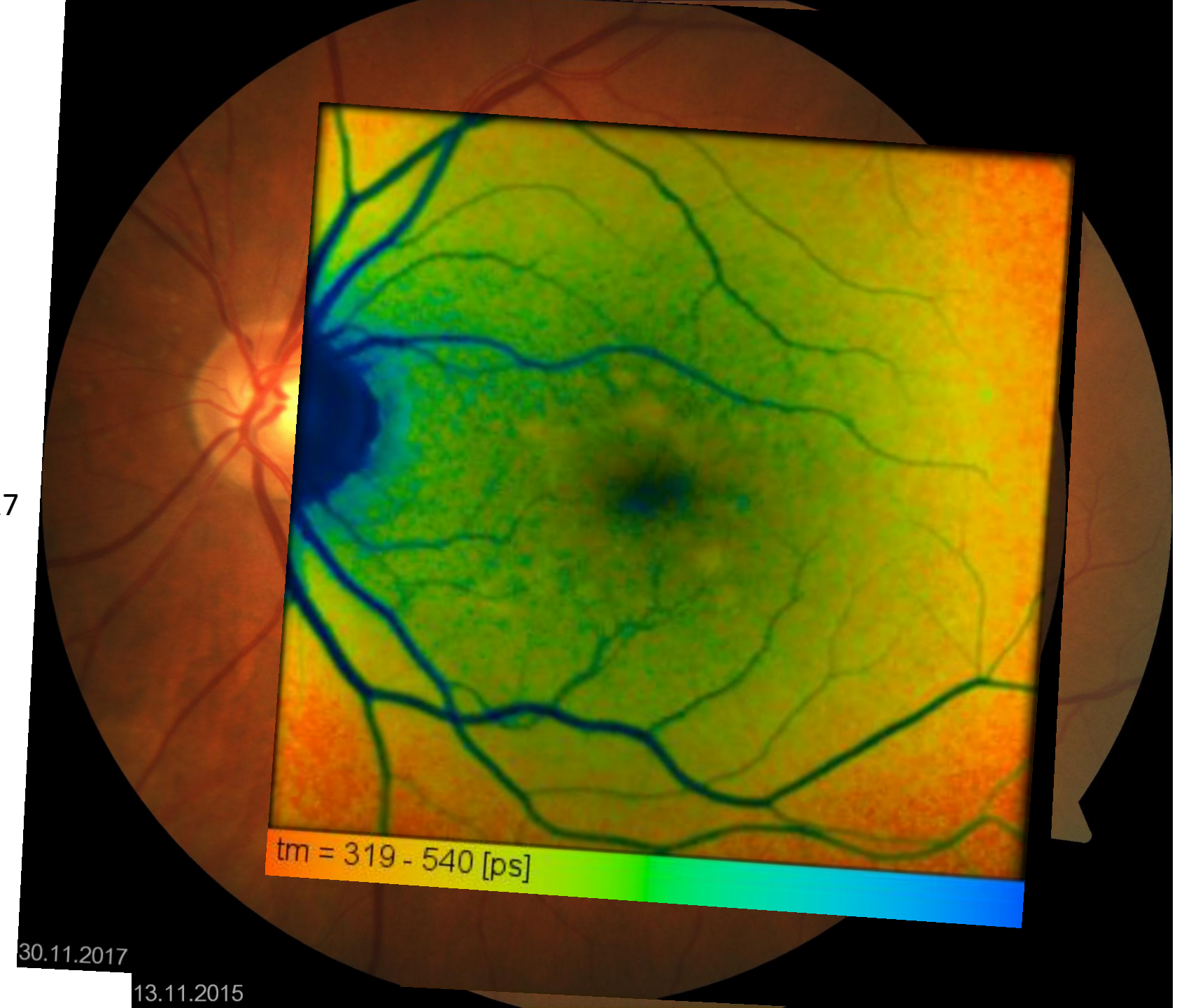
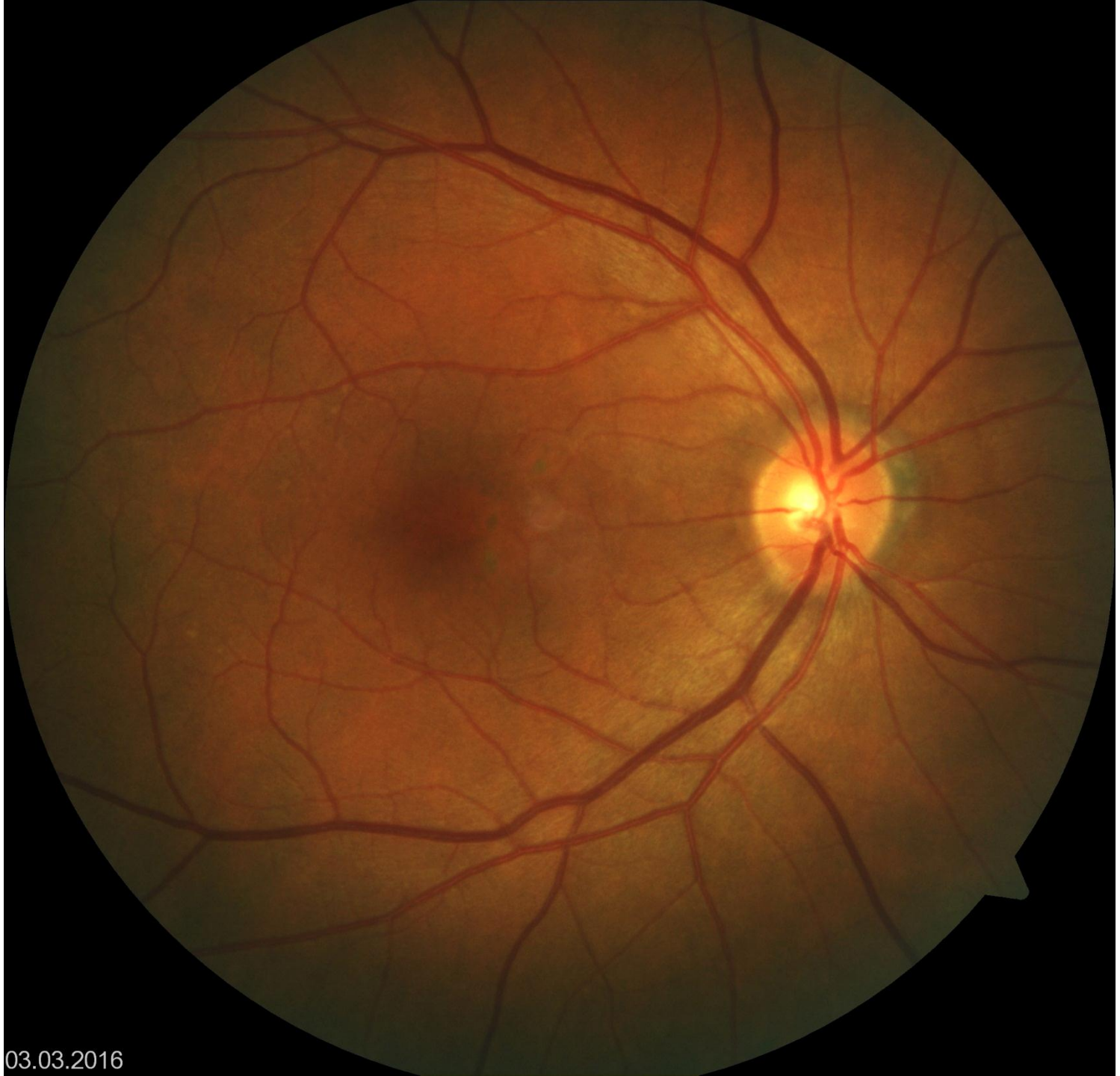


FLIO LSC

AMD_24, 74 years at baseline

The arrowheads show hyperfluorescent structures. Whereas the purple arrowhead points to a hyperpigmentation, this is not completely clear for the other ones although CFP shows hyporeflectivity. OCT shows a druse at the black arrowhead and breakthrough of EZ and outer limiting membrane by RPE/sub-RPE structures. Whereas FLIO is blurred in SSC due to incipient cataract, LSC shows short lifetimes for the druse(n) (Black and white arrowheads), but long ones for the hyperpigmentation (purple arrowhead). During follow up (less than 2 years), migrated RPE (purple arrowhead) disappeared and so did the hyperfluorescence. At black and white arrowhead OCT showed a disruption of the EZ and migration of RPE, However, lifetimes remained short at the white arrowhead, but got long at the black one.
Any suggestions welcome!

03/03/2016



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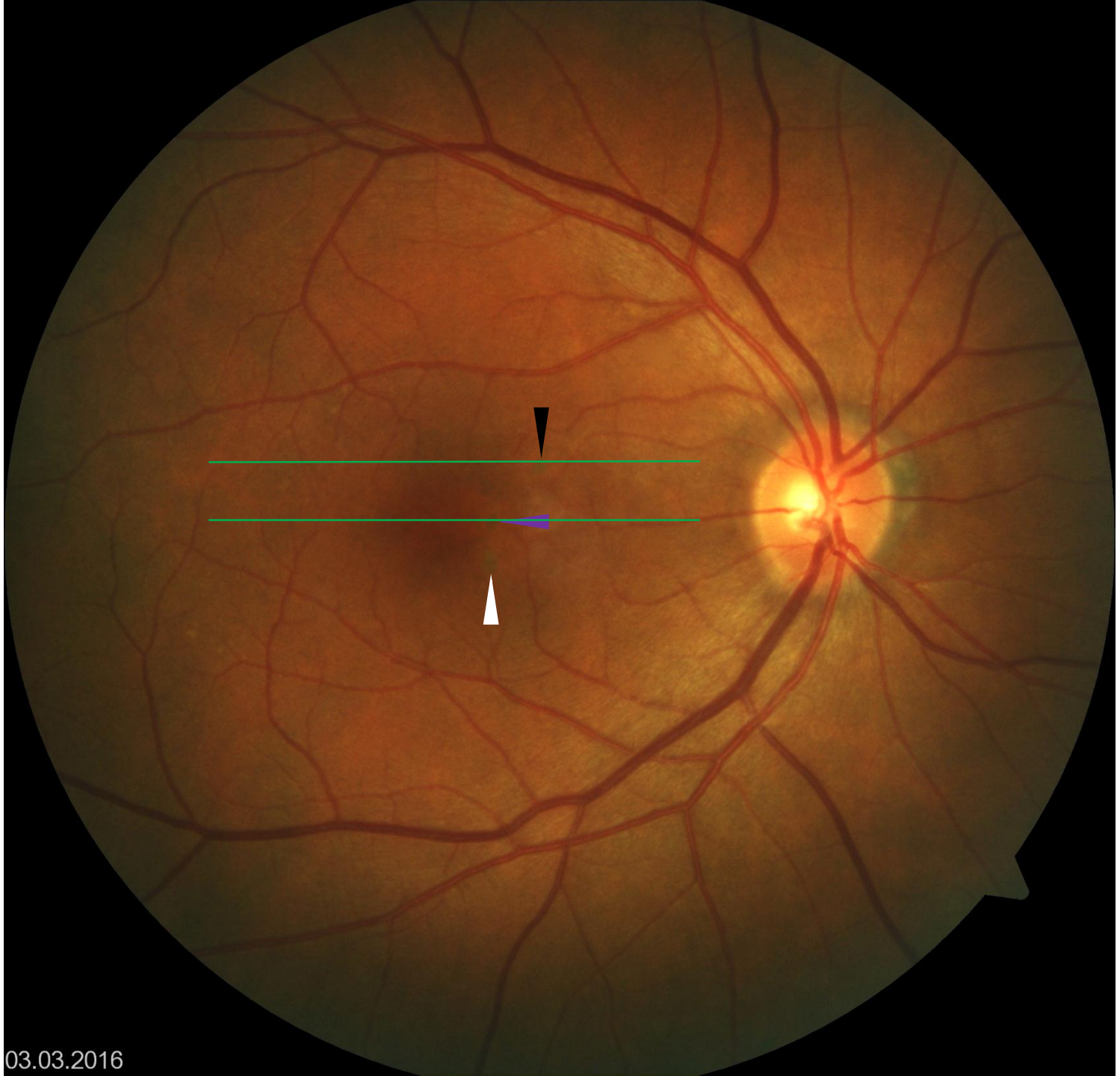

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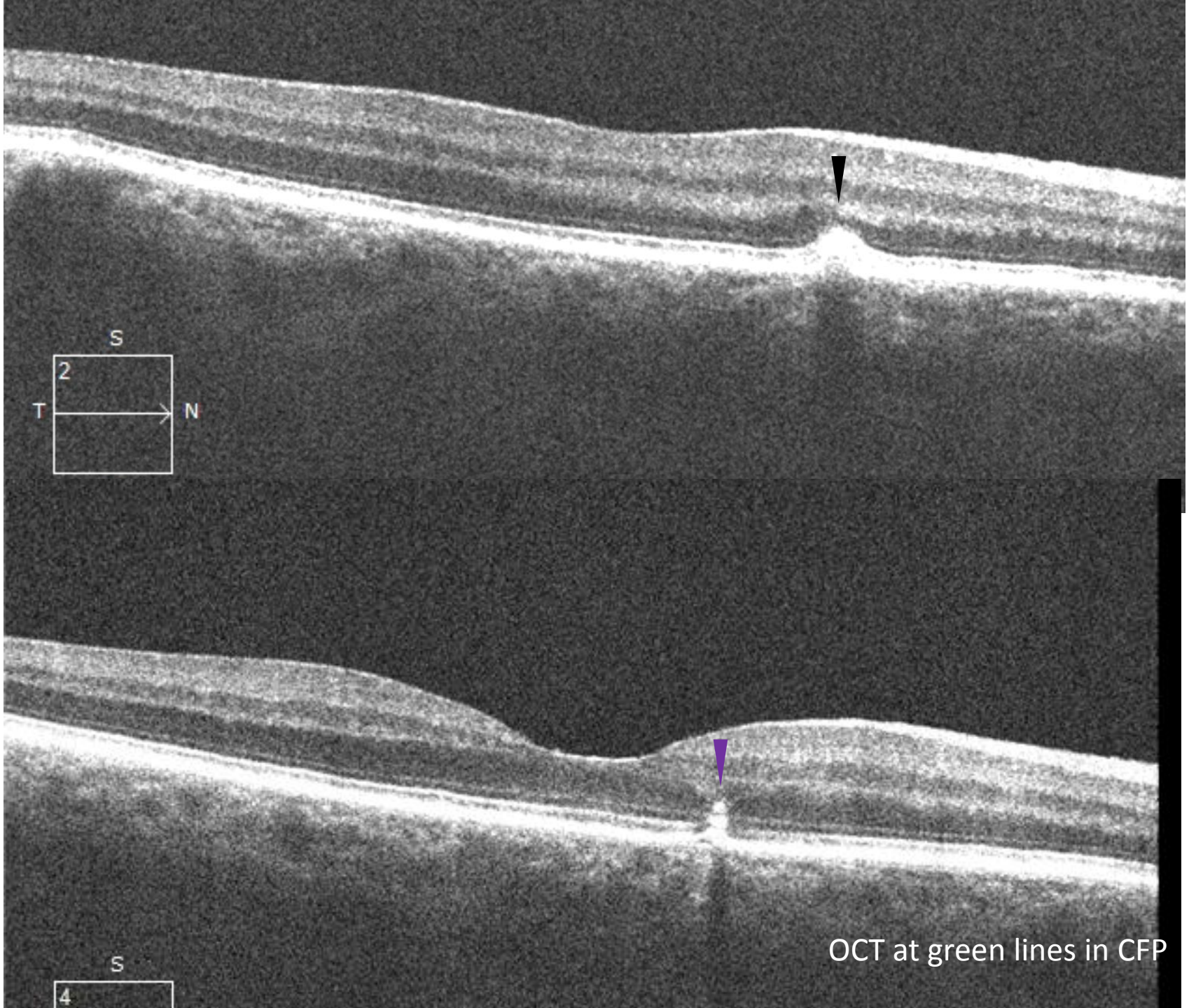

OCT at green lines in CFP

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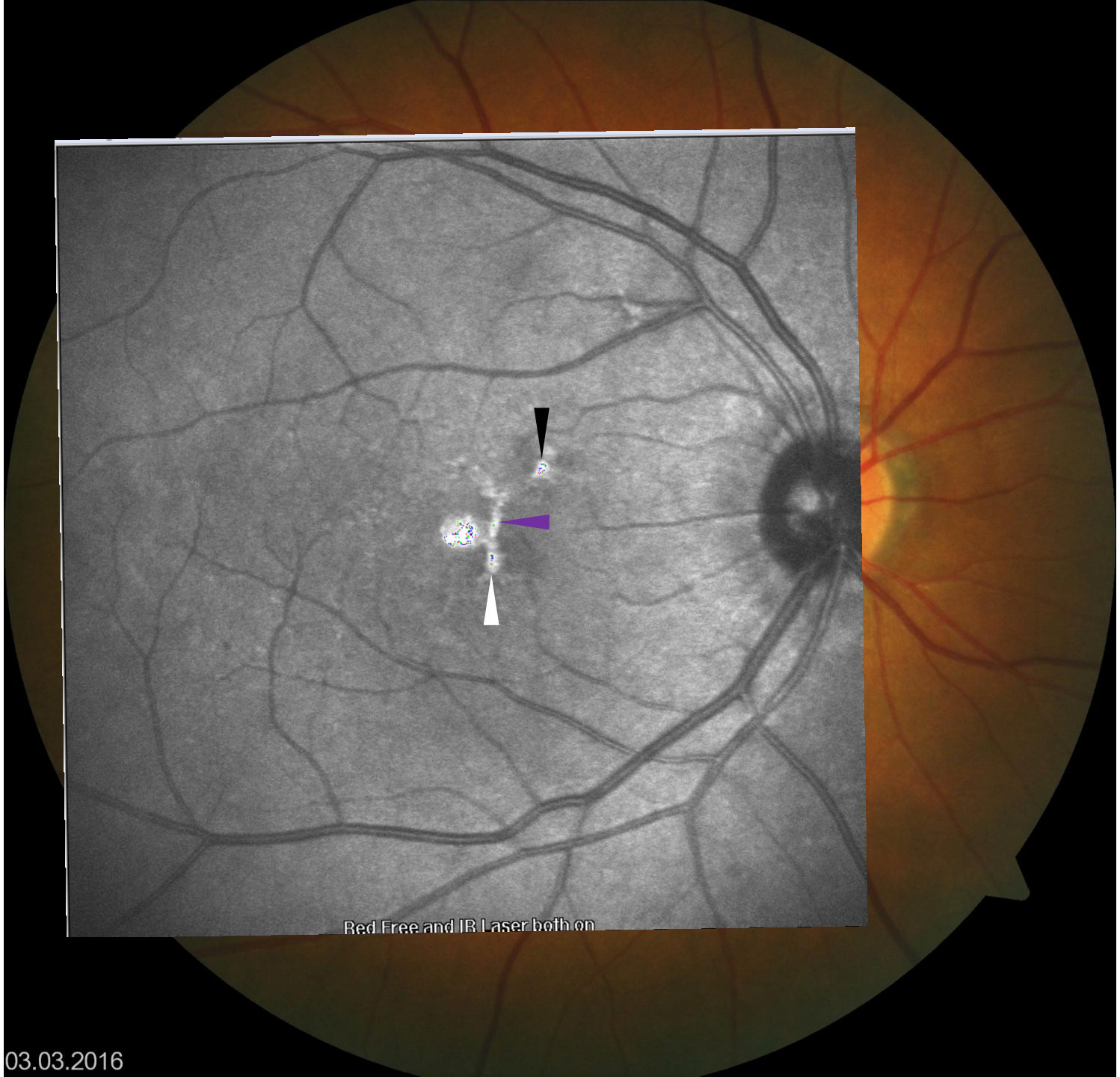

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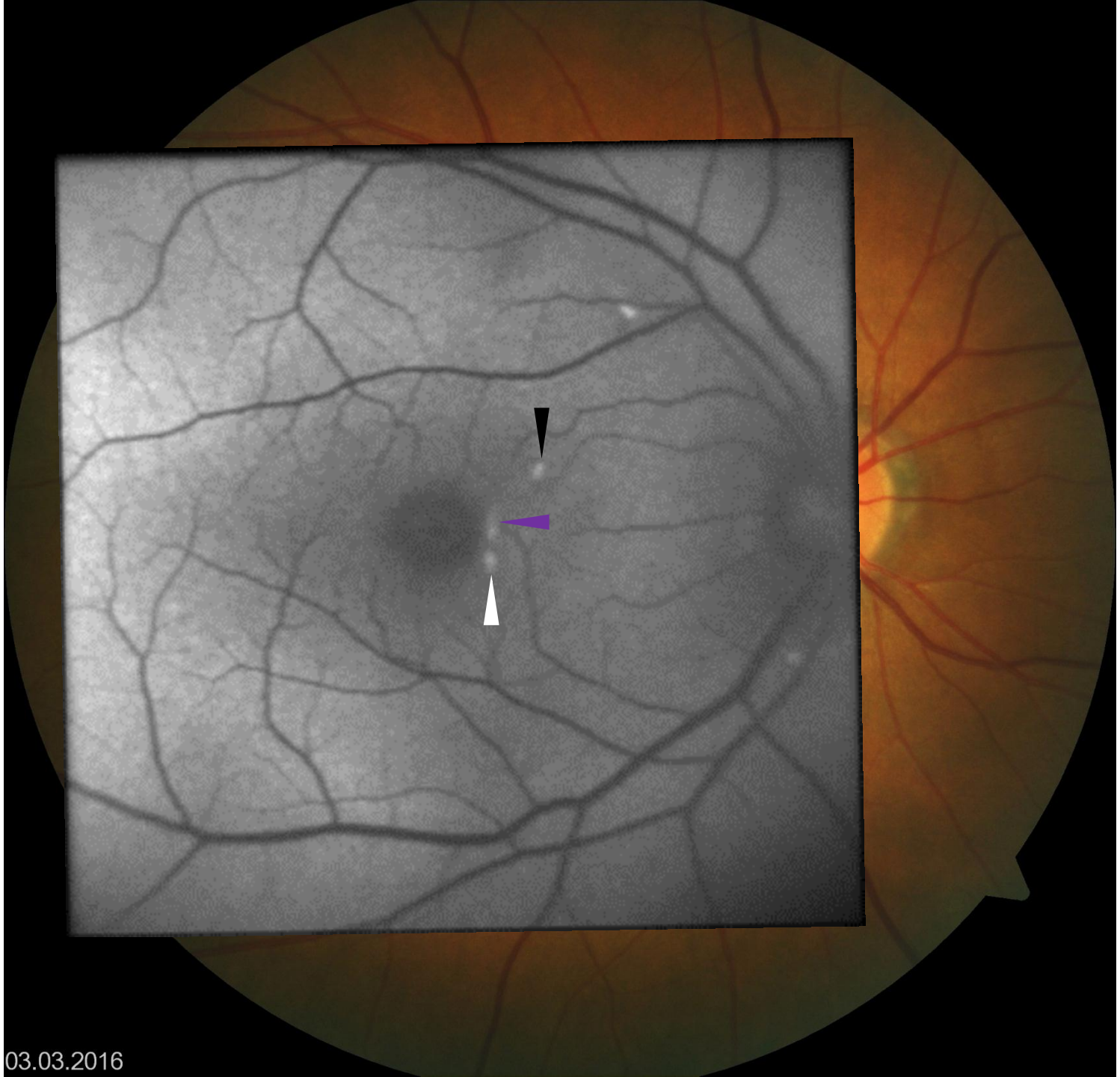

FAF SSC

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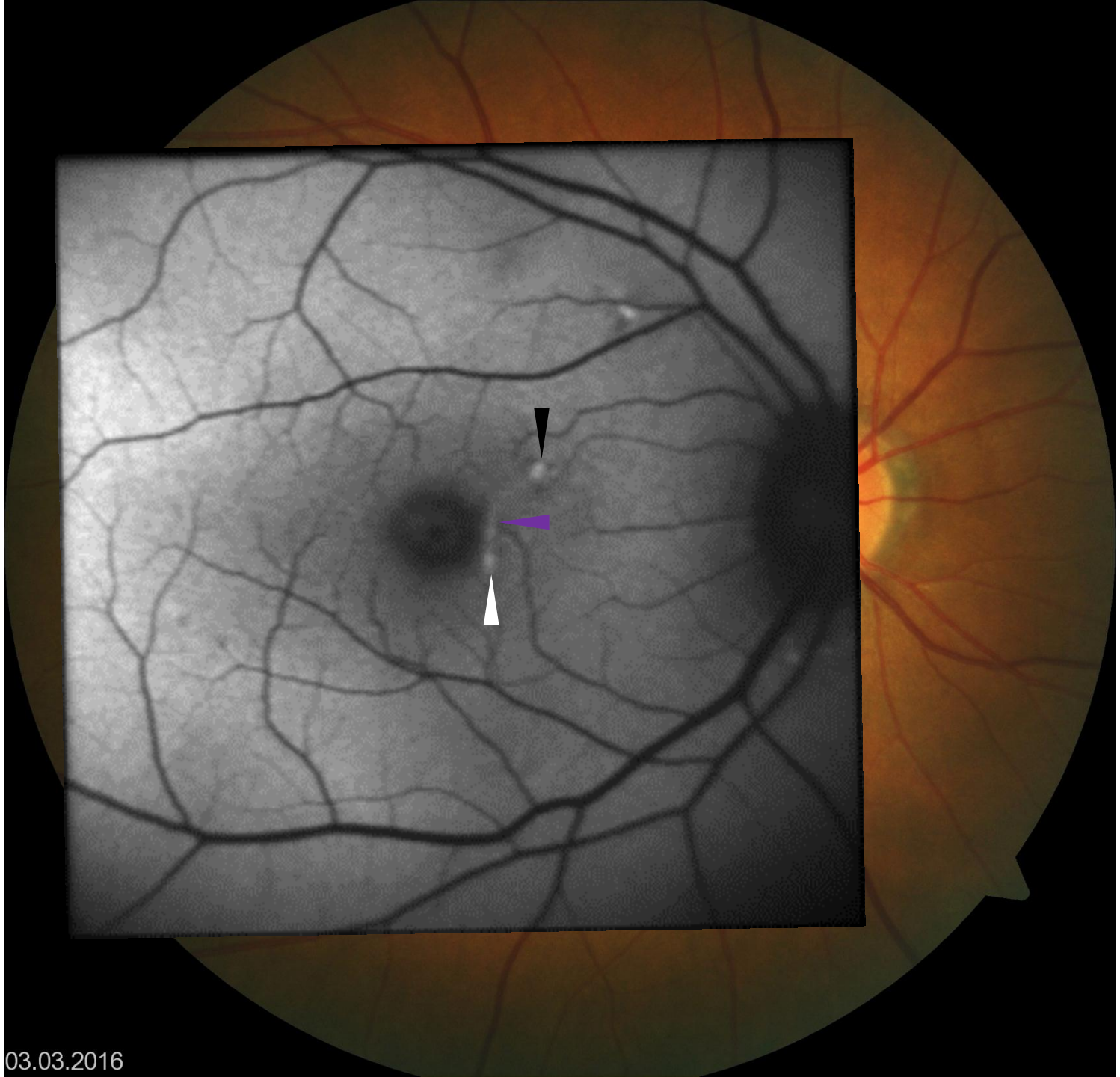

FAF LSC

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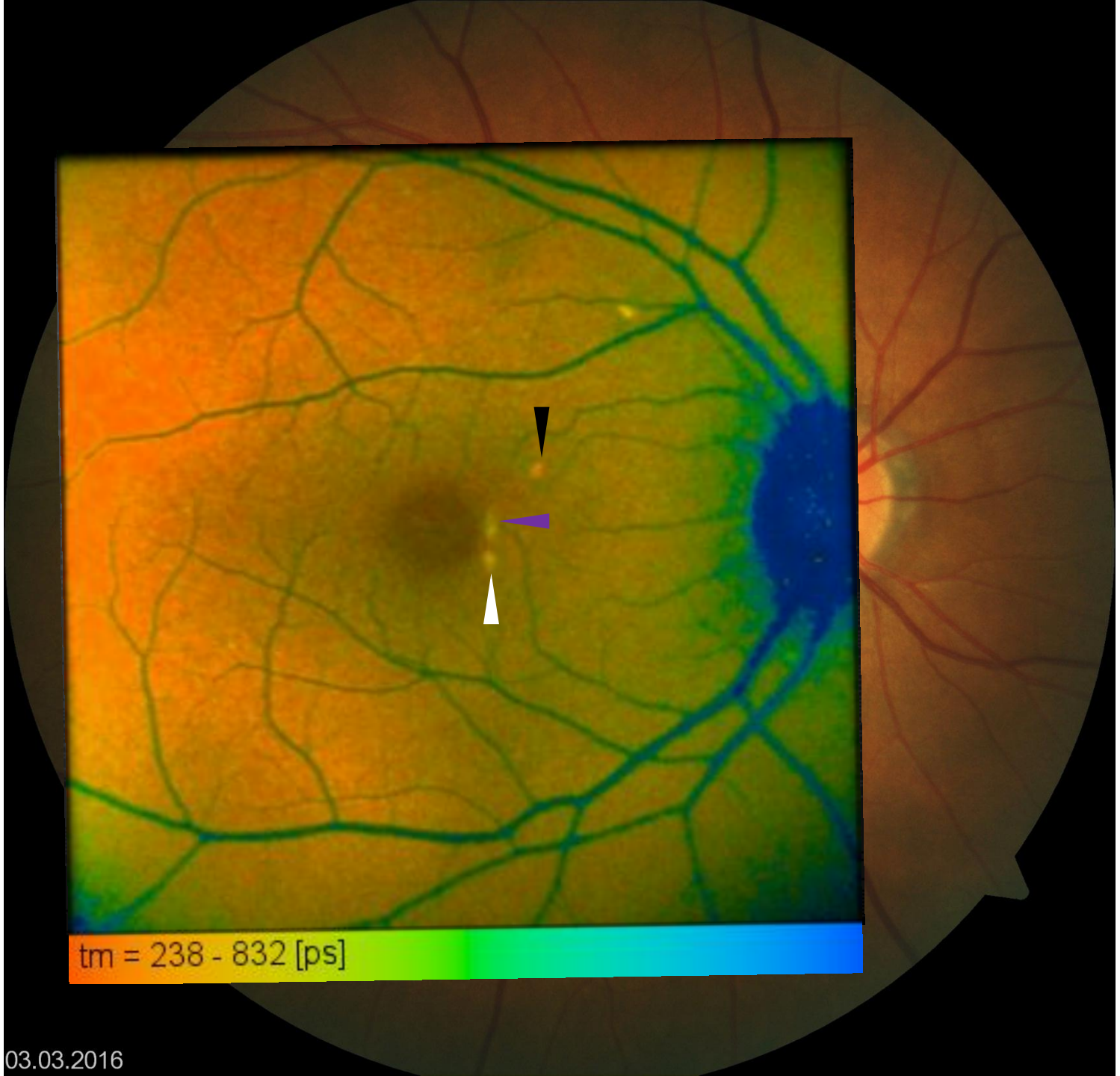

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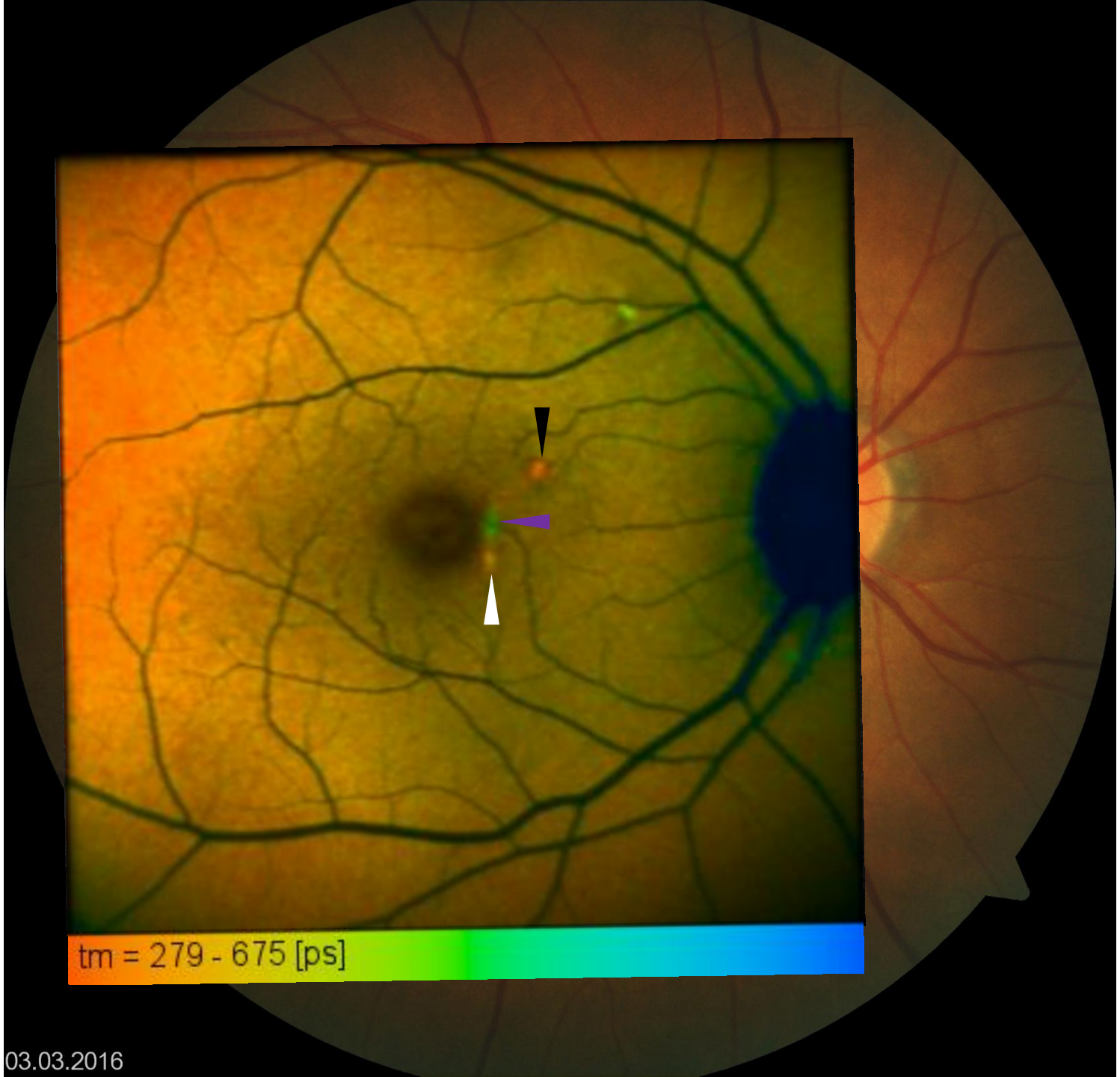

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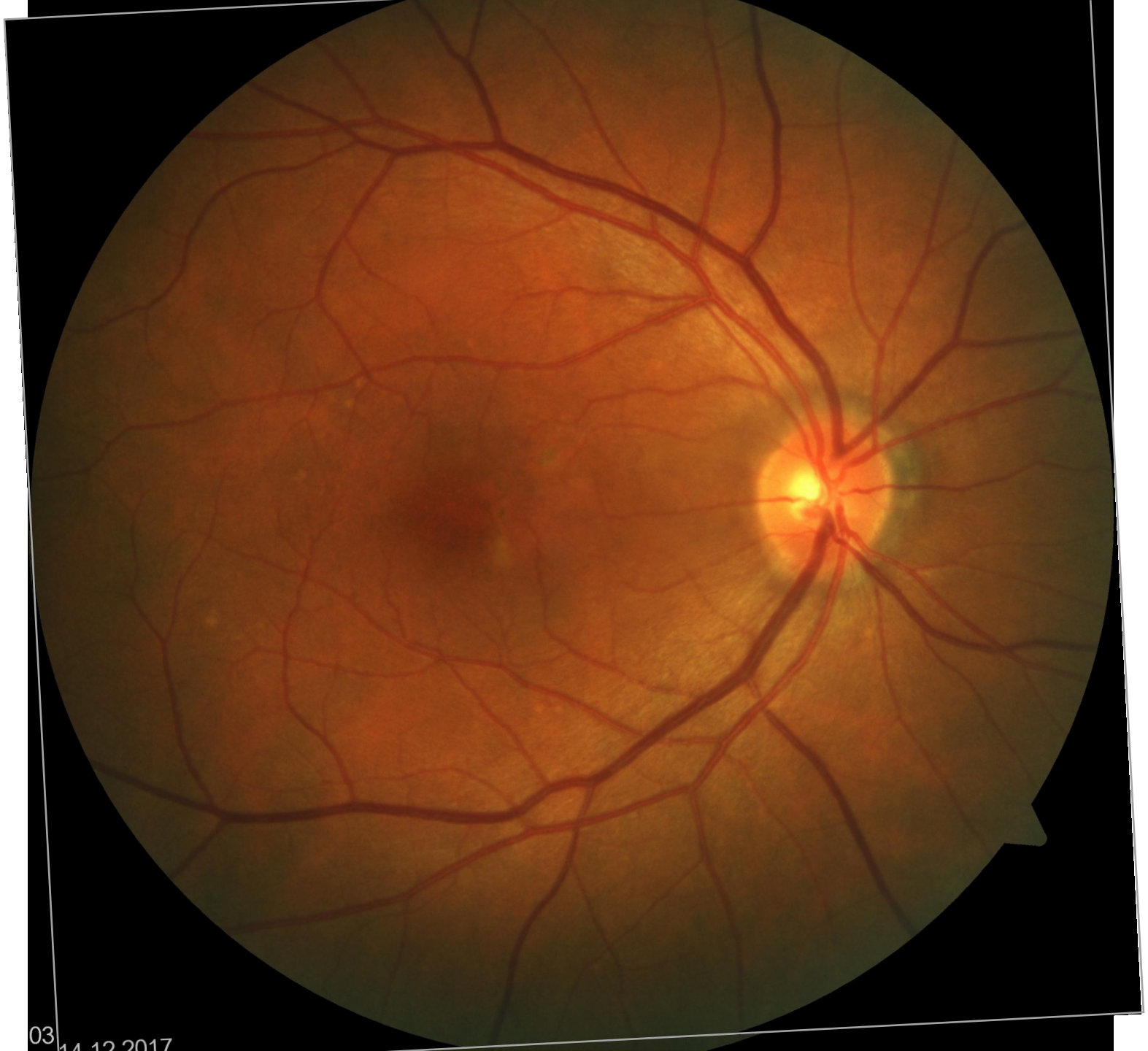

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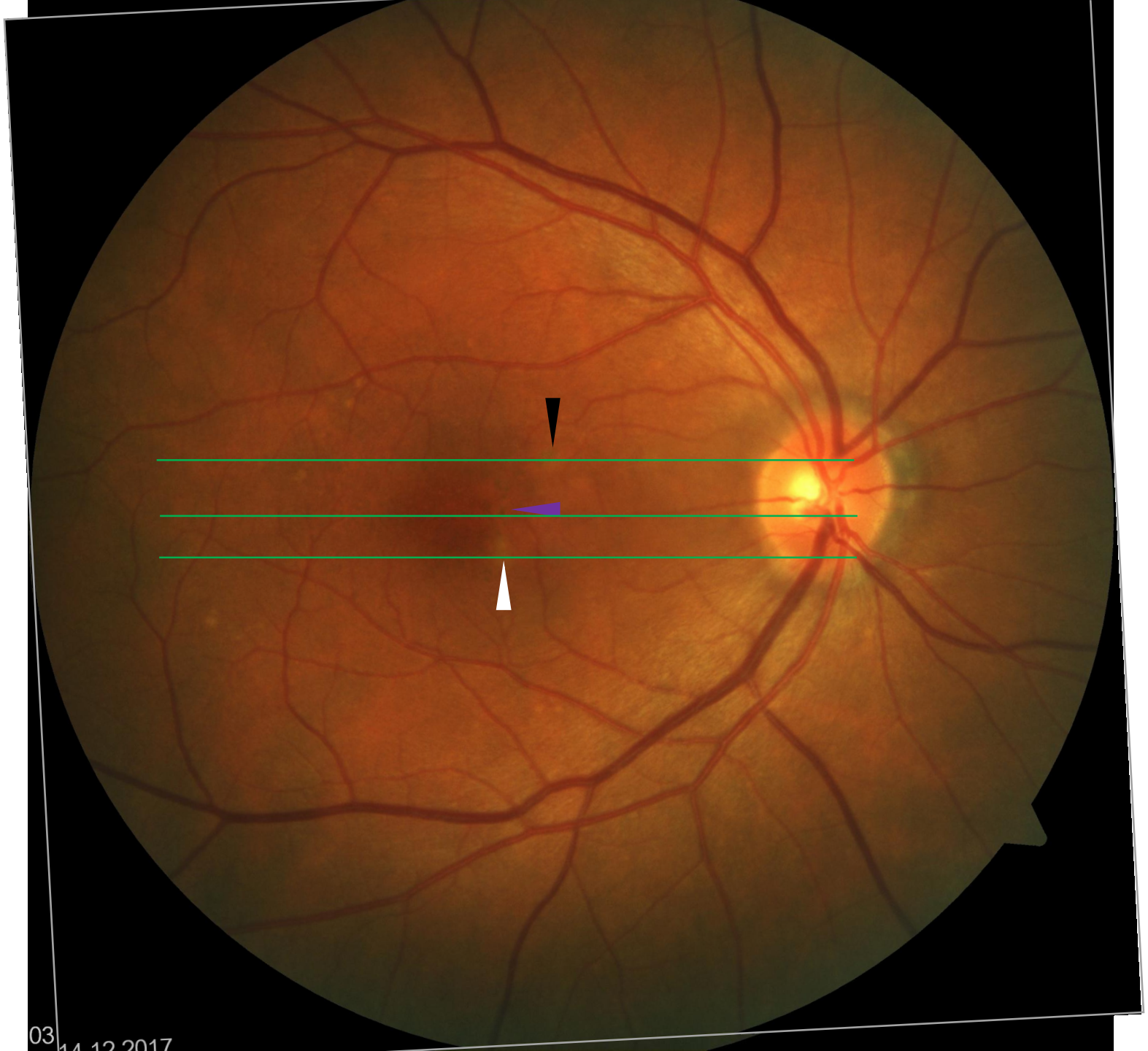

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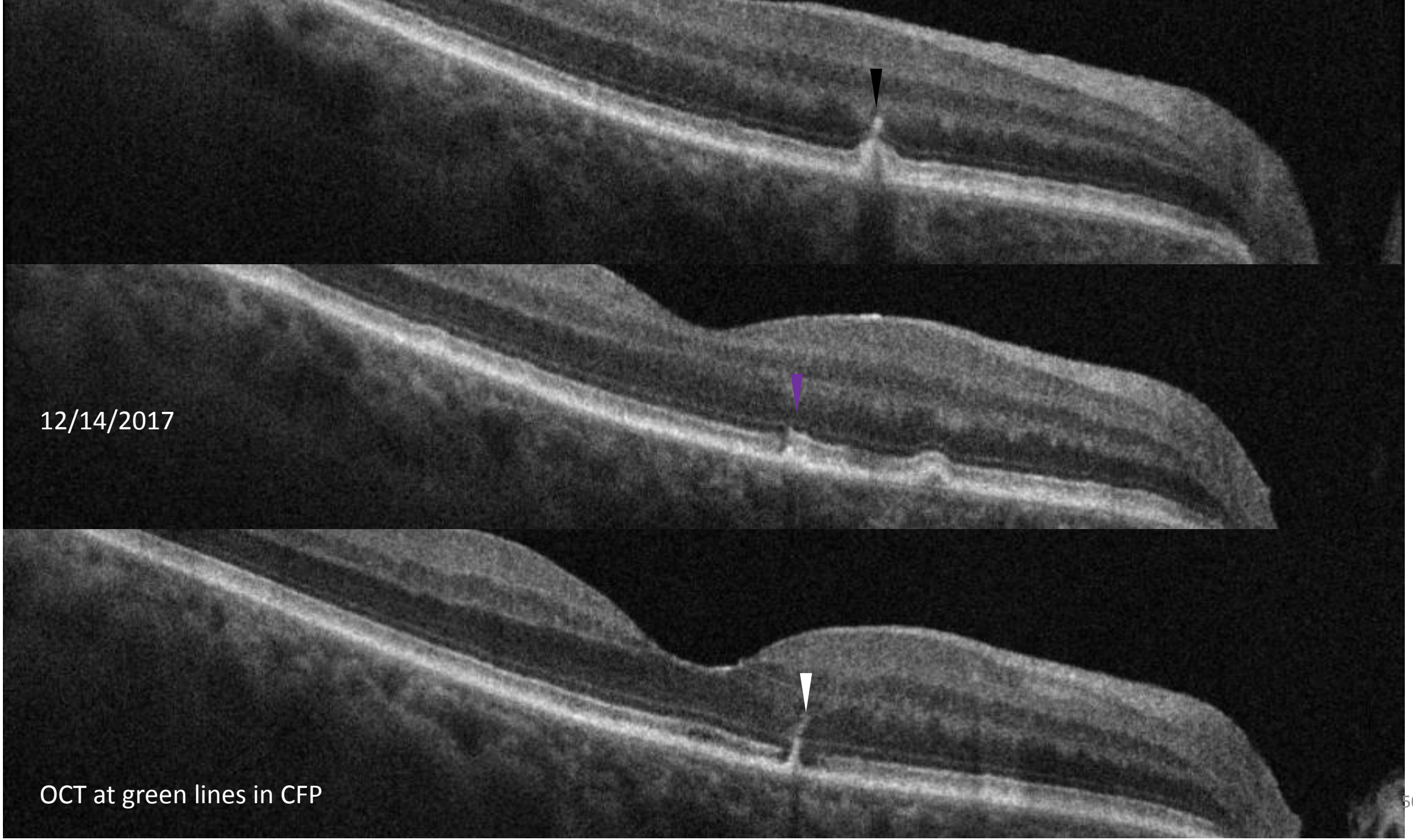
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OCT at green lines in CFP

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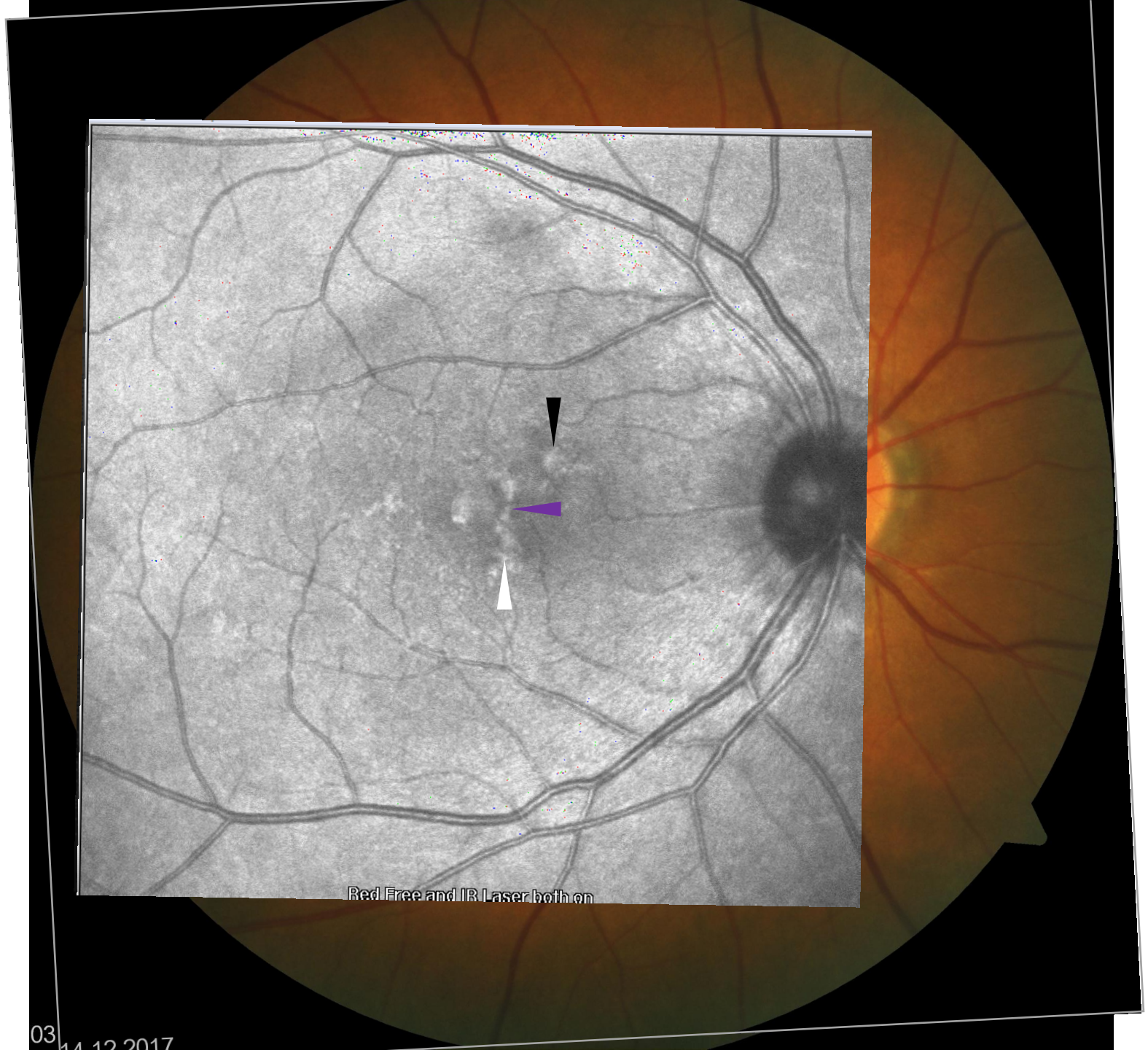

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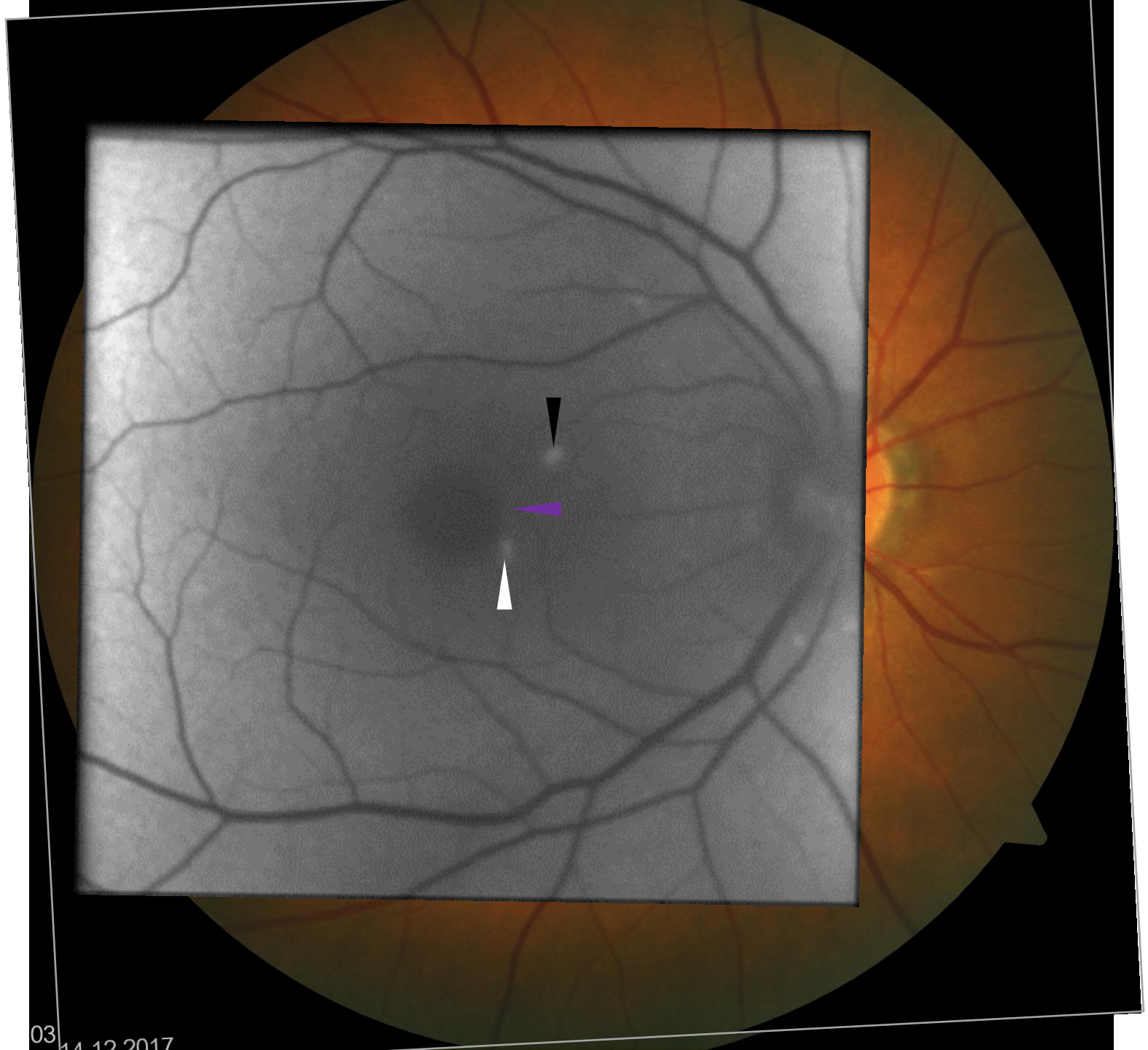

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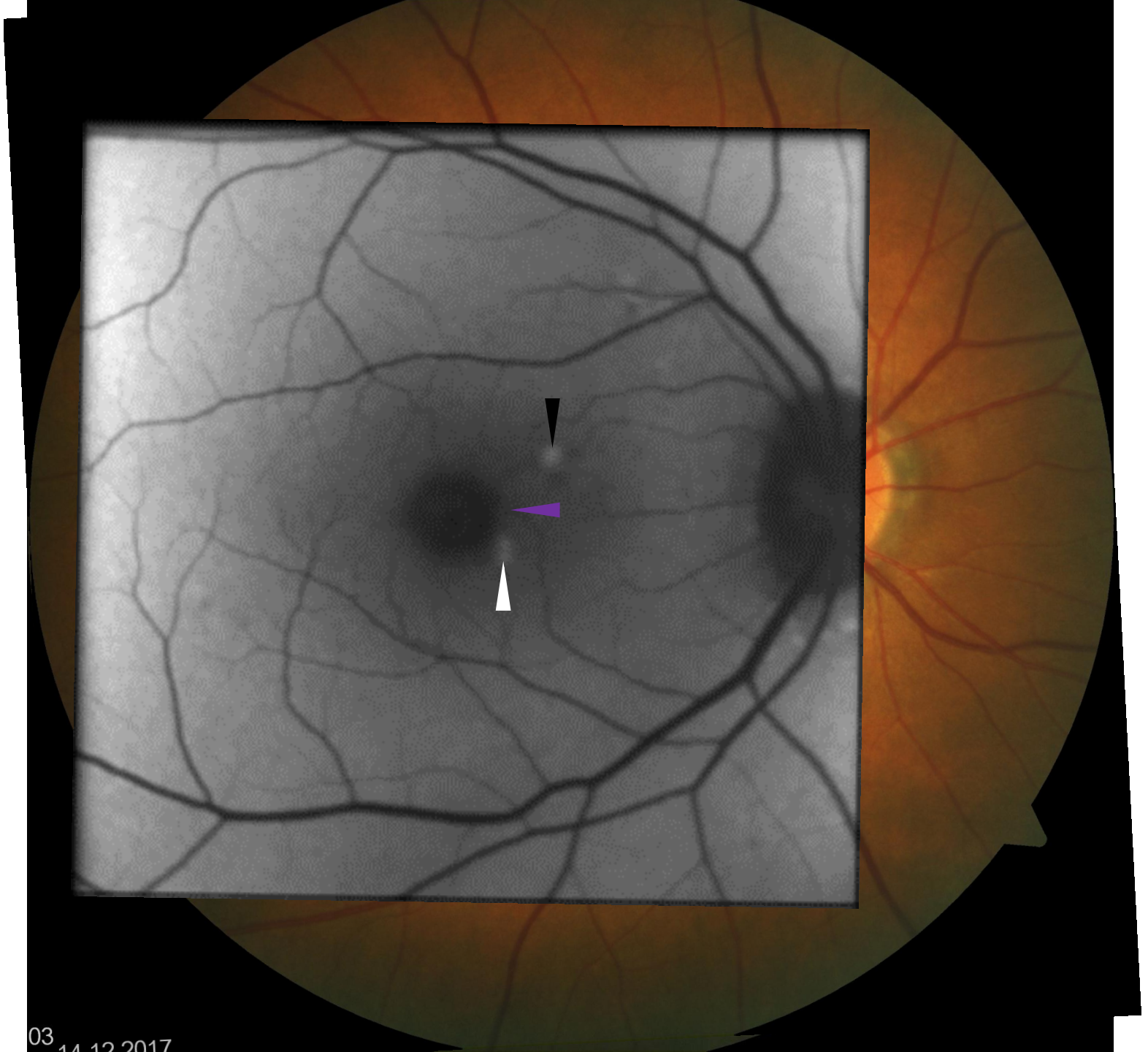

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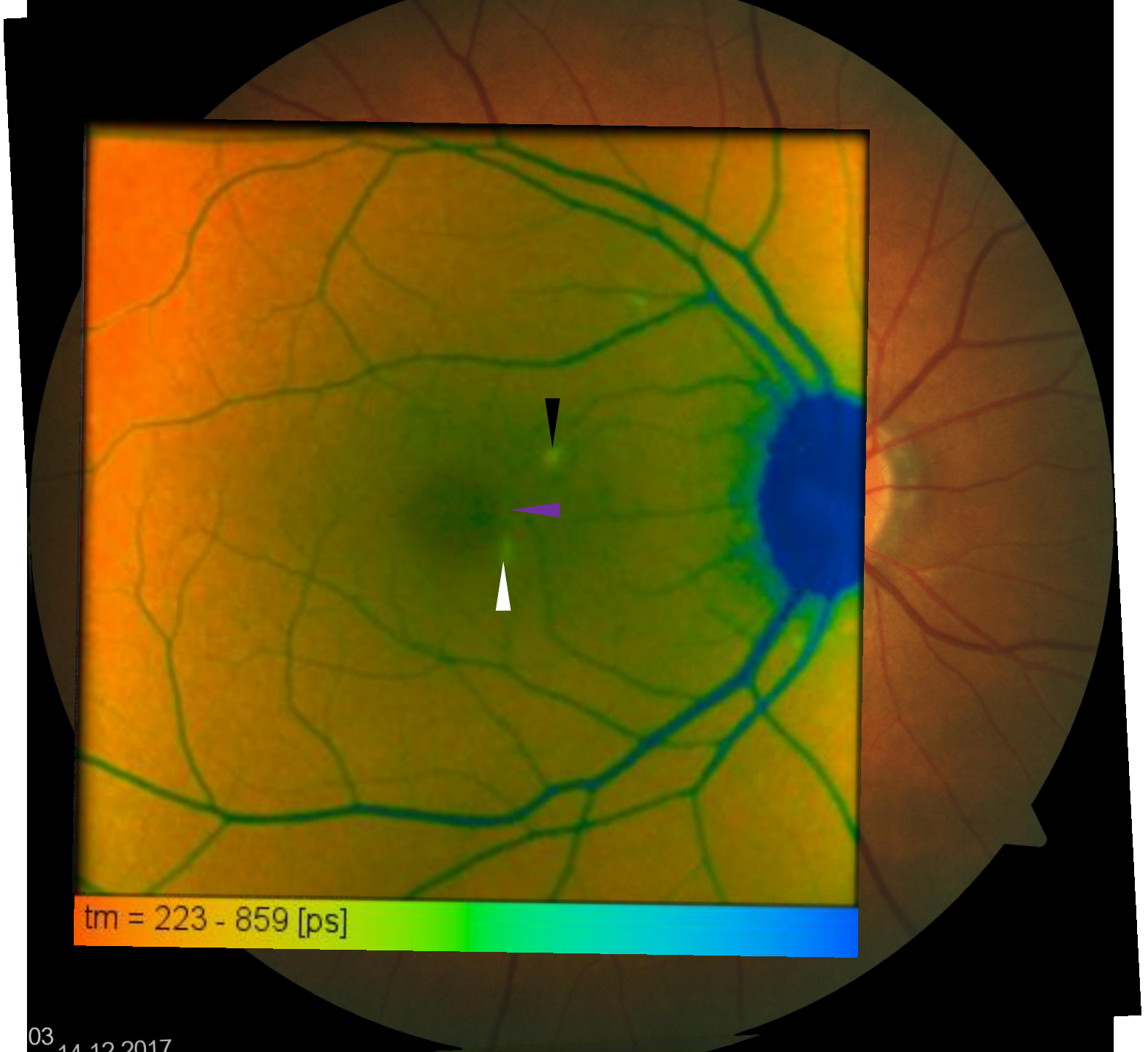
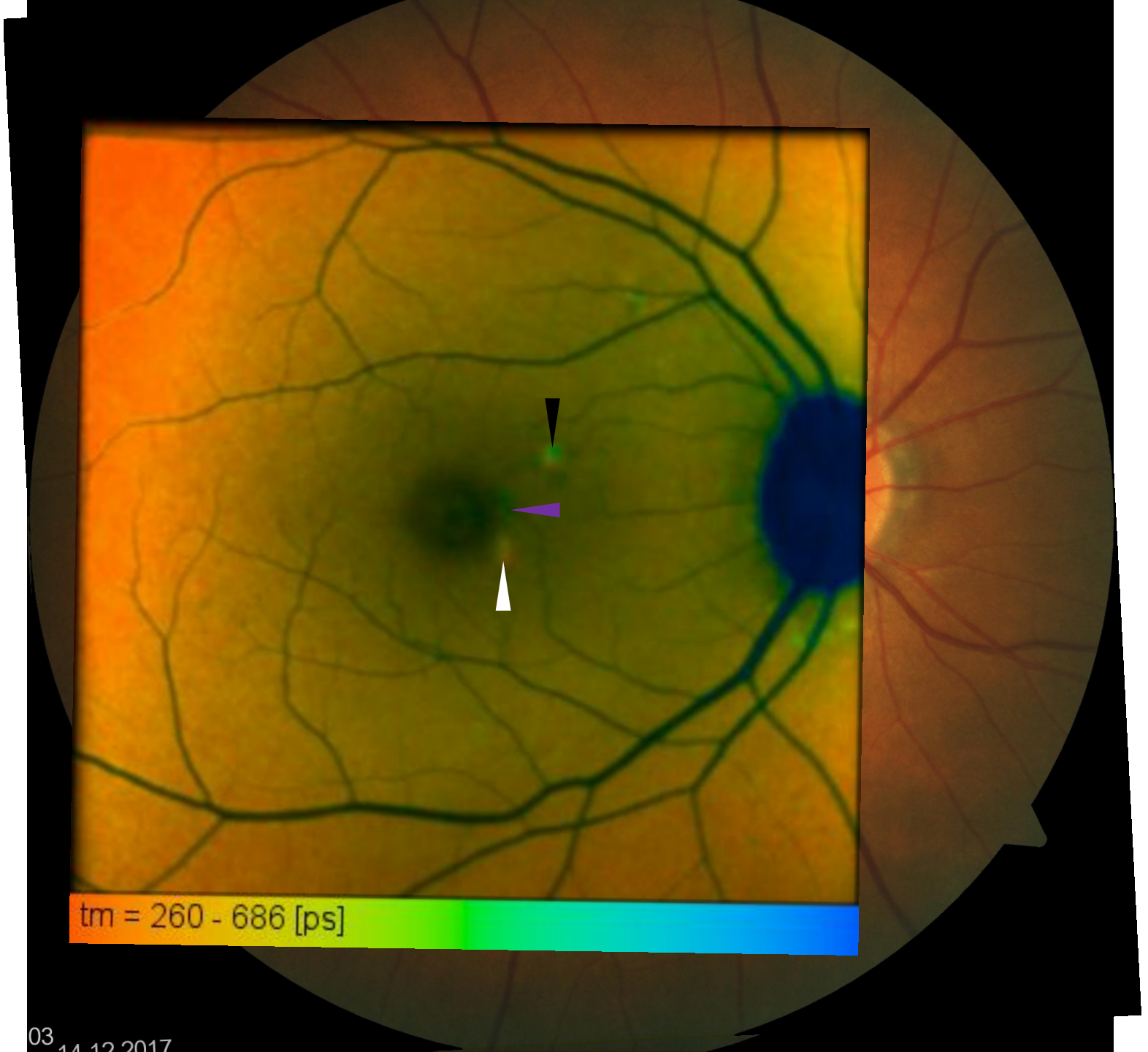


12/14/2017

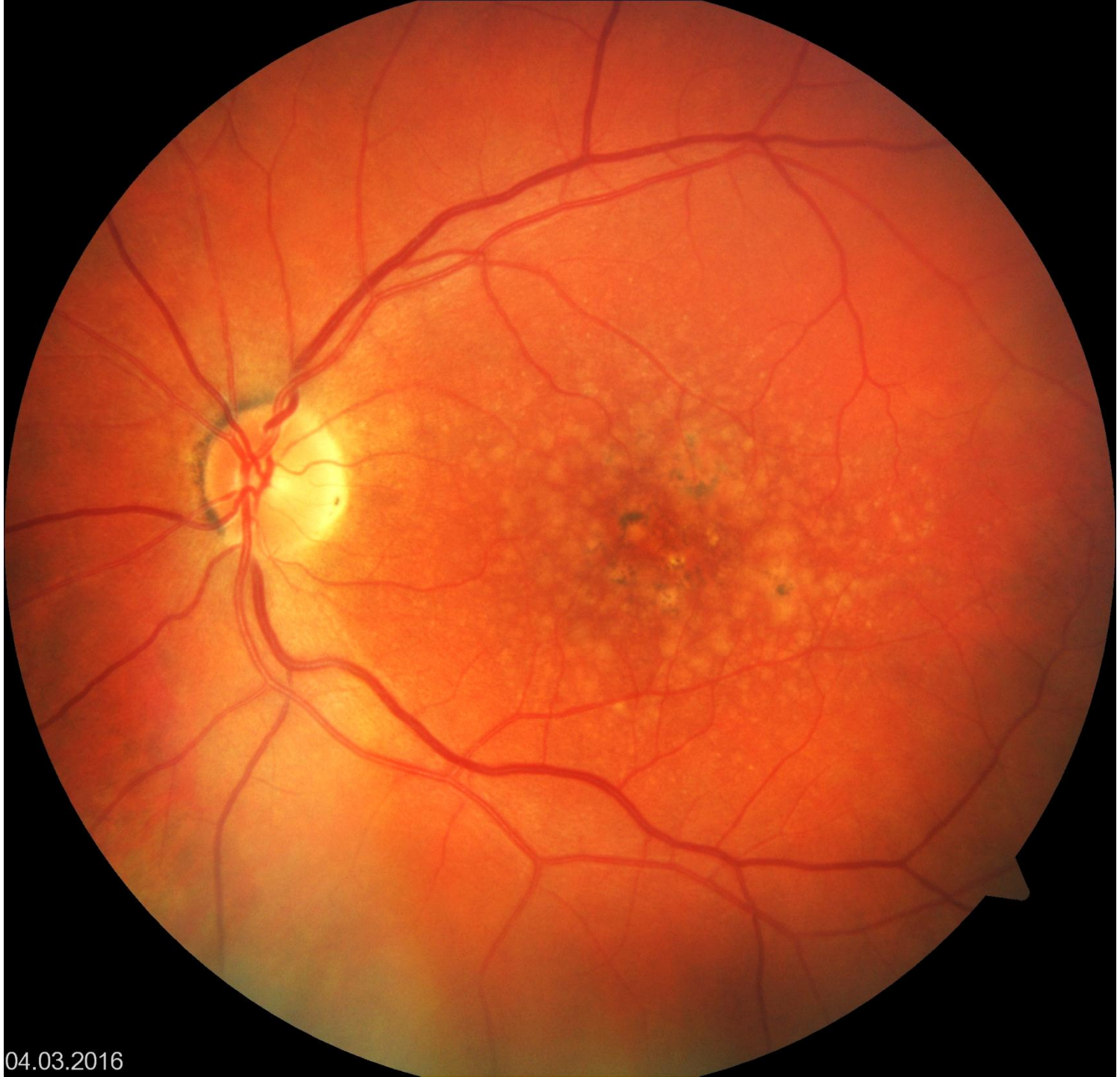


FLIO LSC

AMD_26, 61 years

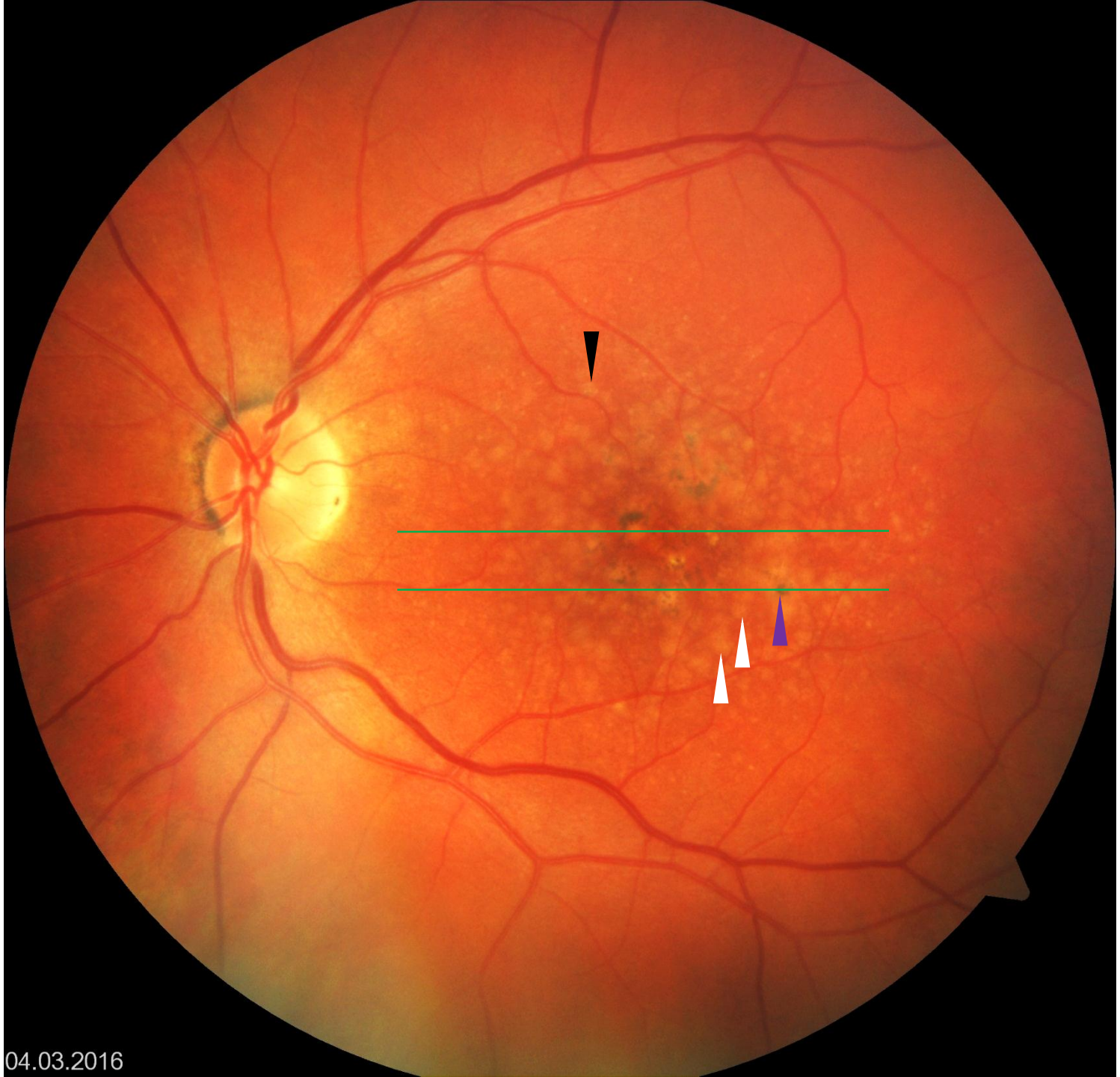
This patient (whom we, unfortunately, lost from follow up) showed multiple large drusen, SDD, and hyperpigmentations. Whereas most drusen showed shorter lifetimes than their environment (white arrowheads show two examples), the druse at the black arrowhead had longer lifetime (best seen in SSC). We don't see hyperpigmentation there but can't exclude it. Eventually, RPE changes occur before detachment from Bruch's and migration into the outer retina. At the purple arrowhead, a strong hyperpigmentation is seen in CFP, OCT showed thickened RPE but no migration yet. In contrast to all other hyperpigmentations, this one had very short lifetime. **What could make the difference?**

03/04/2016



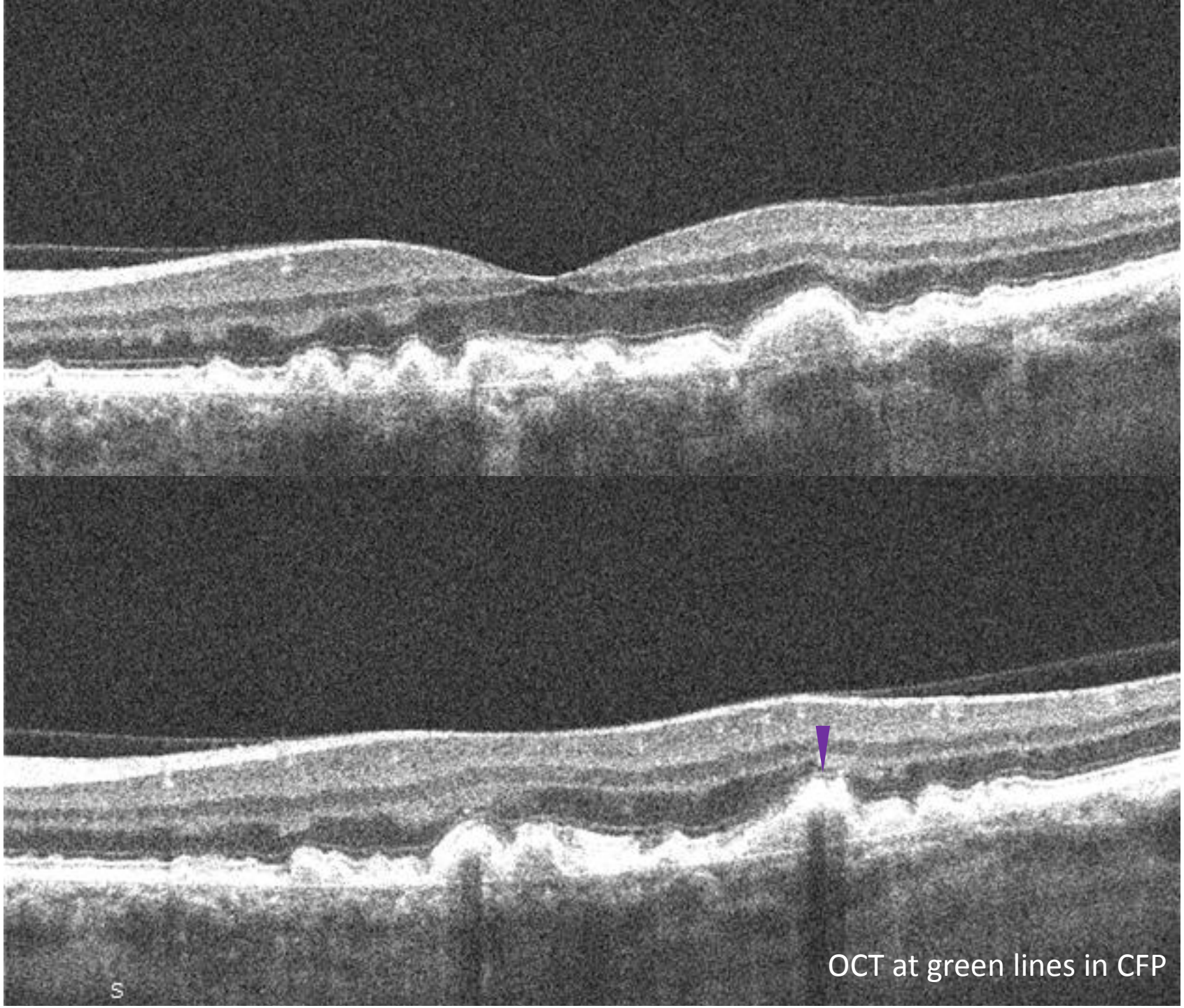
CFP

03/04/2016



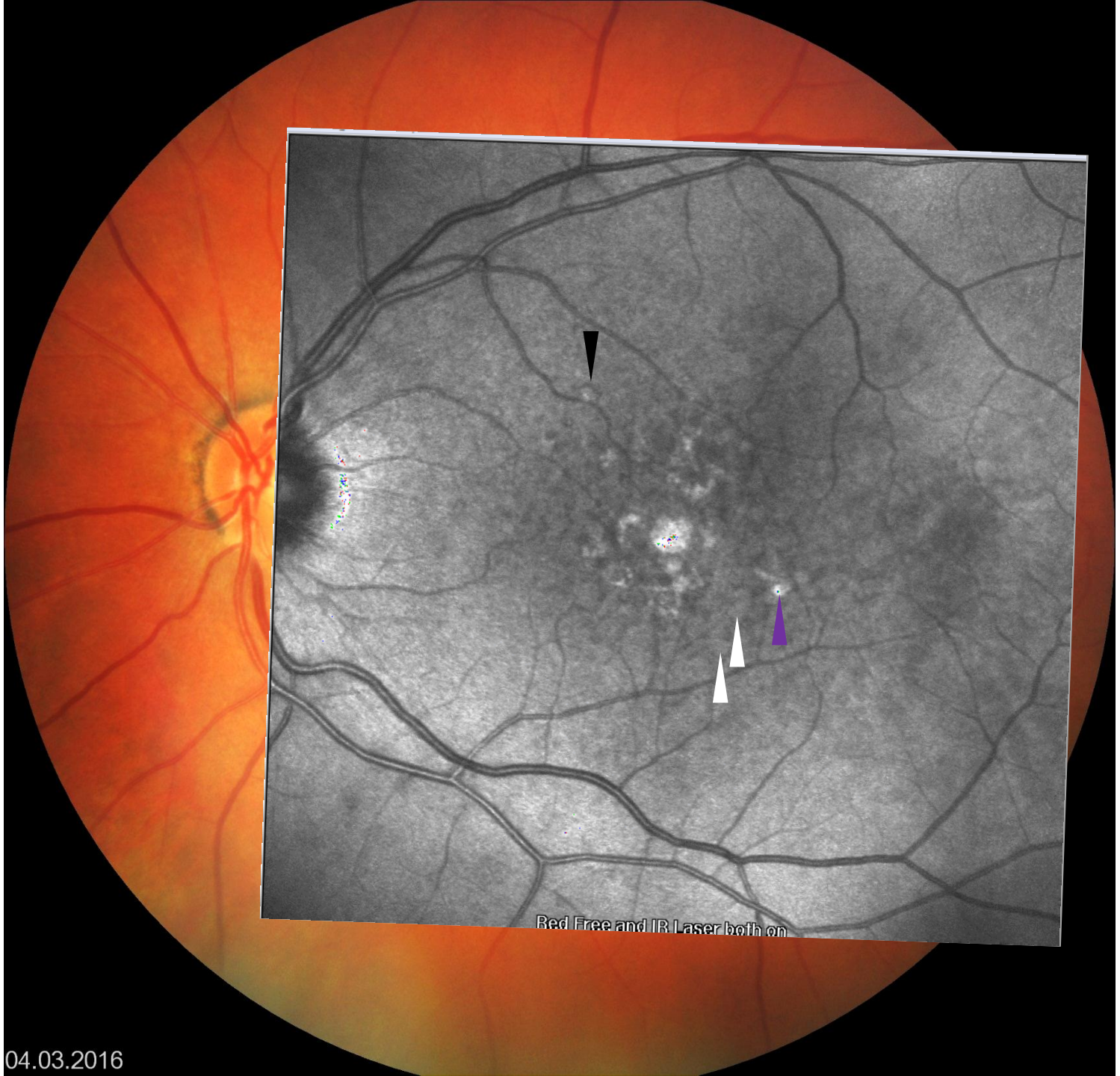
CFP

03/04/2016



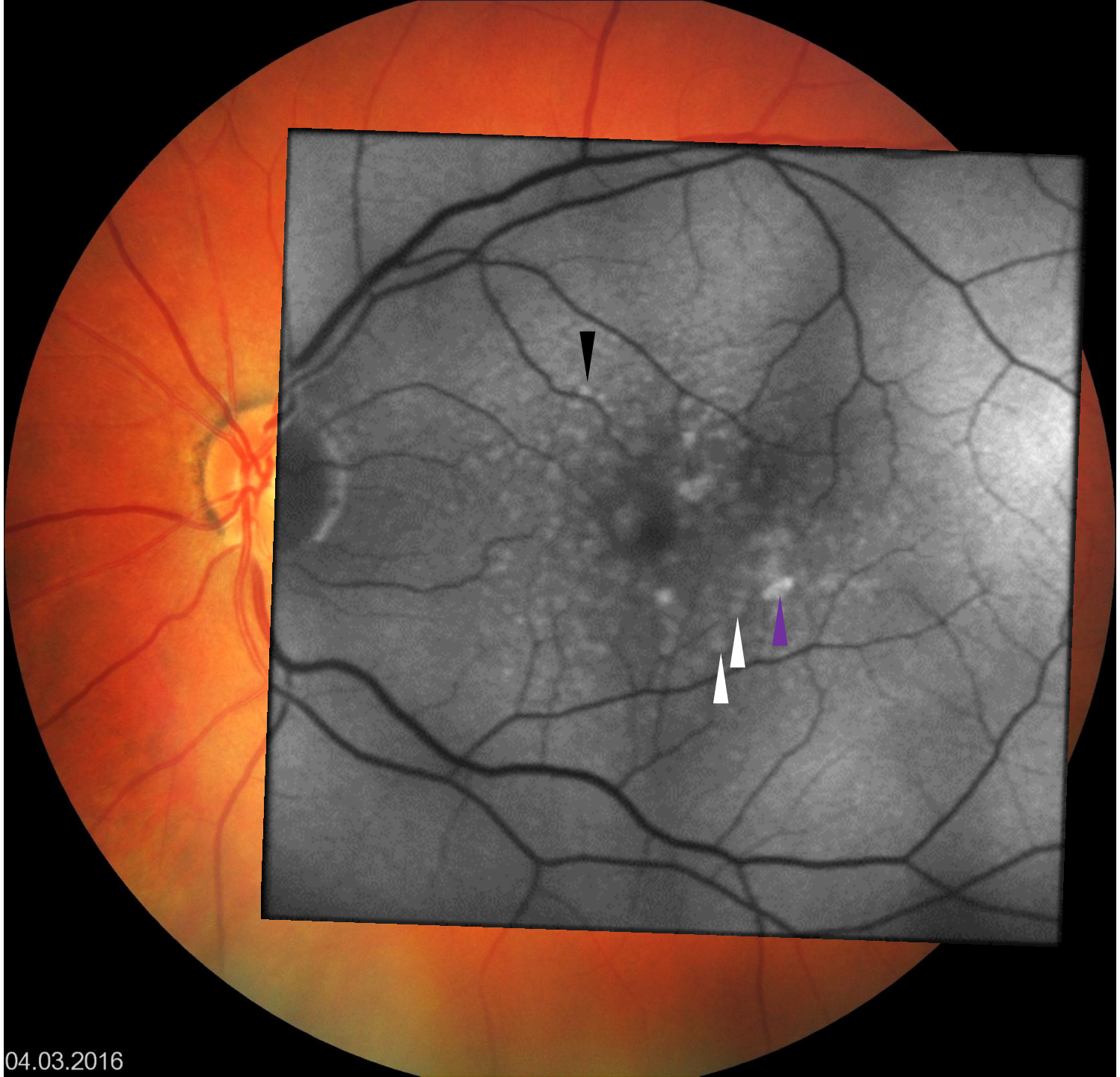
OCT at green lines in CFP

03/04/2016



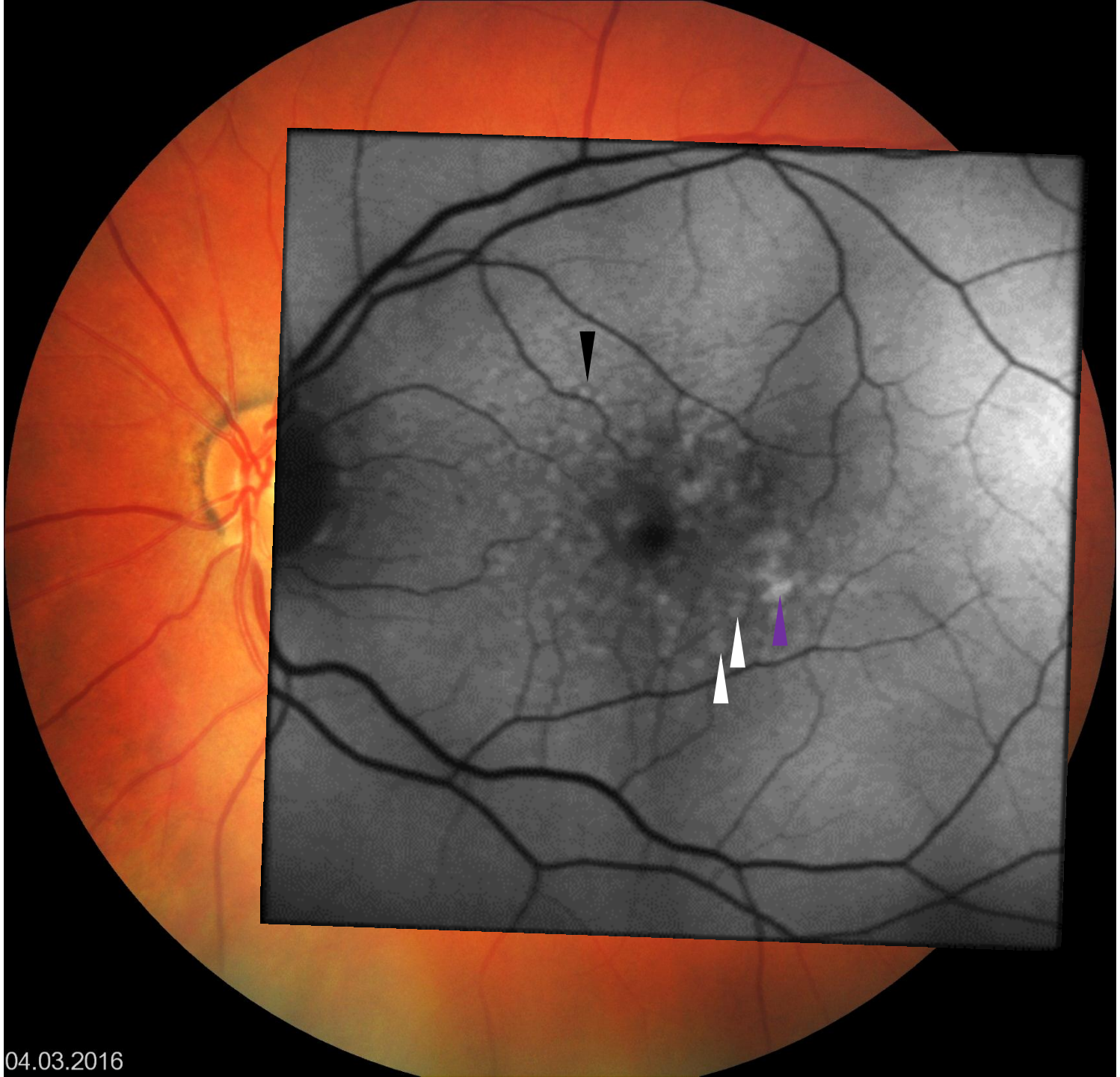
IR

03/04/2016



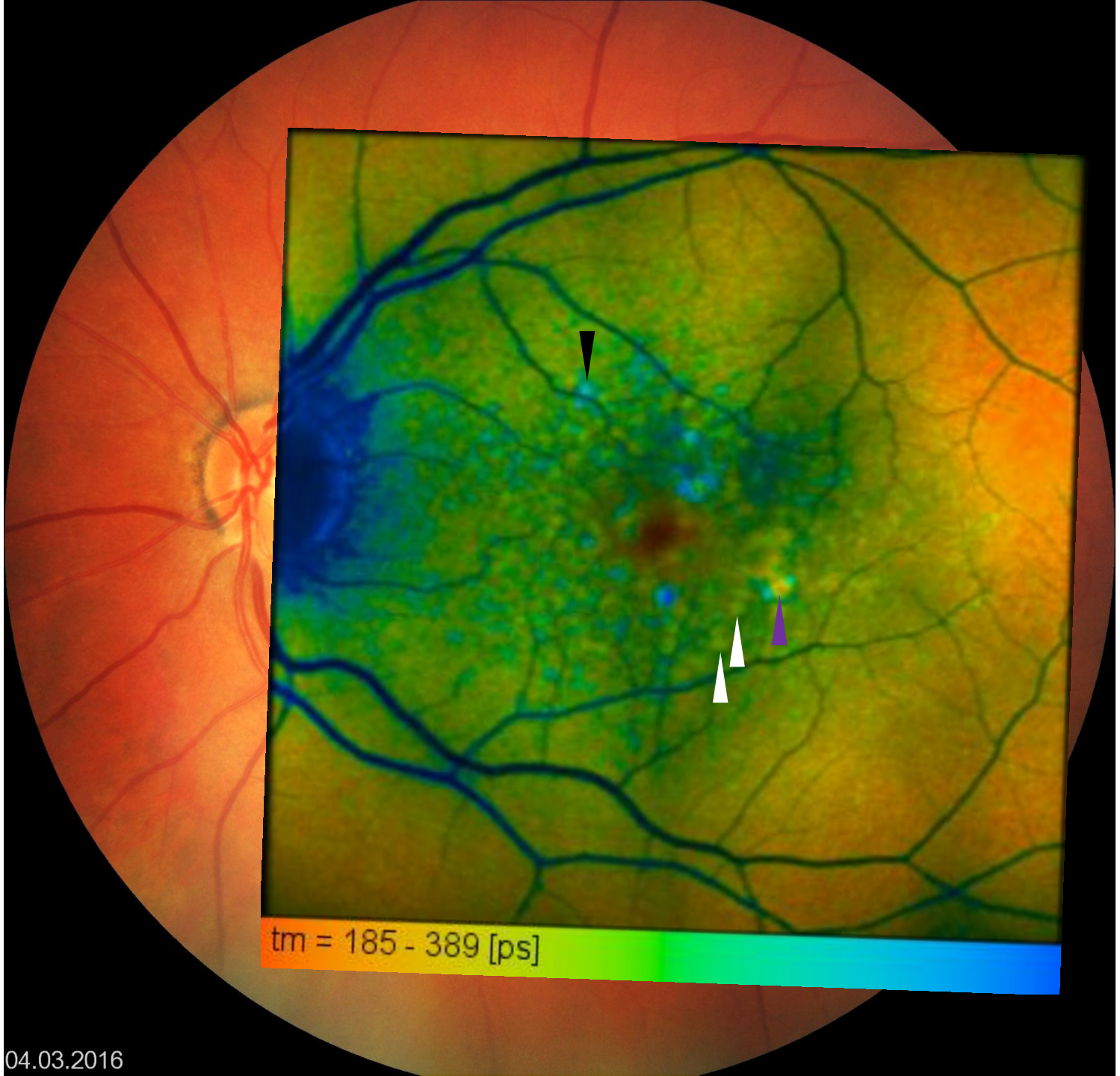
FAF SSC

03/04/2016



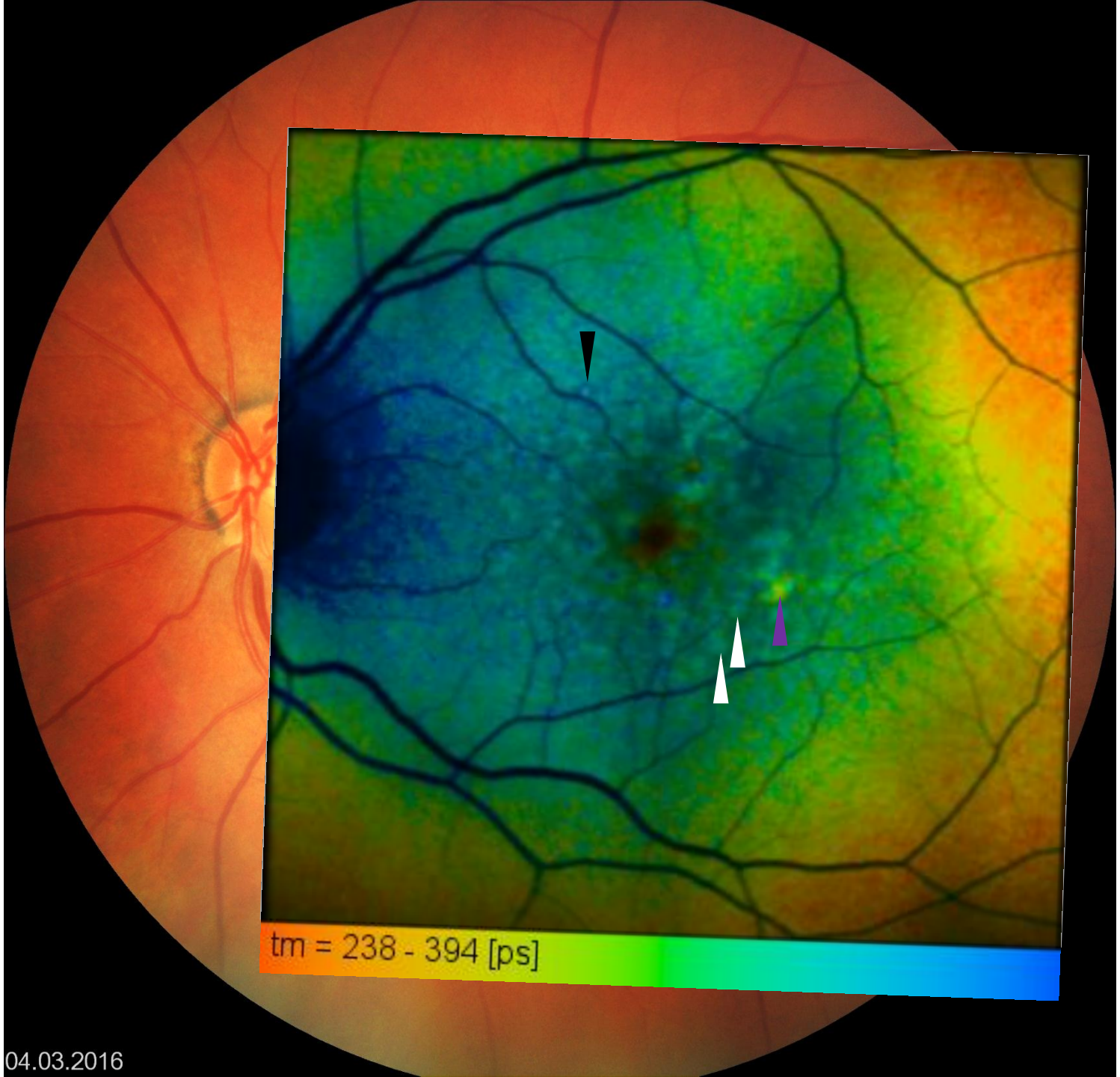
FAF LSC

03/04/2016



FLIO SSC

03/04/2016

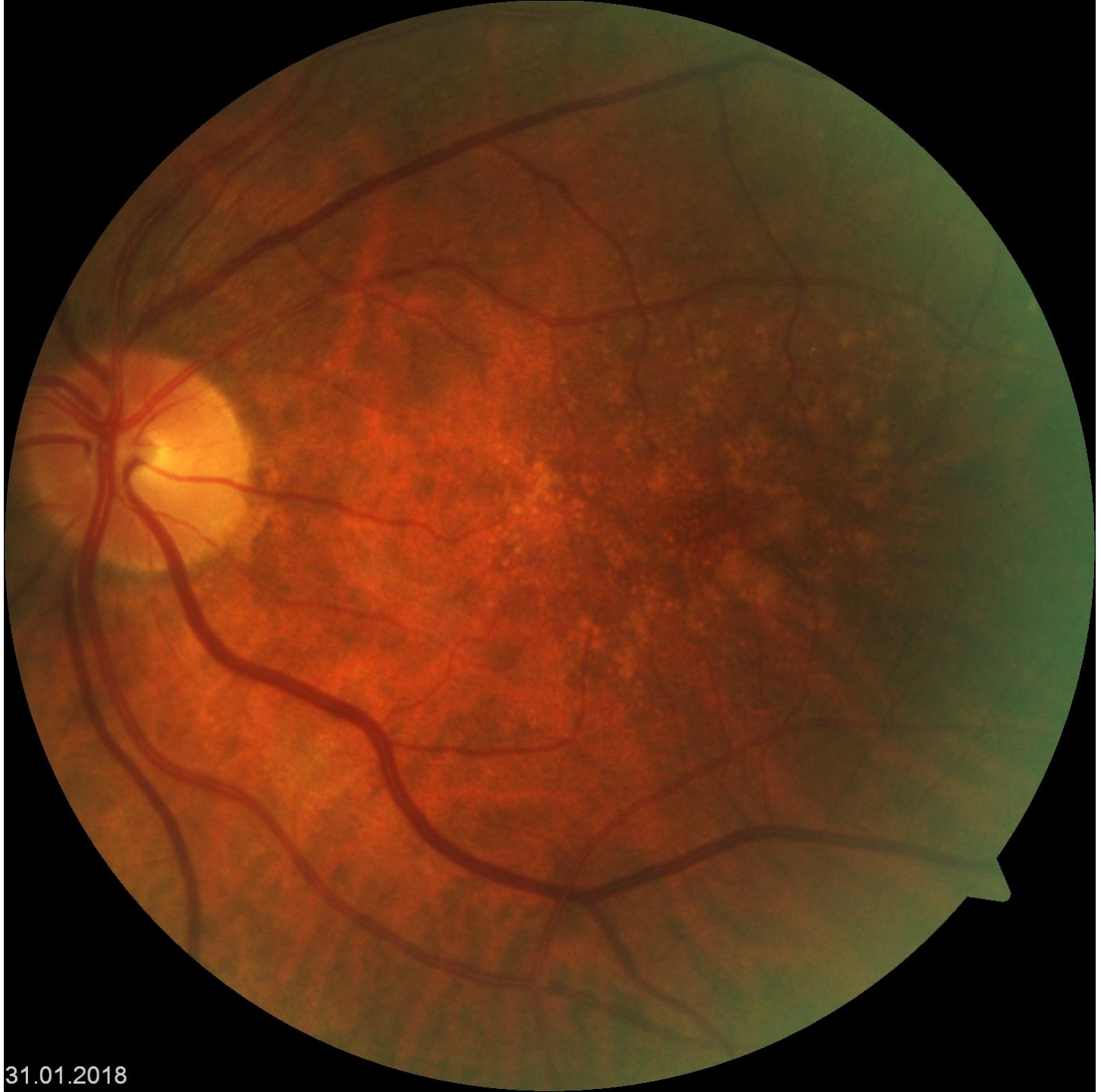


FLIO LSC

AMD_27, 72 years

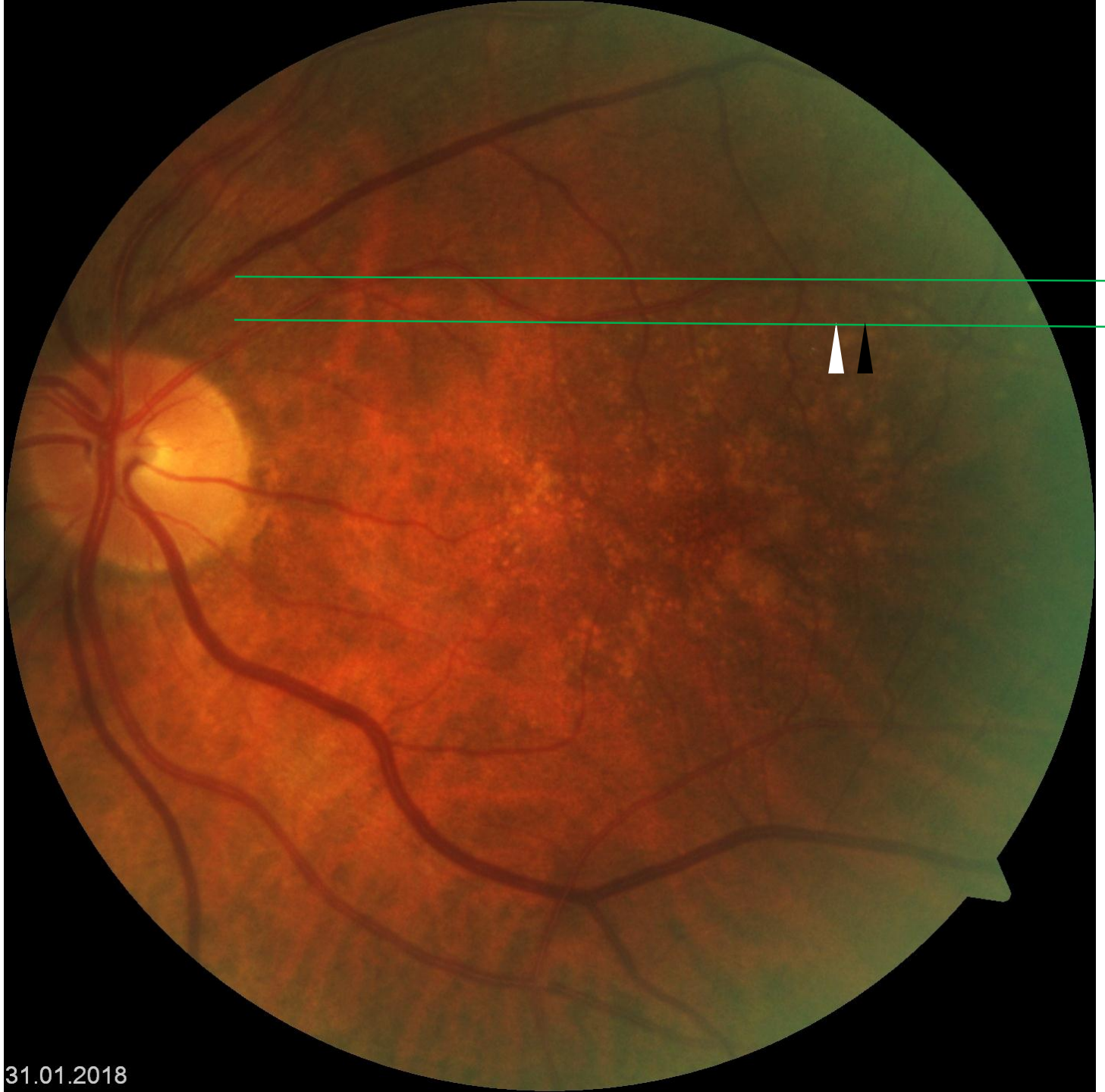
SDD show longer lifetimes as known. However, drusen (white arrowhead) and SDD (black arrowhead) can occur in very close proximity. Here, we can distinguish them by AF intensity and lifetime.
No challenge for this case ☺.

01/31/2018

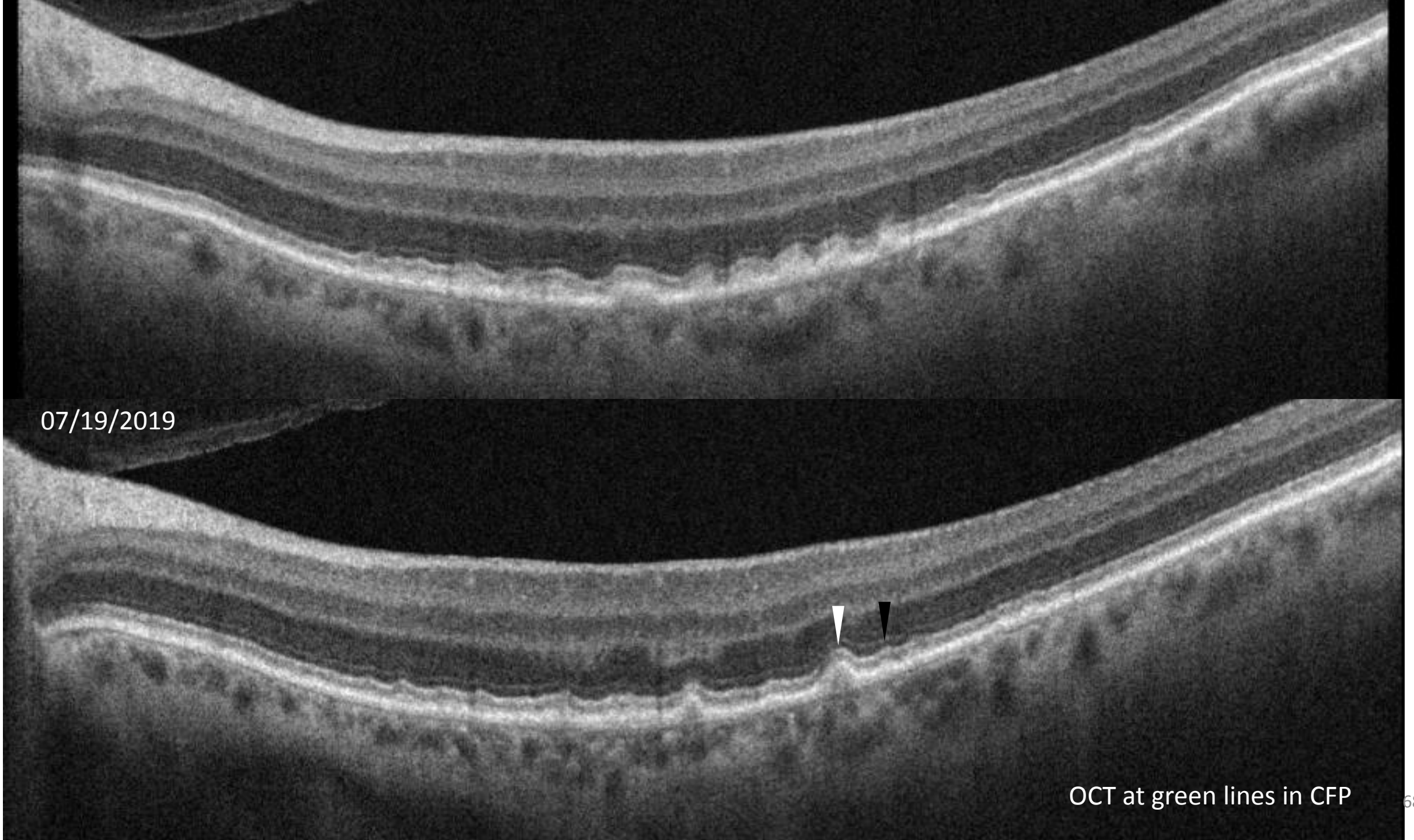


CFP

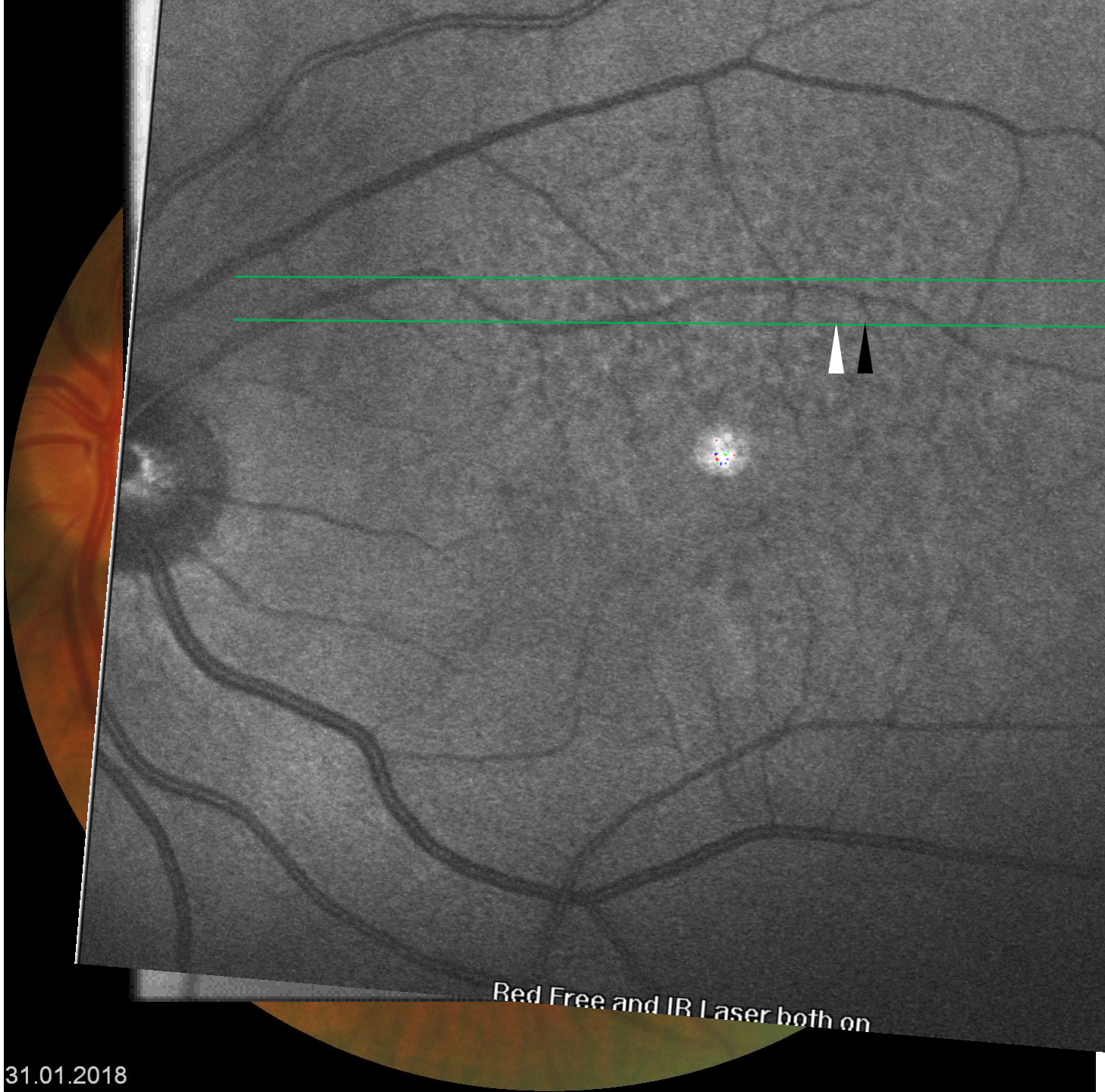
01/31/2018



CFP

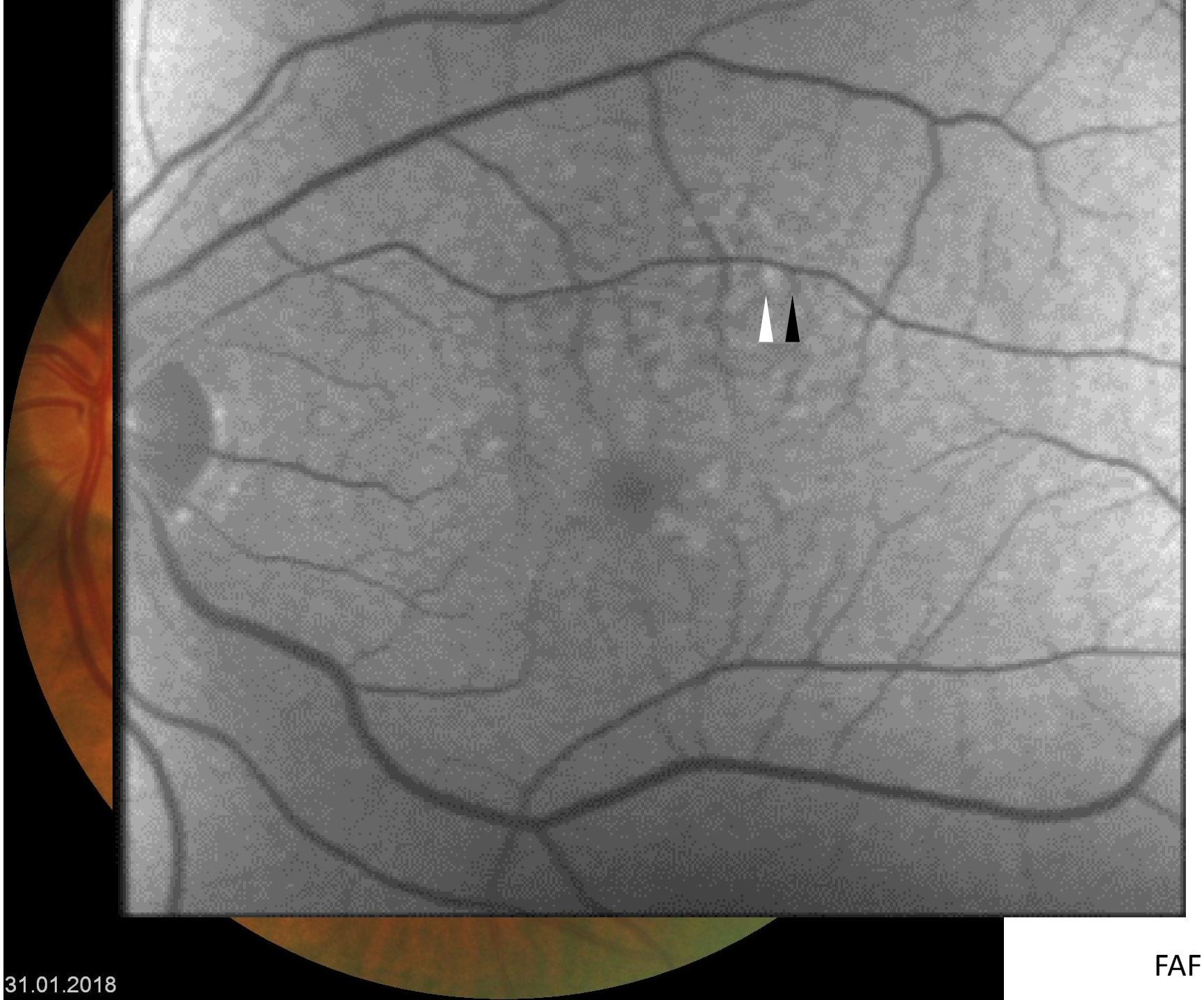

07/19/2019
OCT at green lines in CFP

07/19/2019



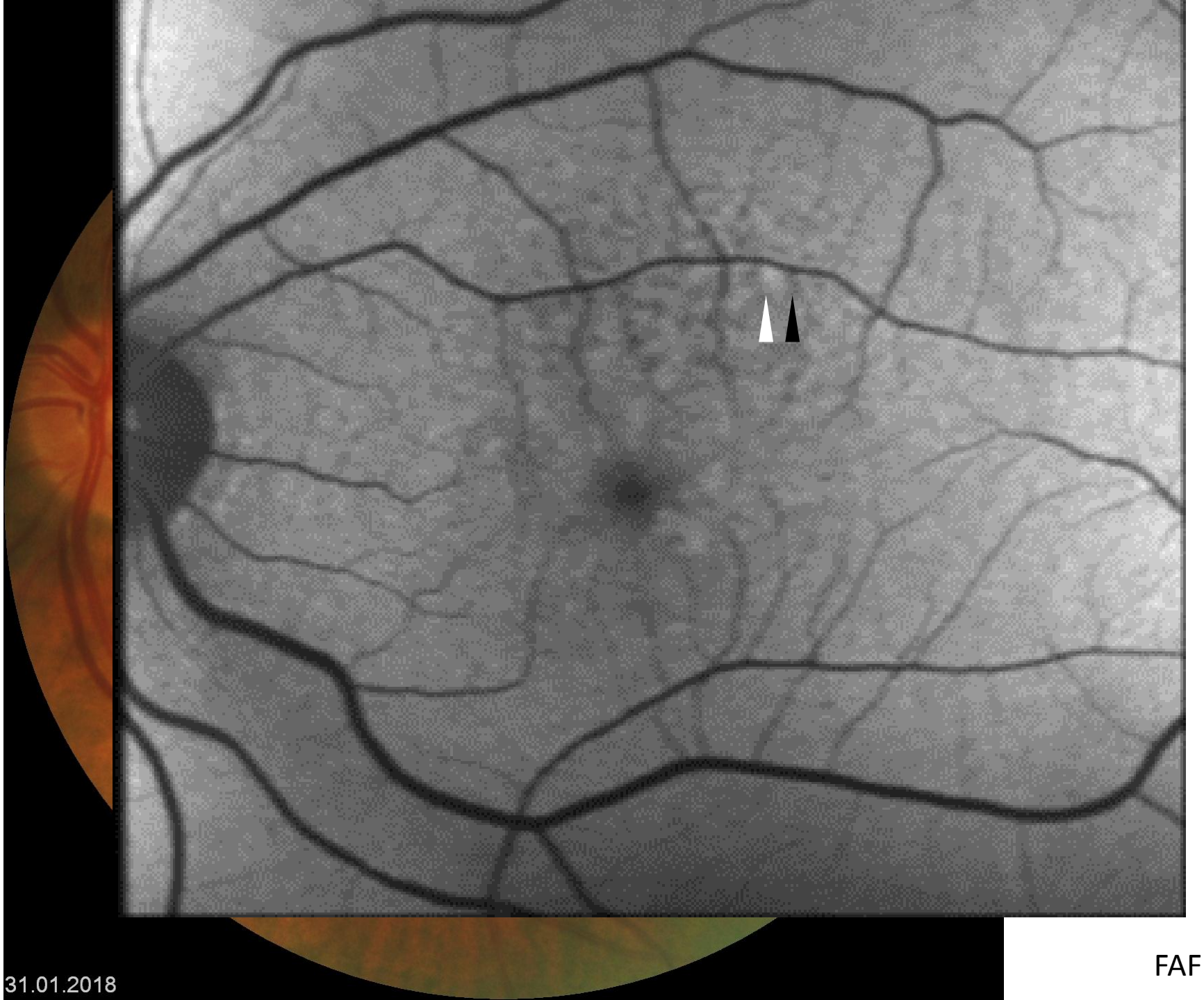
IR

07/19/2019



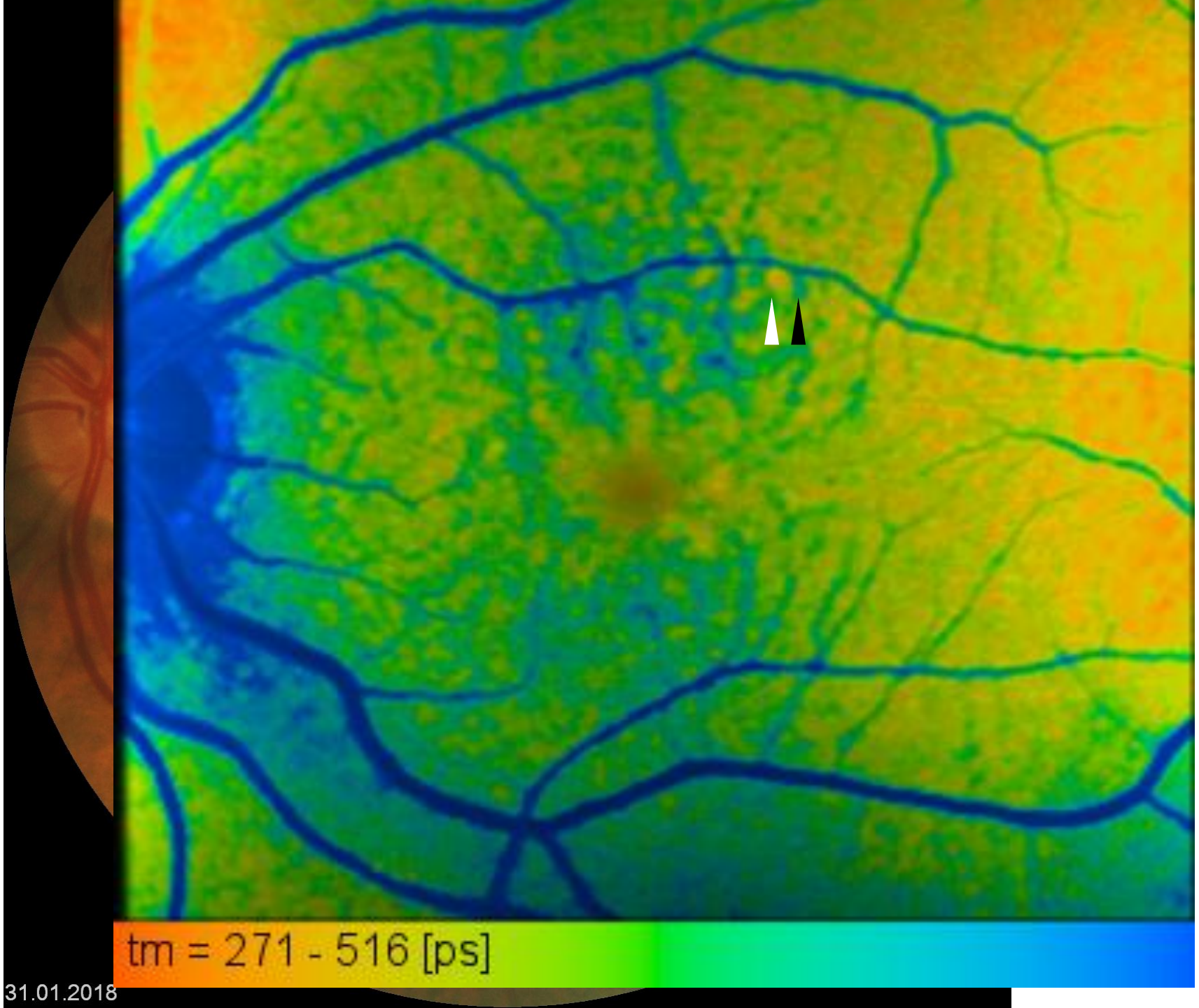
FAF SSC

07/19/2019



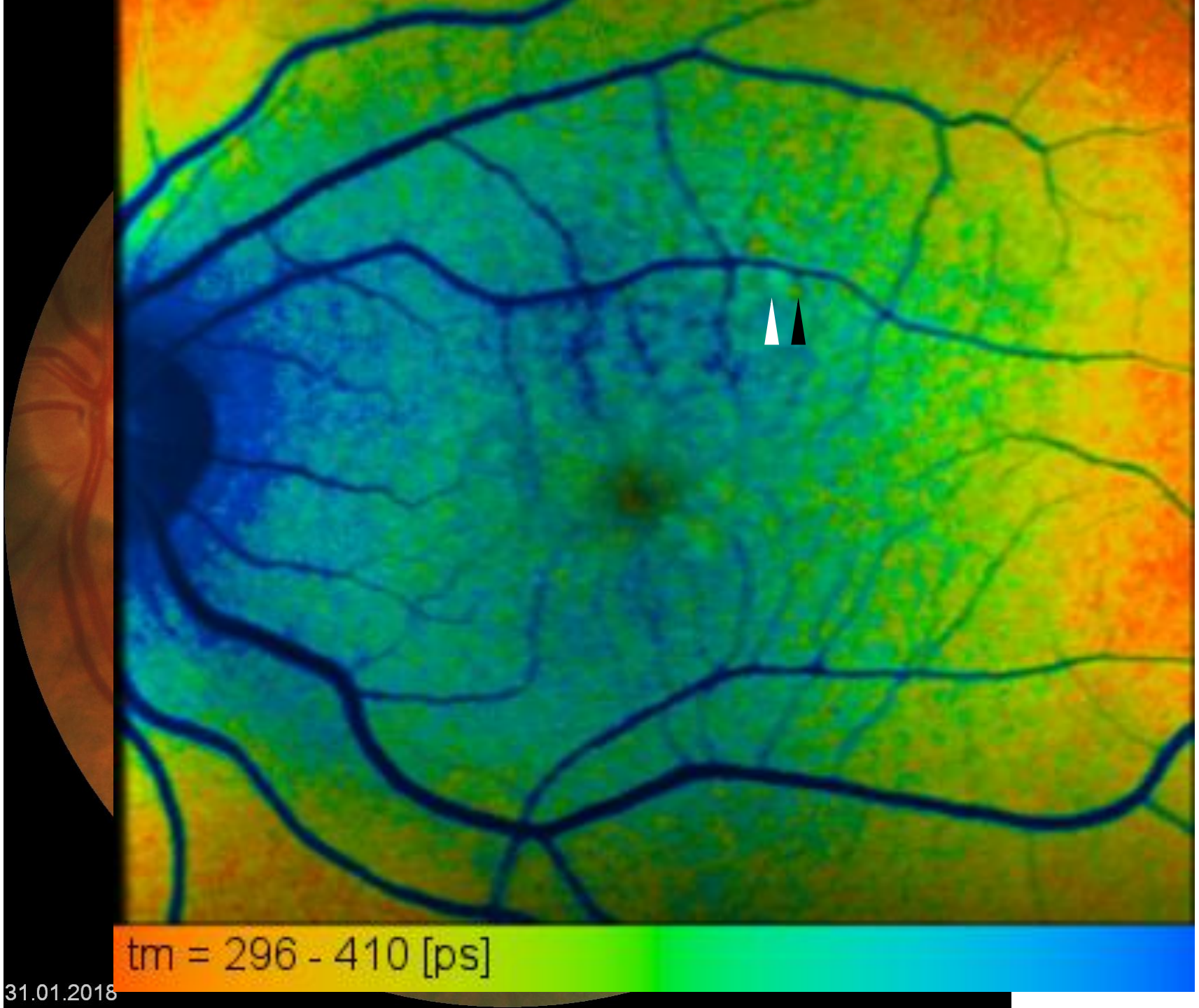
FAF LSC

07/19/2019



FLIO SSC

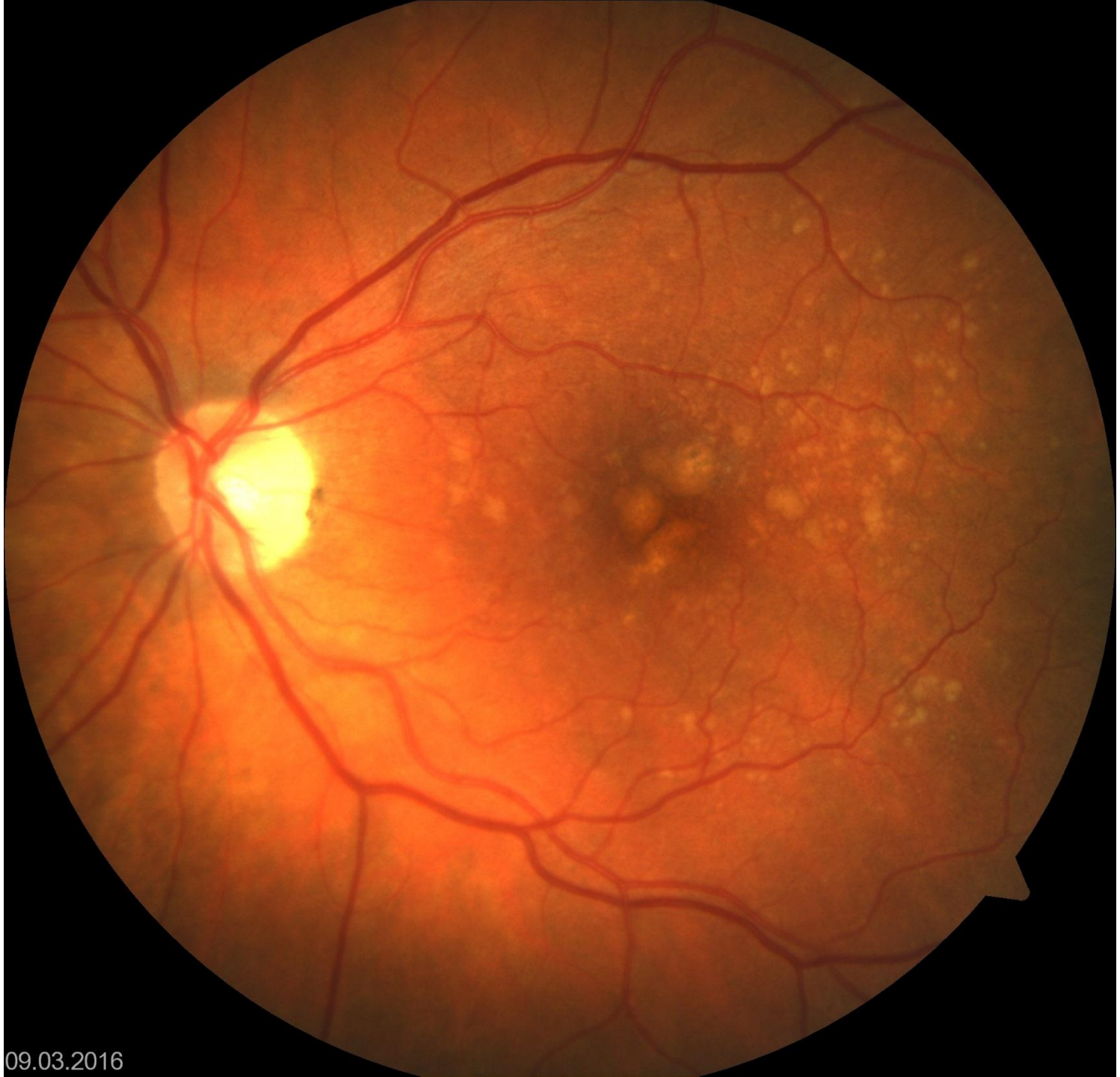
07/19/2019



AMD_28, 78 years at baseline

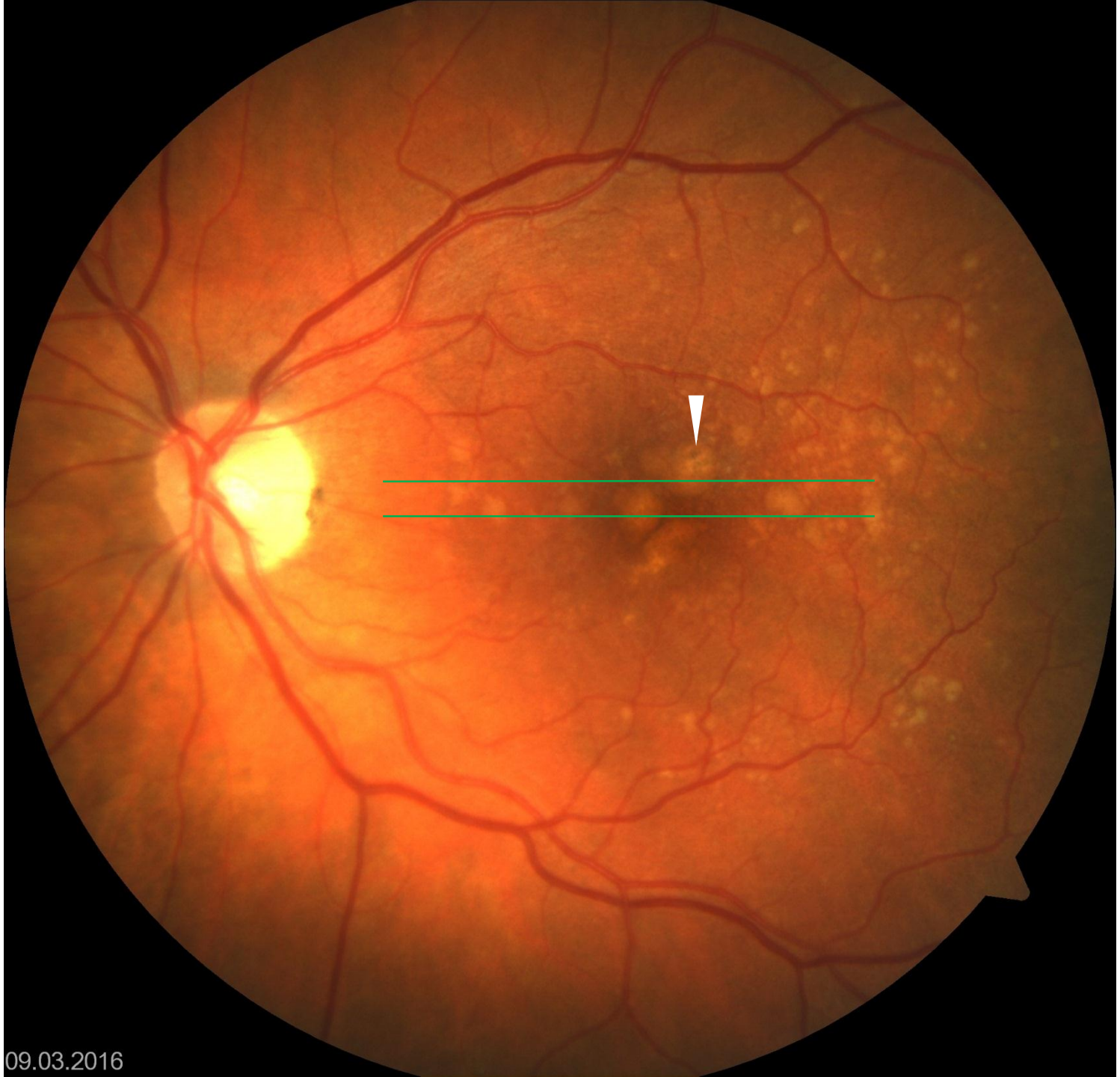
Patient with large macular drusen which, in part, disappeared (collapsed?) during follow up for 4 years (actually were not found any more in OCT one year after baseline, images not shown) without leading to GA. Arrowhead: A druse (unfortunately not well met in OCT) with strong AF in both spectral channels and very short lifetime although some hyperpigmentation is seen in CFP. 4 years later, this hyperfluorescence showed long lifetimes. OCT shows a small spot of migrated RPE but only minimal, hardly visible hyperpigmentation is seen in CFP. **Can this explain the hyperfluorescence with long lifetime? Or shall we think about RPE changes before migration? What could have changed the AF lifetime at the arrowhead over time?**

03/09/2016

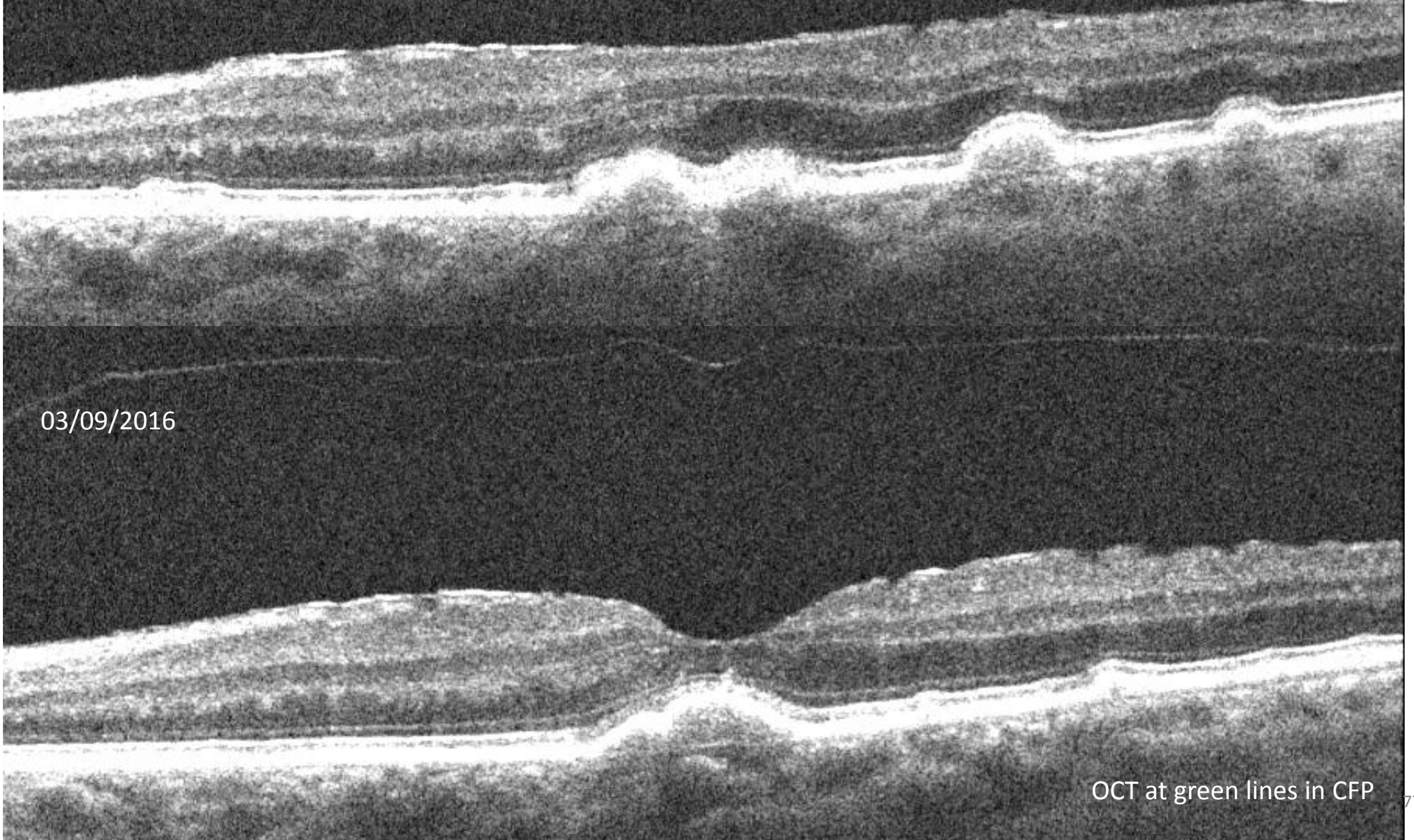


CFP

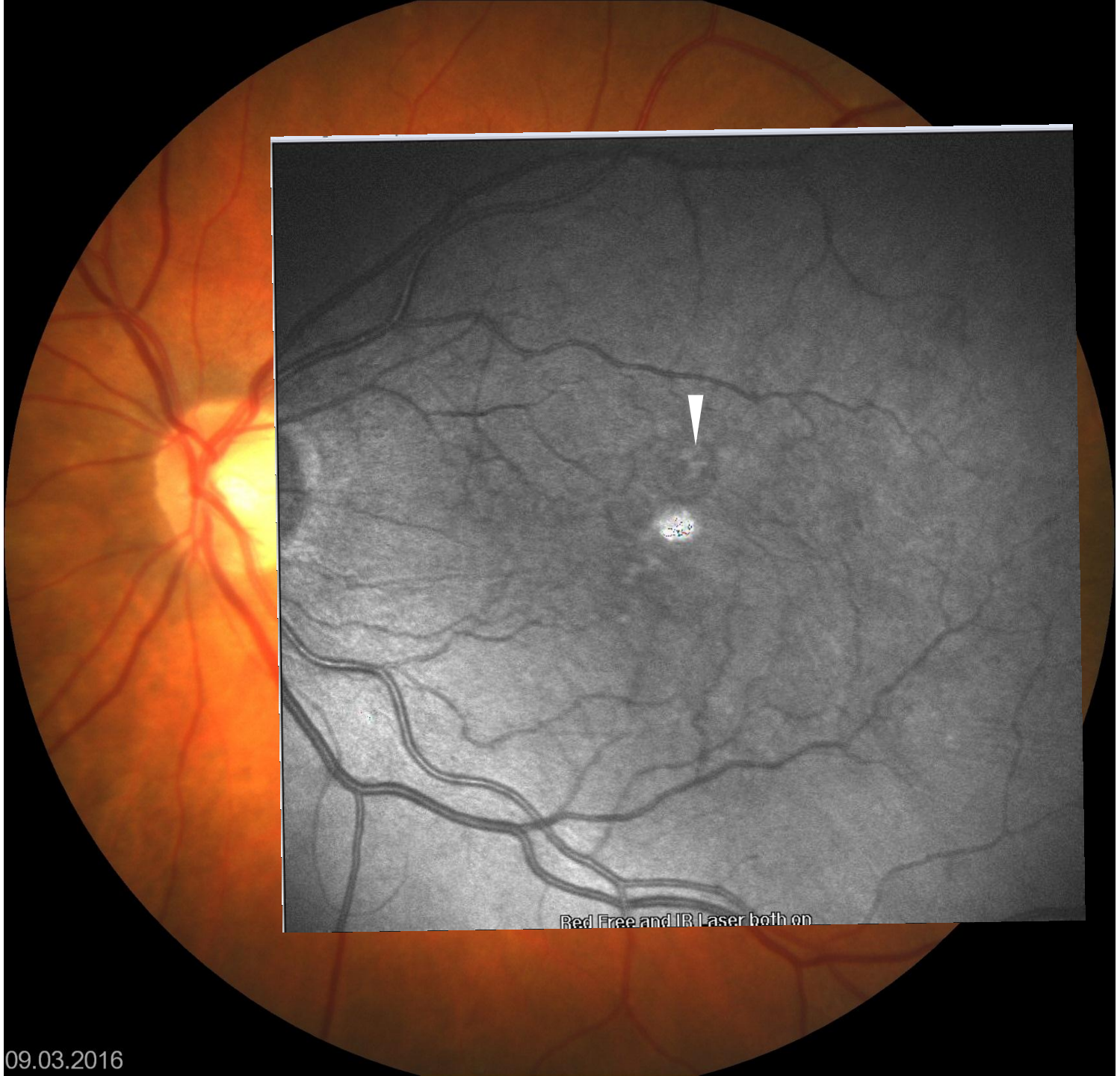
03/09/2016



CFP

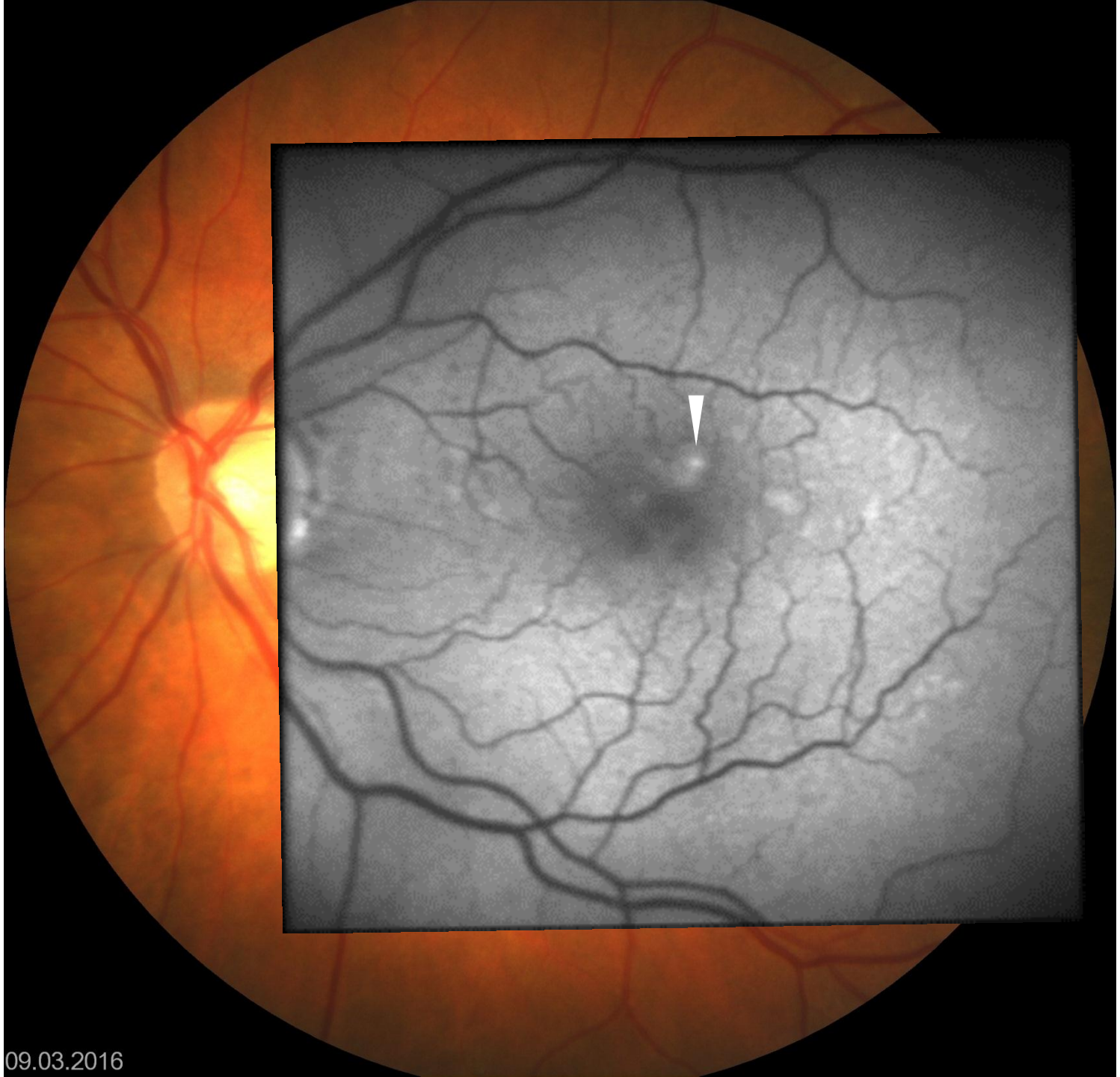

03/09/2016
OCT at green lines in CFP

03/09/2016



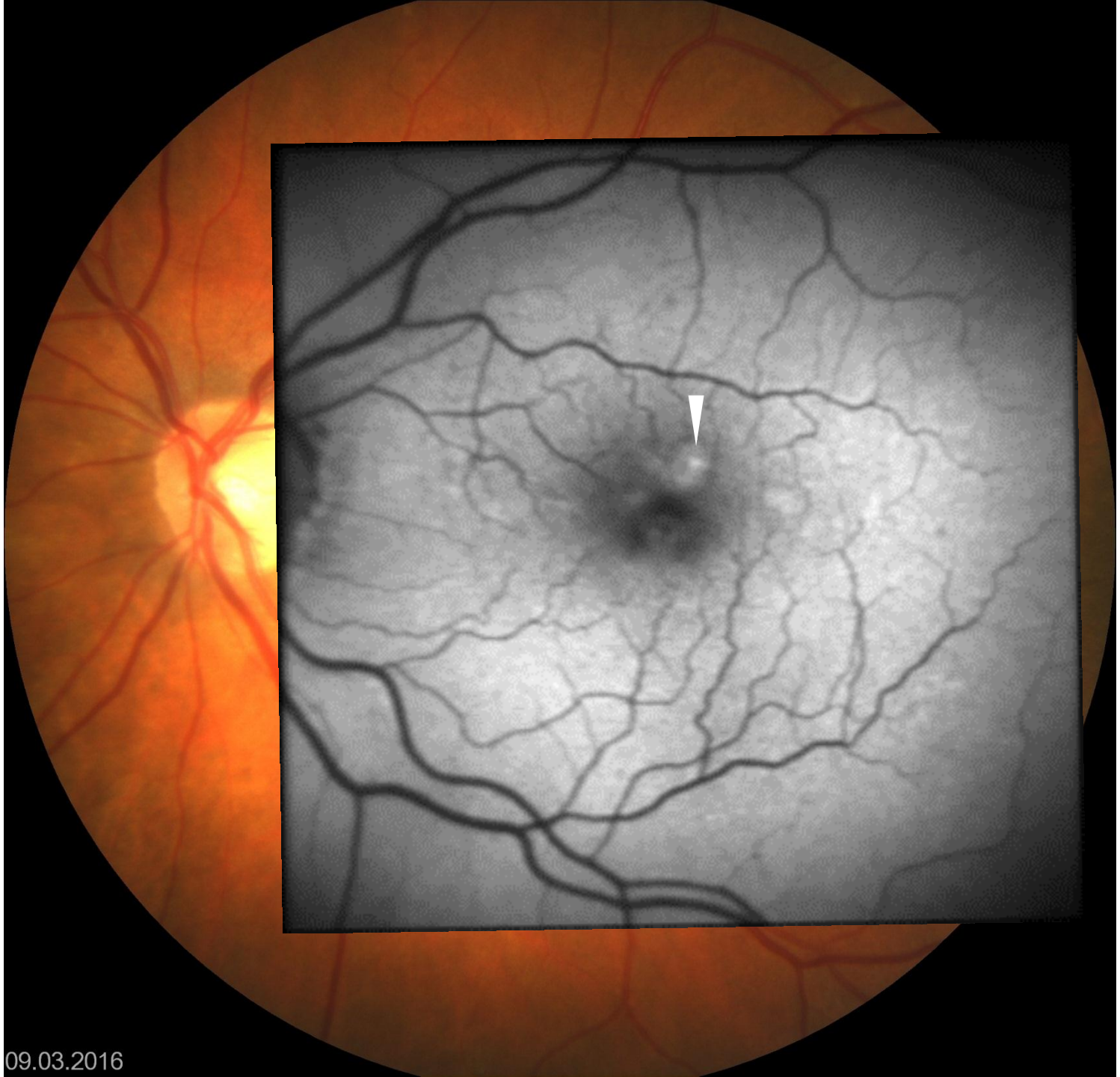
IR

03/09/2016

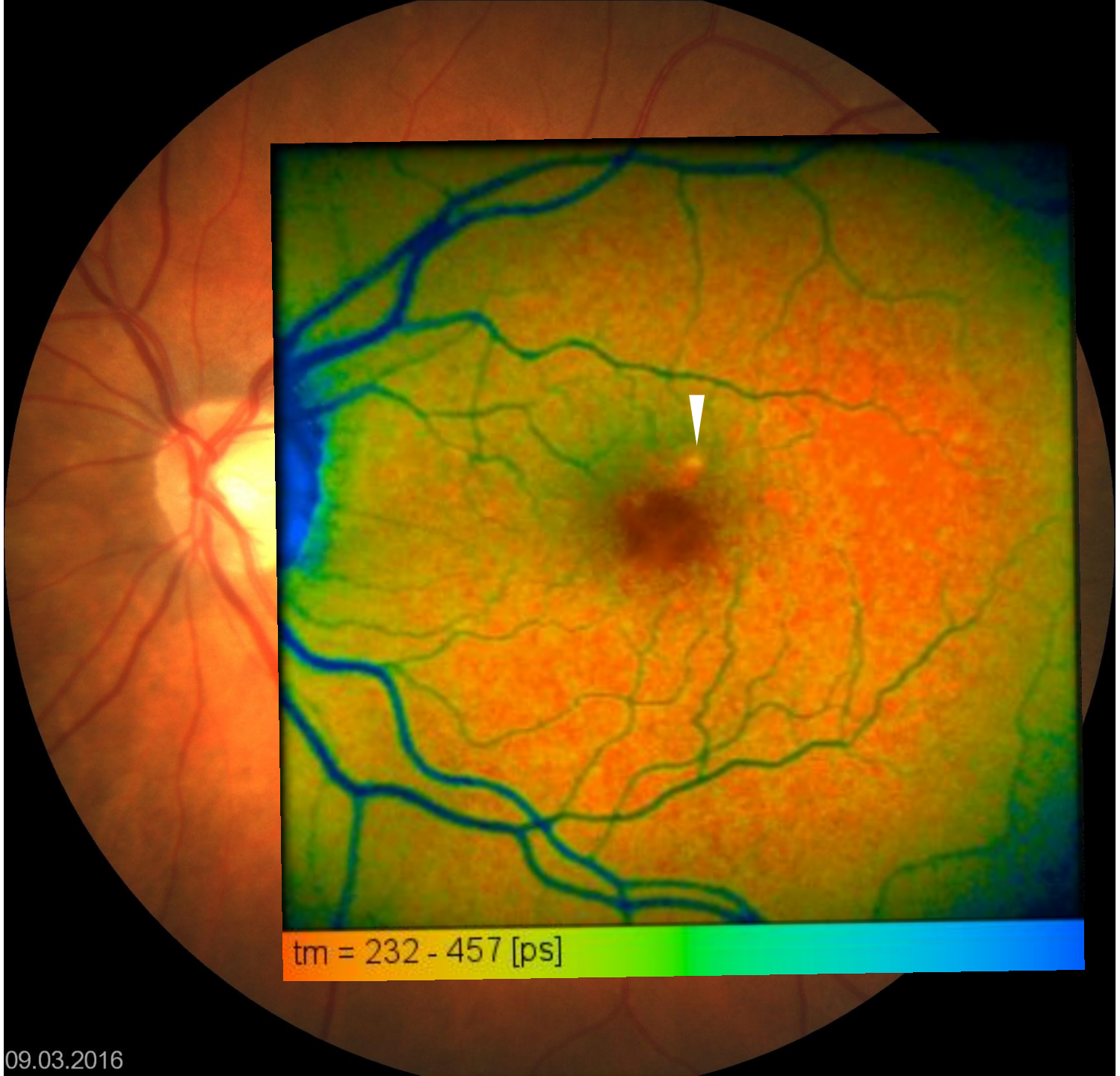


FAF SSC

03/09/2016

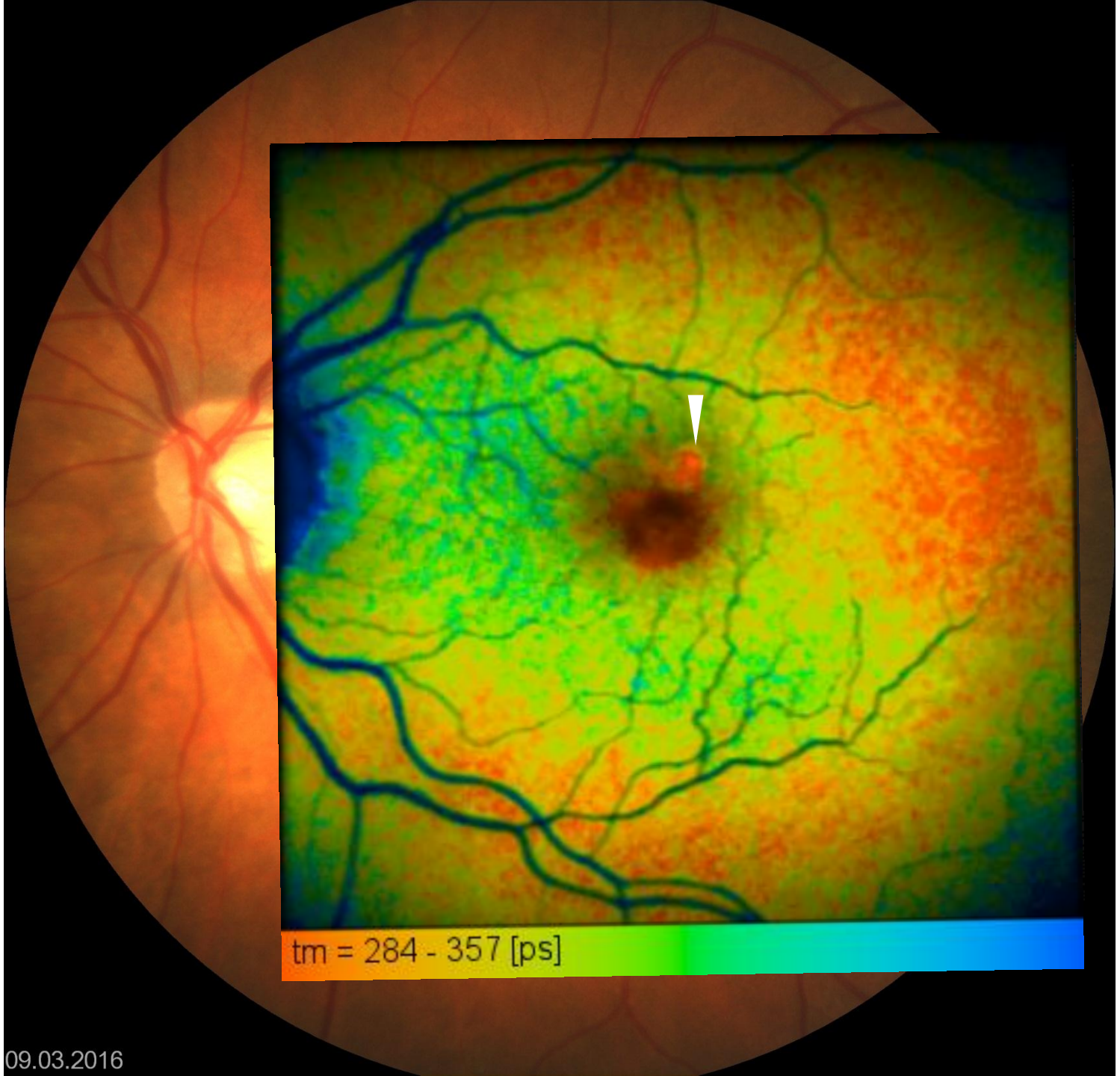


FAF LSC



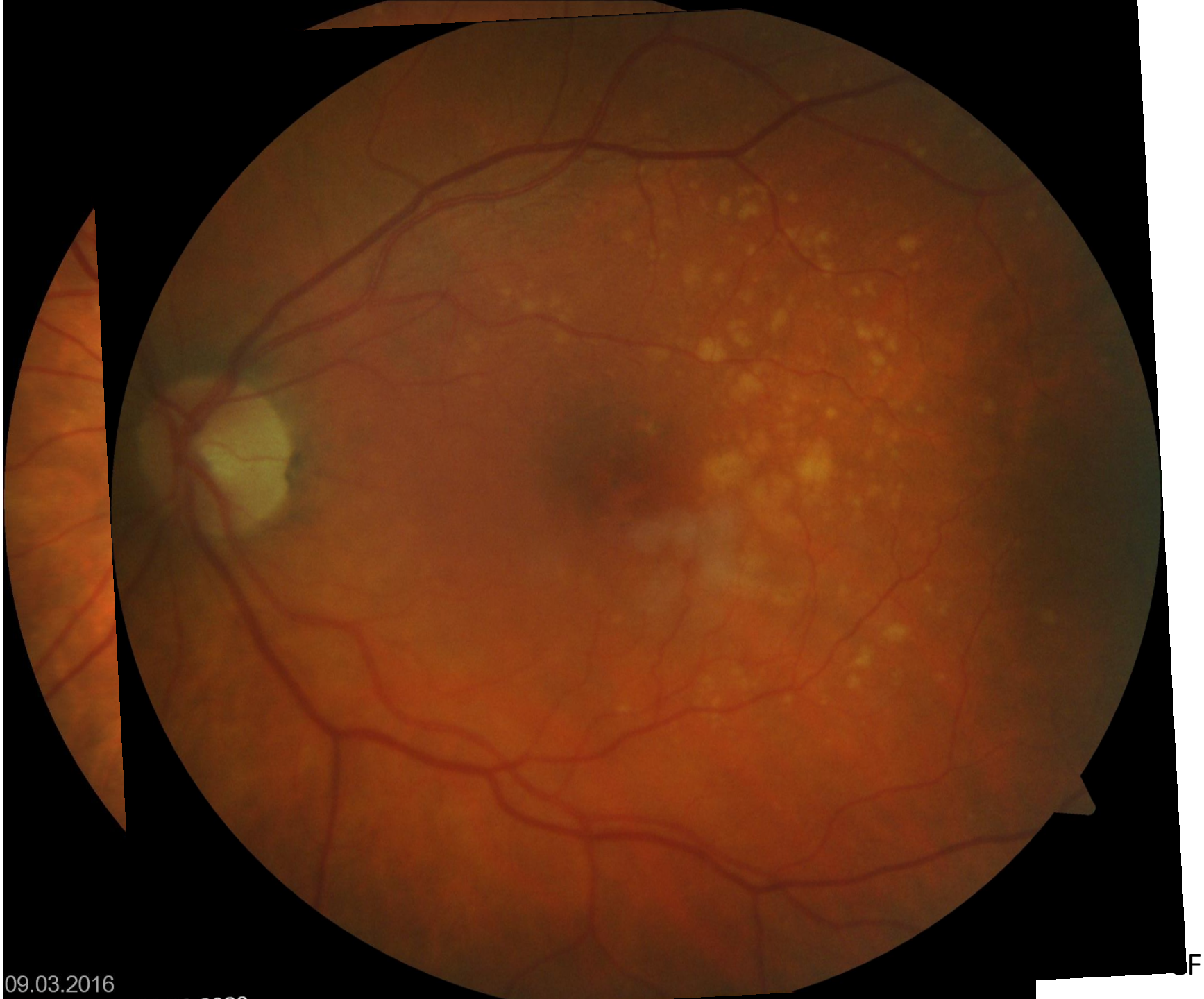
FLIO SSC

03/09/2016



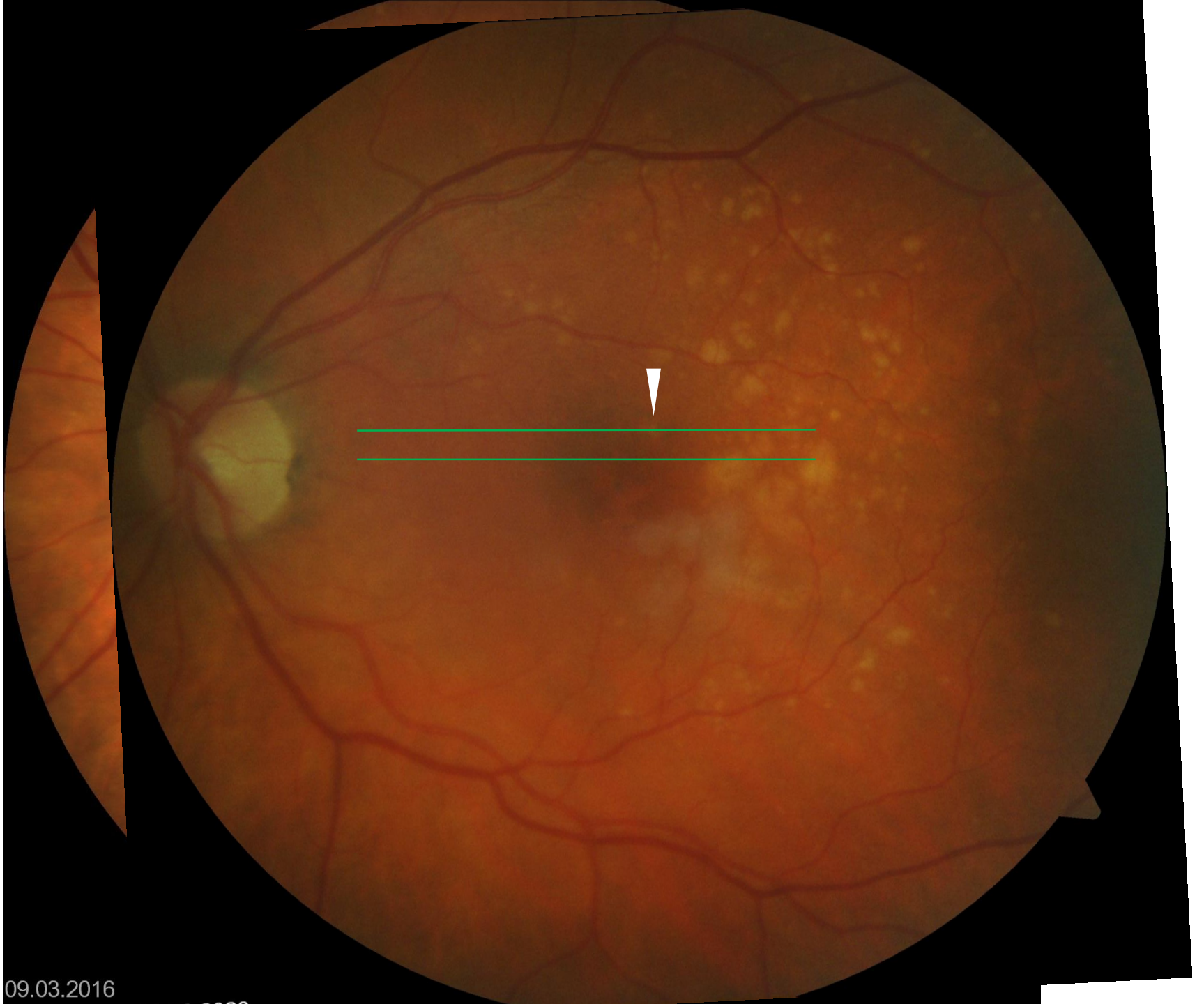
FLIO LSC

08/06/2020



FP

08/06/2020

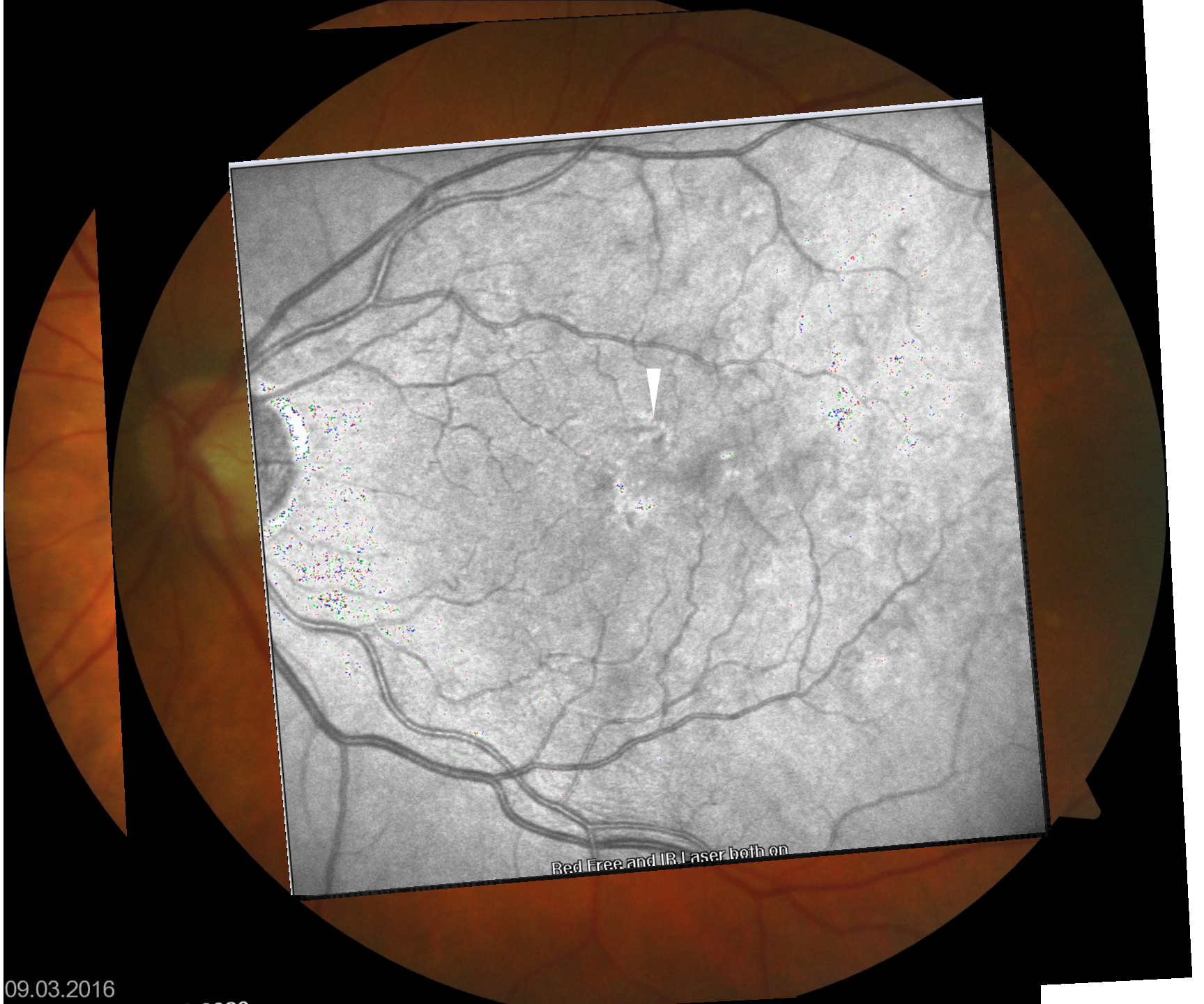


CFP

08/06/2020

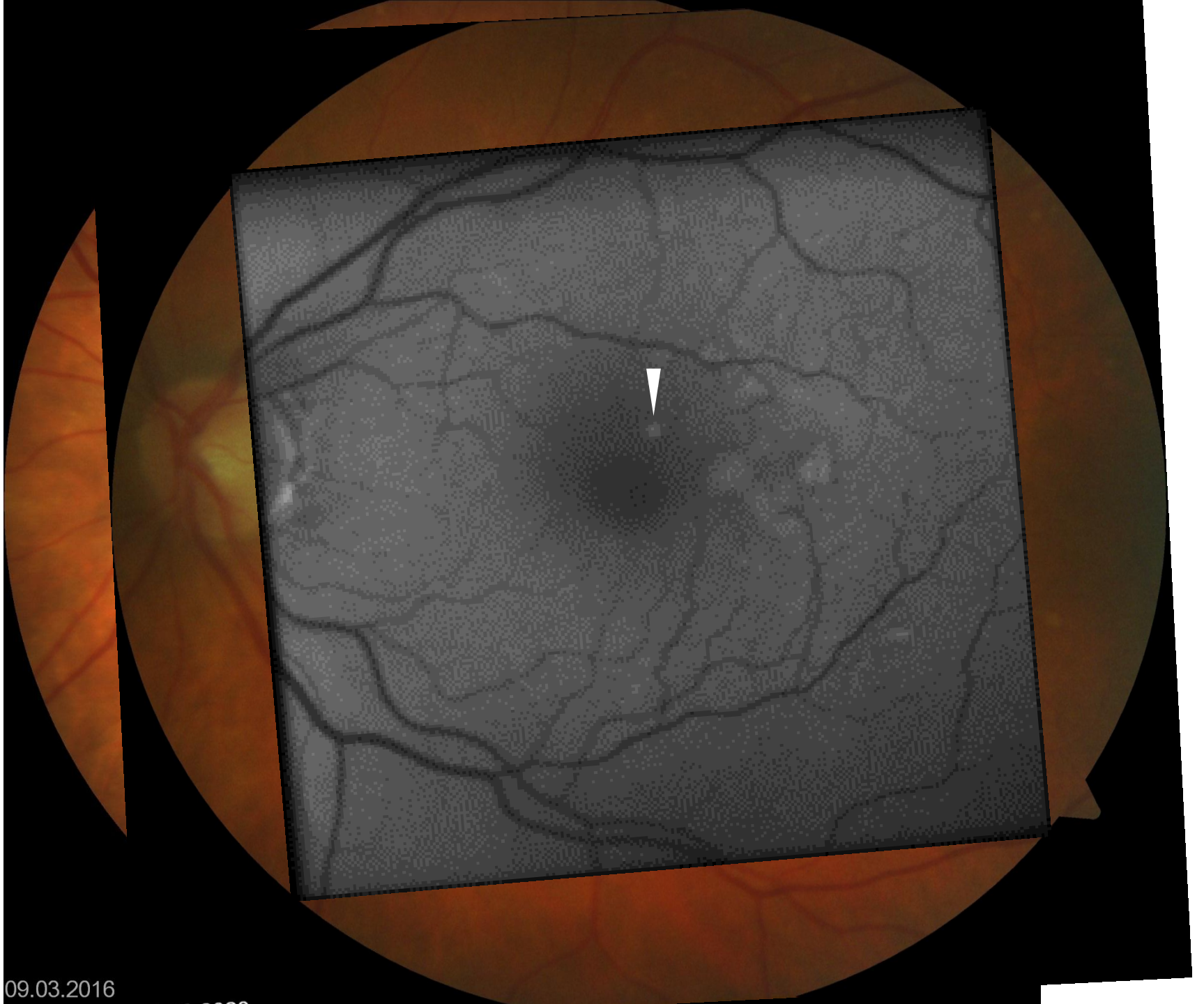
OCT at green lines in CFP

08/06/2020



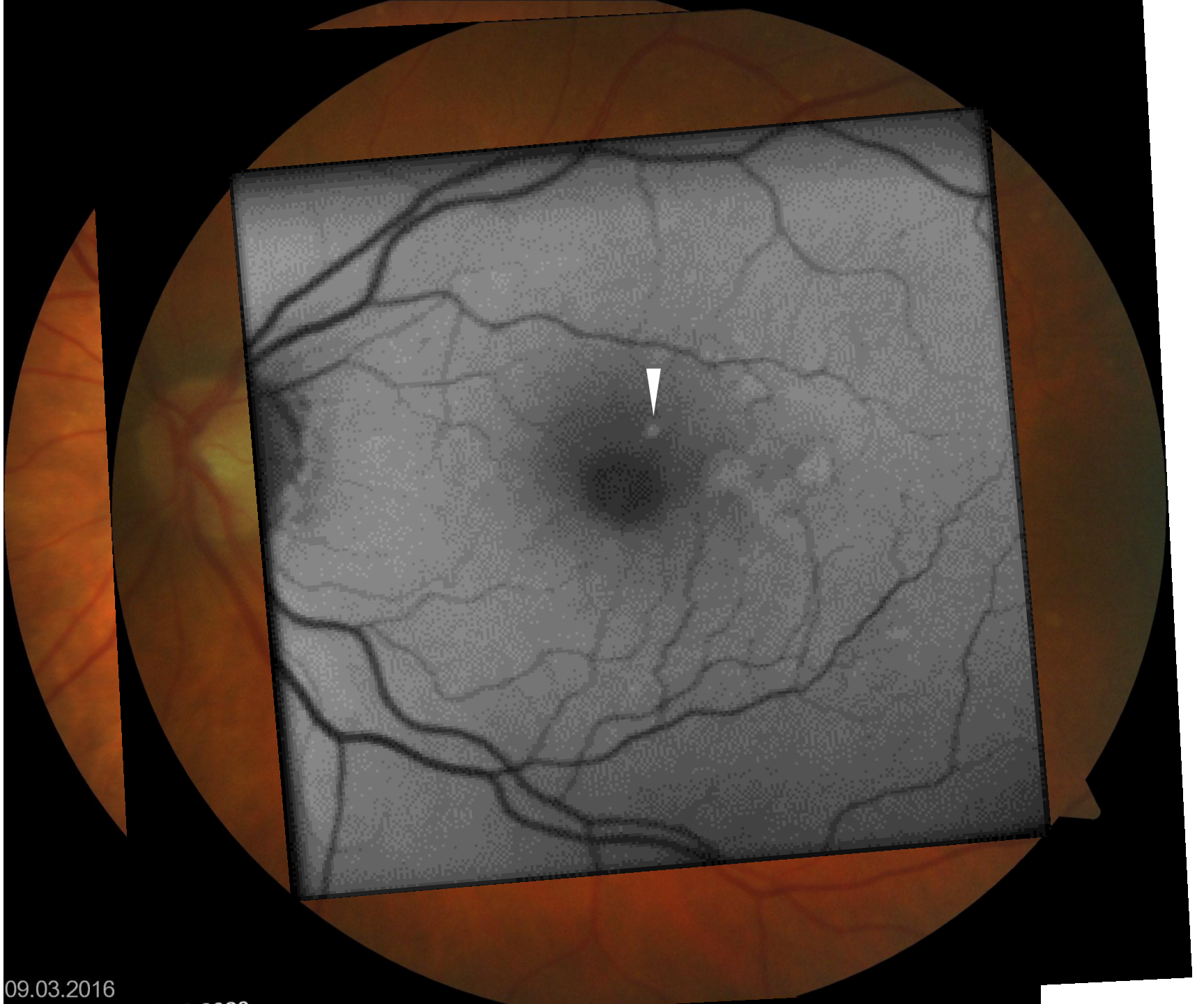
IR

08/06/2020



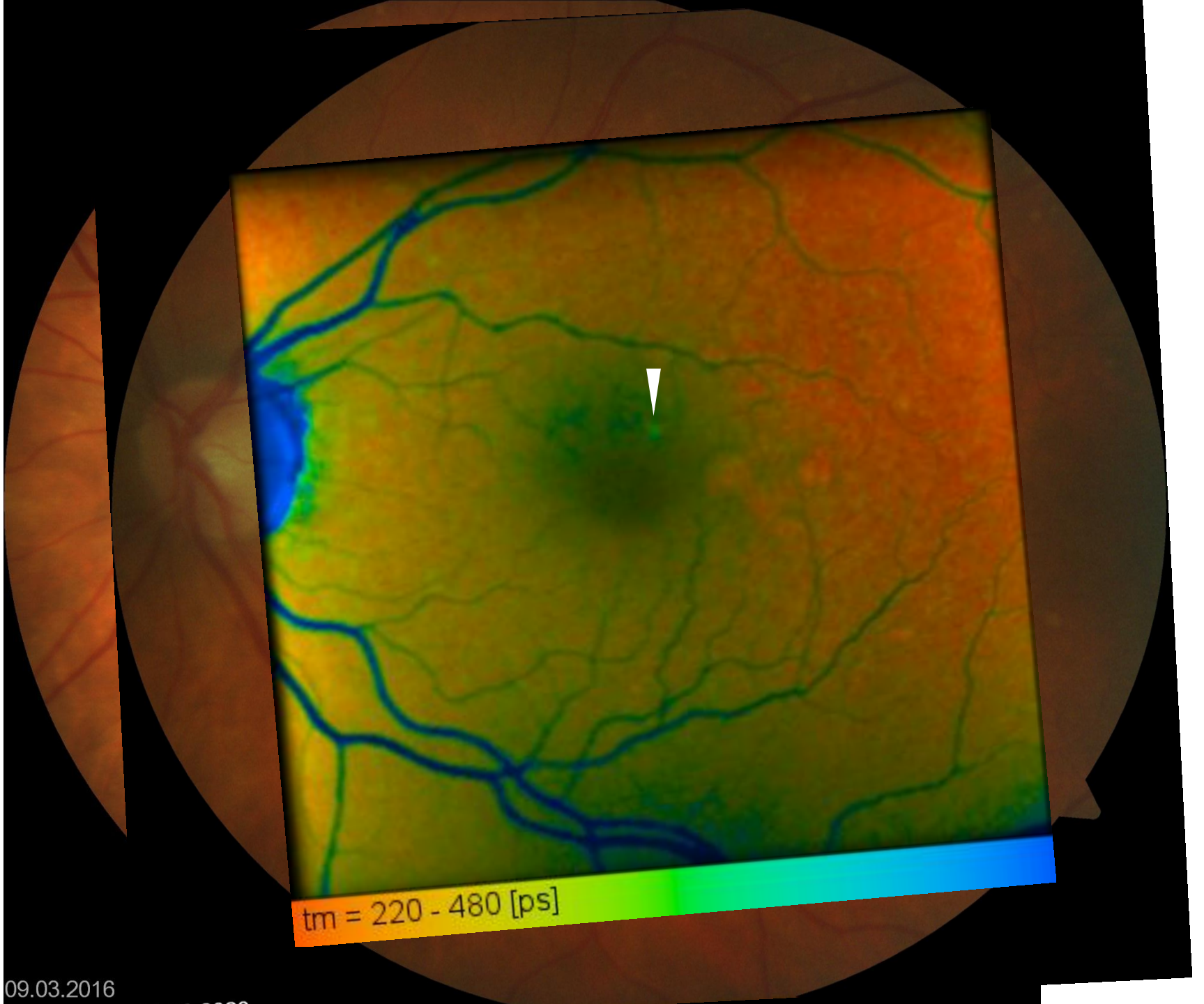
FAF SSC

08/06/2020

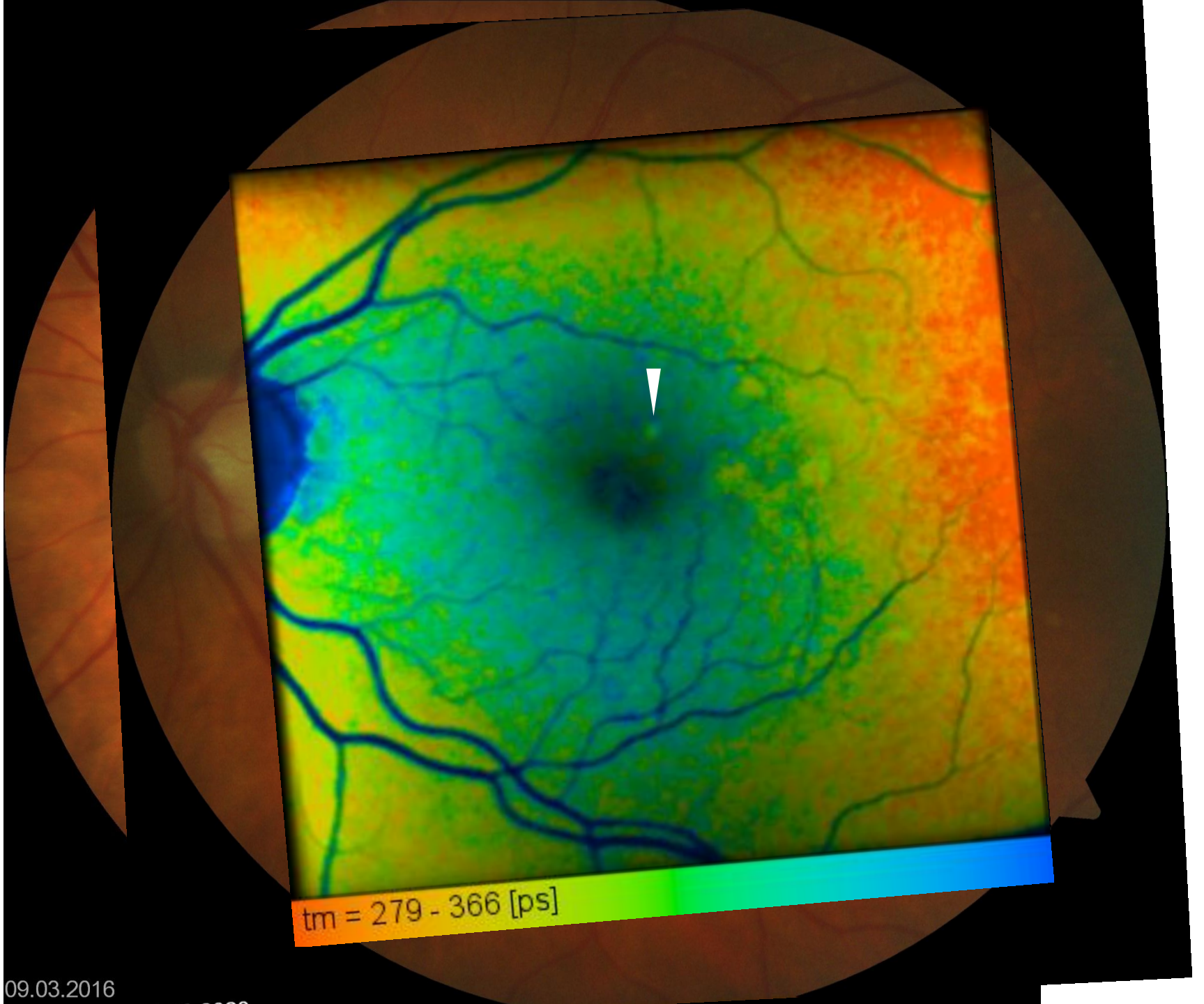


FAF LSC

08/06/2020



08/06/2020

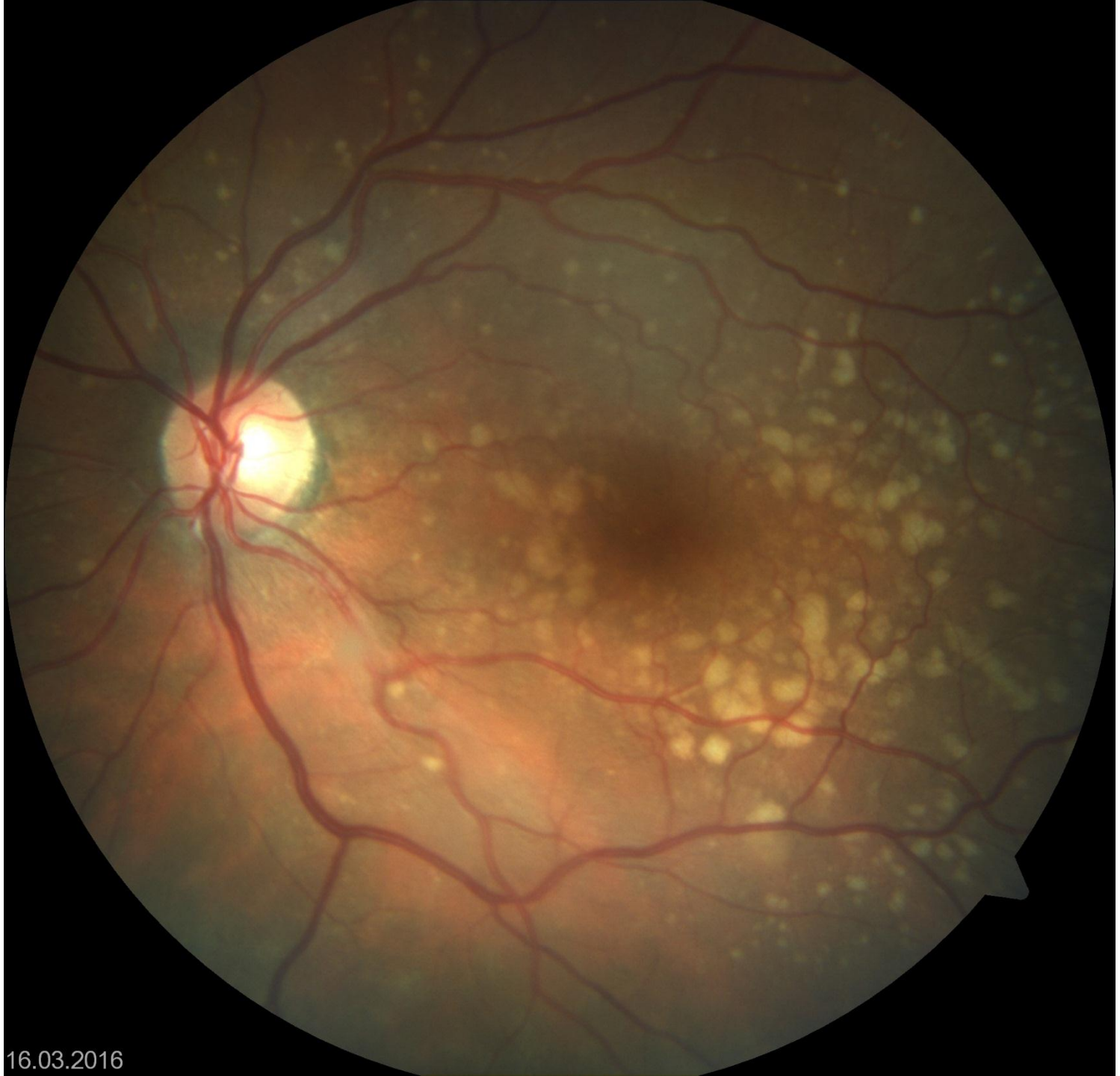


FLIO LSC

AMD_31, 65 years at baseline

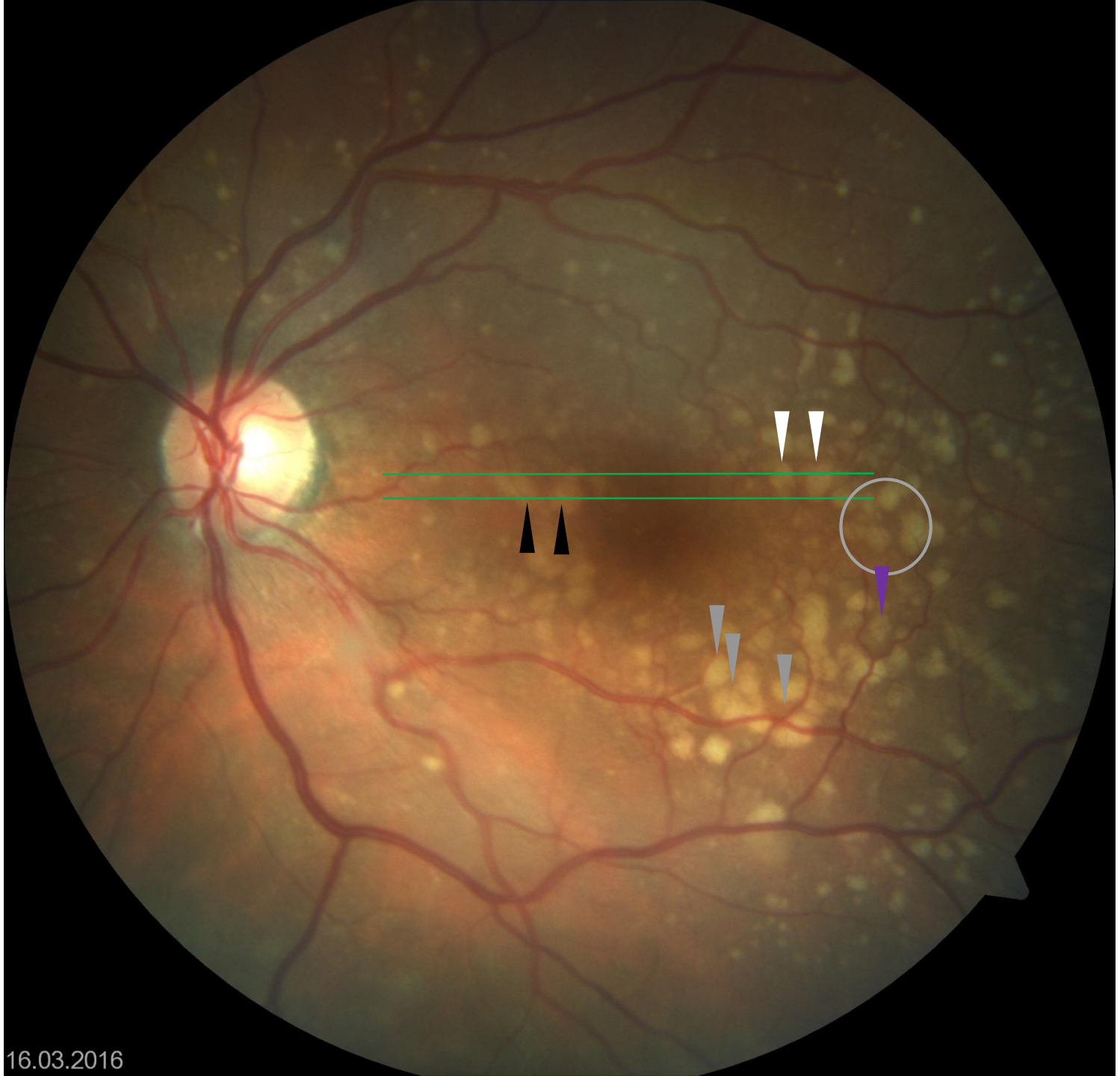
This is a very dynamic case. Some of the soft drusen disappeared over the follow up of 4 ½ years without signs of GA (circle). FAF lifetimes increased dramatically over this time (see color scaling of the FLIO images, lifetimes at baseline and follow up were too different to show them in a common scaling). We can exclude a lens effect, the patient was pseudophacic at baseline already. Prolongation of lifetime over the follow up also holds for areas where drusen disappeared. **Could it be that long lifetime is not due to drusen and BlinD** (what investigations in histology might suggest (Schultz et al, IOVS 61(2020), p.9)) **but to BlamD or RPE itself?** At baseline, we found hyperfluorescent (white arrowheads) and non-hyperfluorescent (black arrowheads) drusen which might correspond to drusen with and without hyperreflective material inside in OCT (although hard to tell from the low quality OCT, apologize for that). The non-fluorescent drusen became hyperfluorescent with rather long lifetimes at follow up. Some of the drusen developed very long lifetimes at follow up (grey arrowheads). No obvious hyperpigmentation was found there in CFP, however, OCT showed small hyperreflective foci. **Again: might focal prolongation of lifetimes indicate RPE changes already before formation of hyper-pigmentation?** At follow up, we can distinguish white and yellow drusen in CFP. White drusen showed large RPE elevations without reflective material inside in OCT. We could unabigeouly identify one of the yellow drusen (purple arrowhead) which was small in OCT but was rather unremarkable in FAF. **What could be the reason for different color in CFP?**

03/16/2016



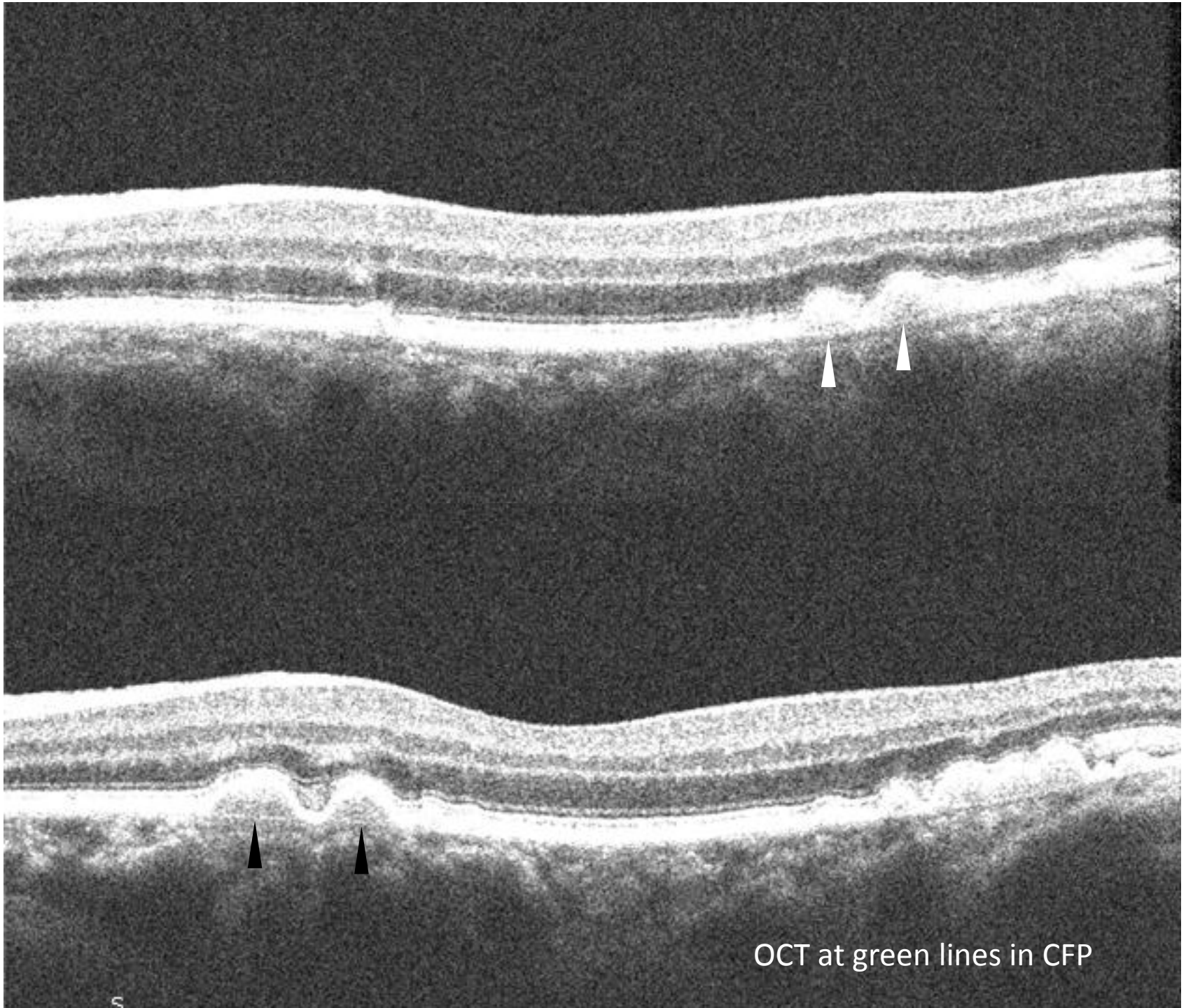
CFP

03/16/2016



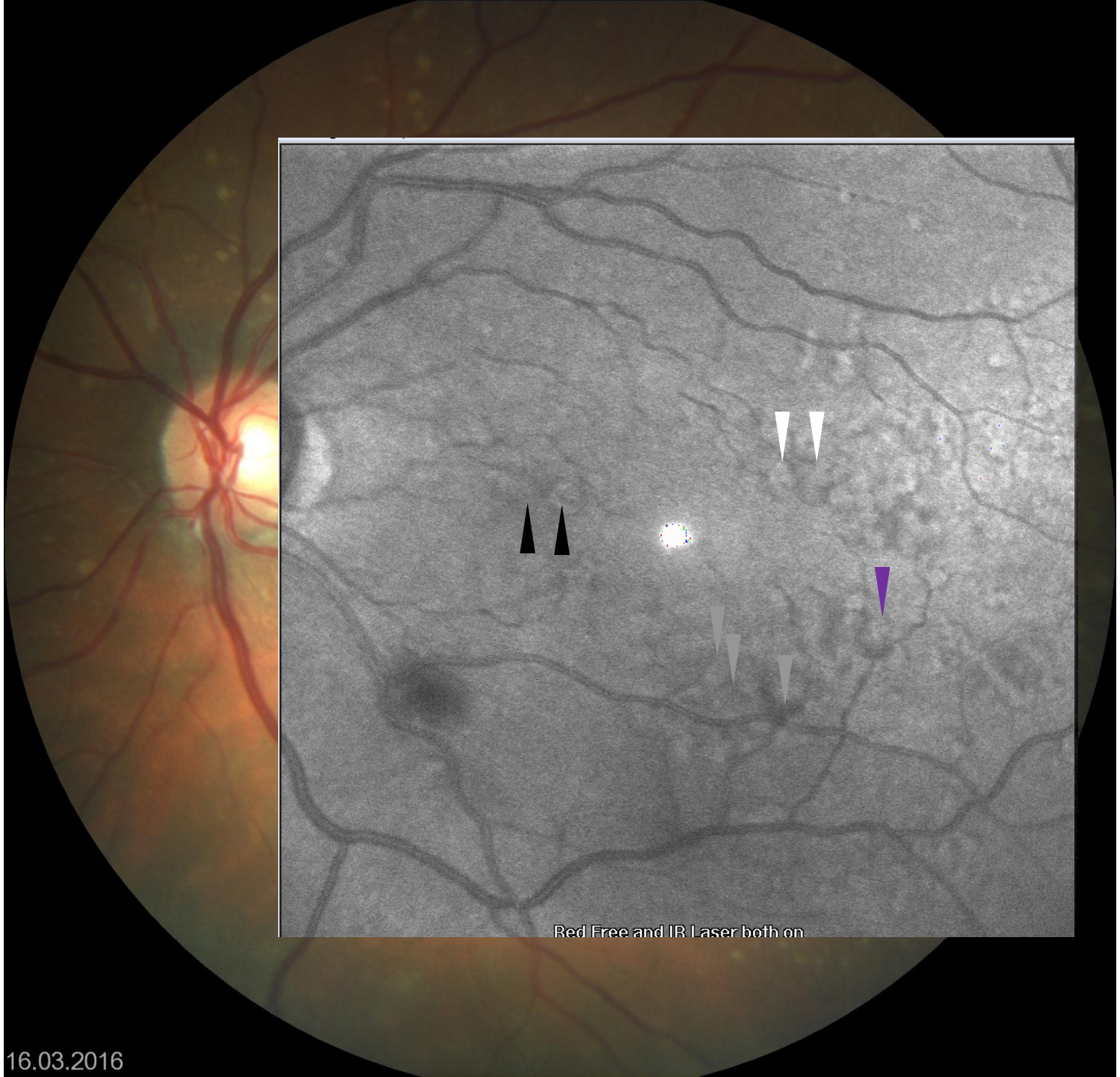
CFP

03/16/2016



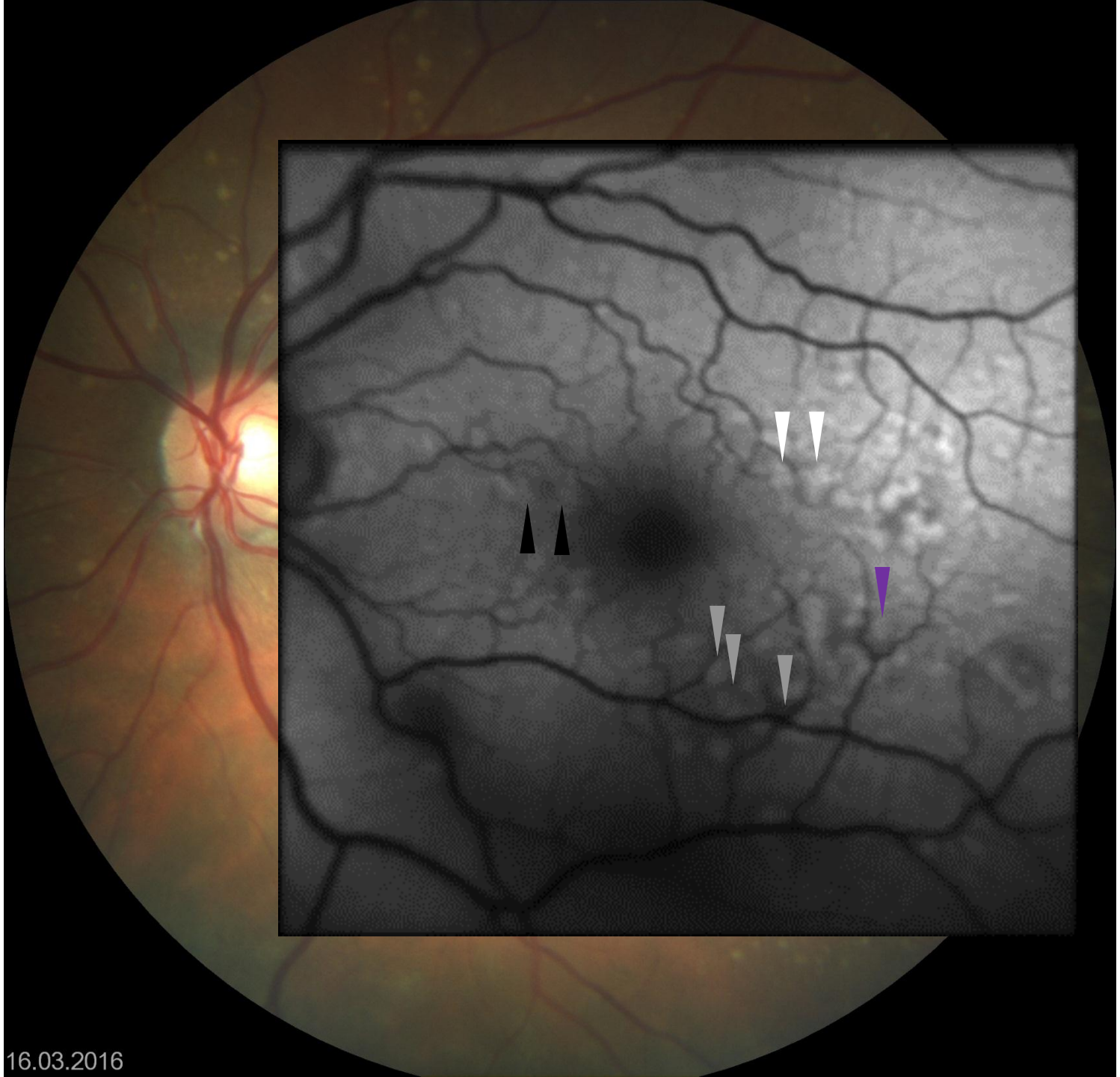
OCT at green lines in CFP

03/16/2016



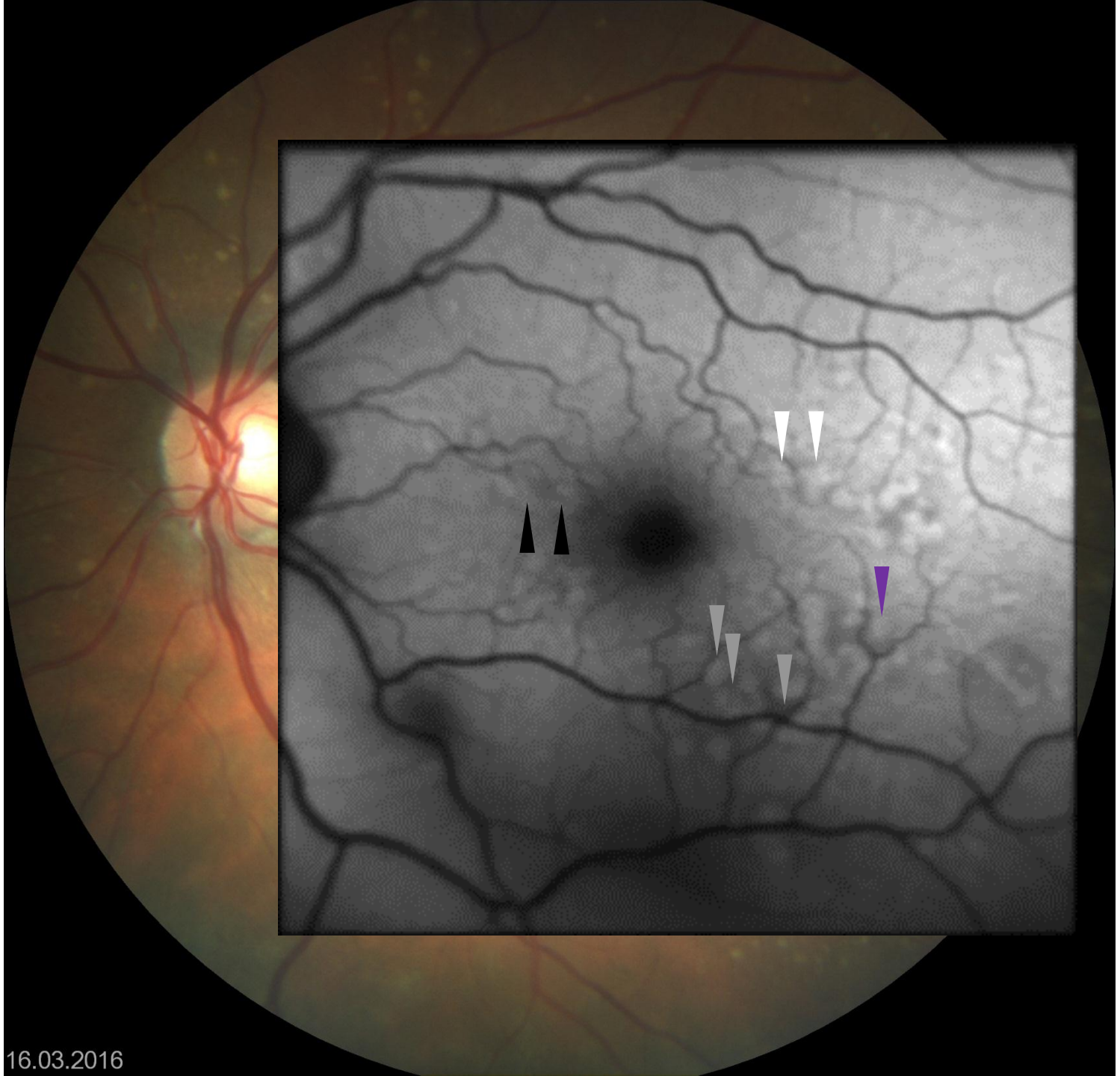
IR

03/16/2016



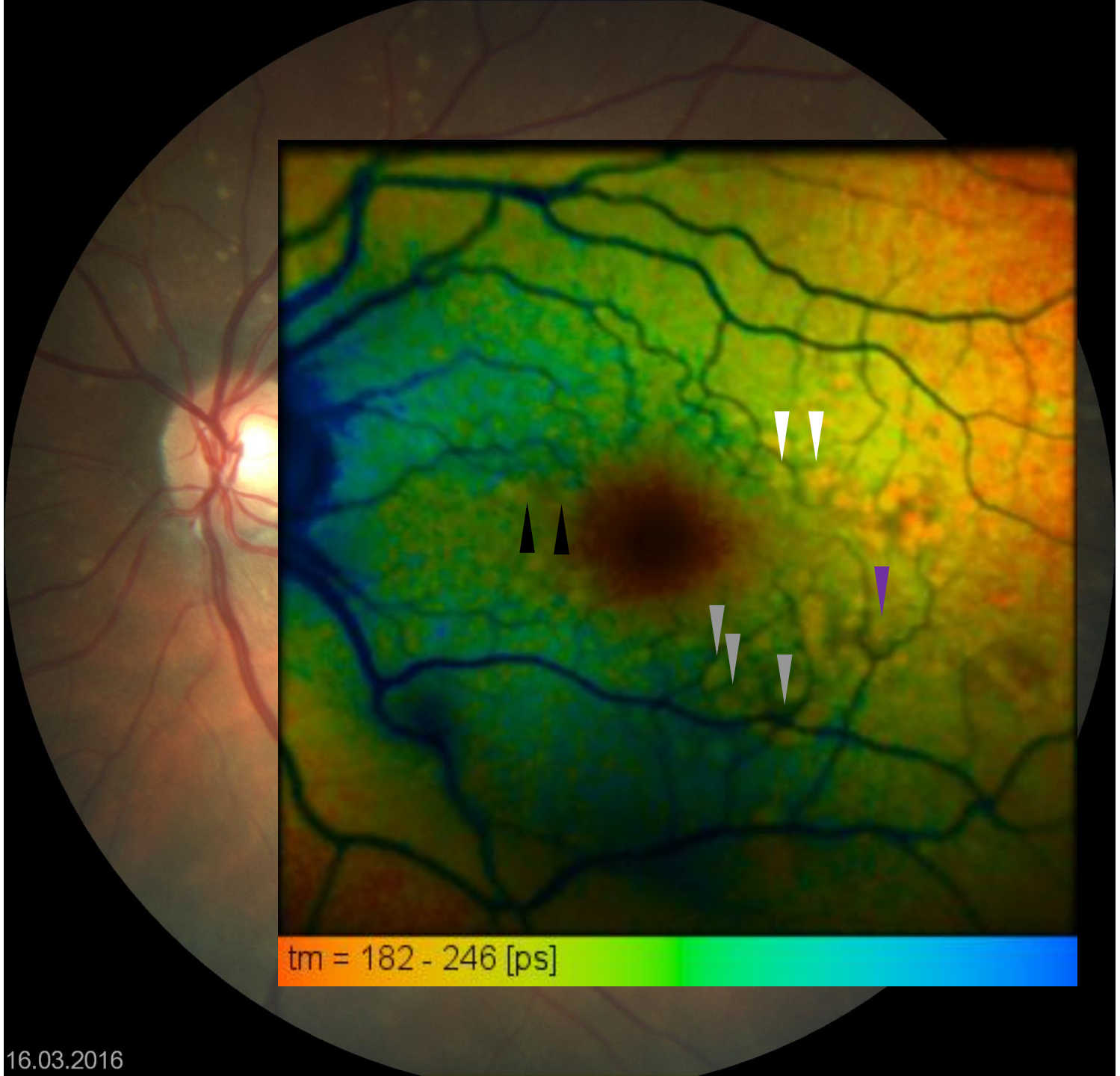
FAF SSC

03/16/2016



FAF LSC

03/16/2016



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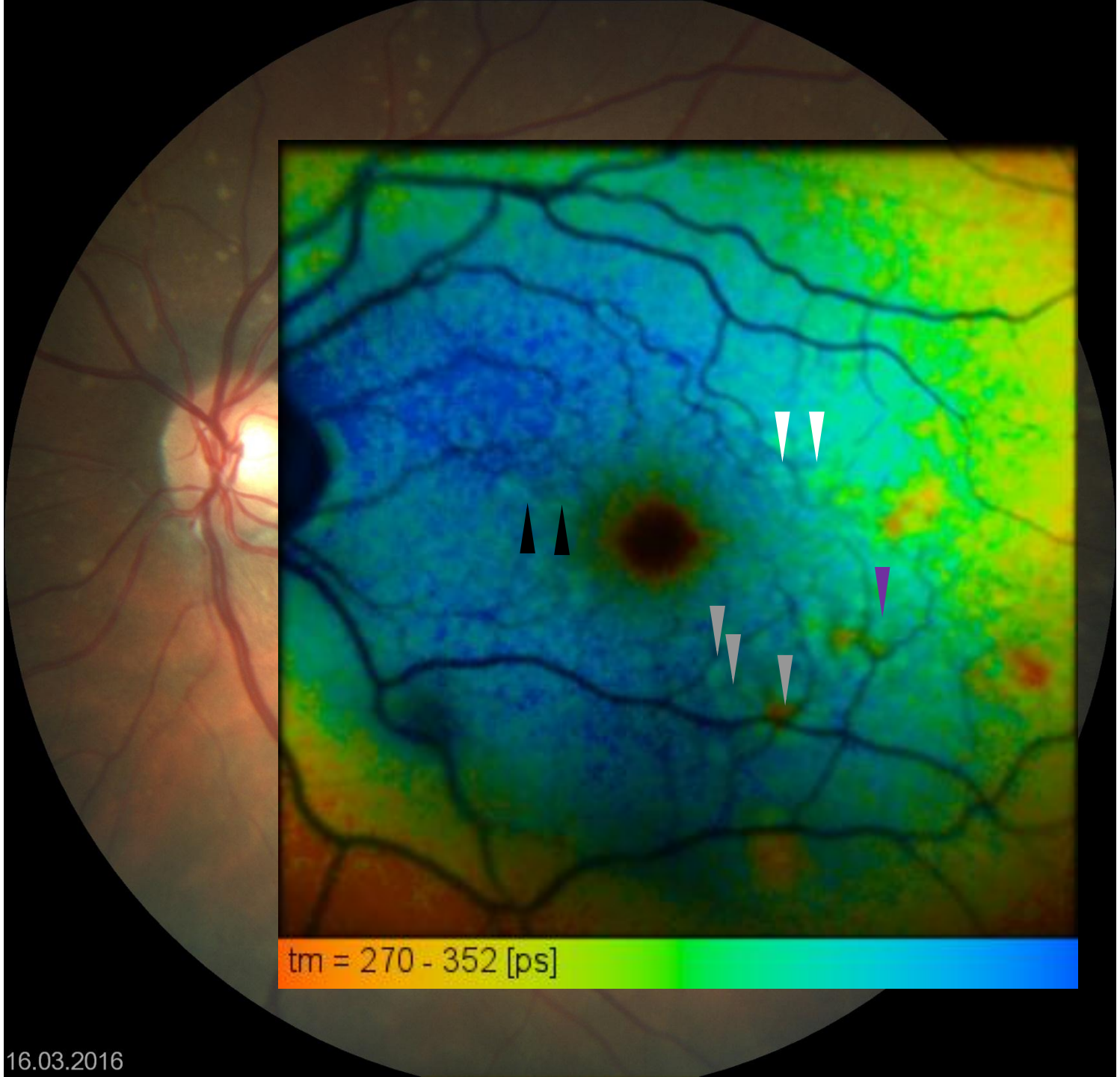
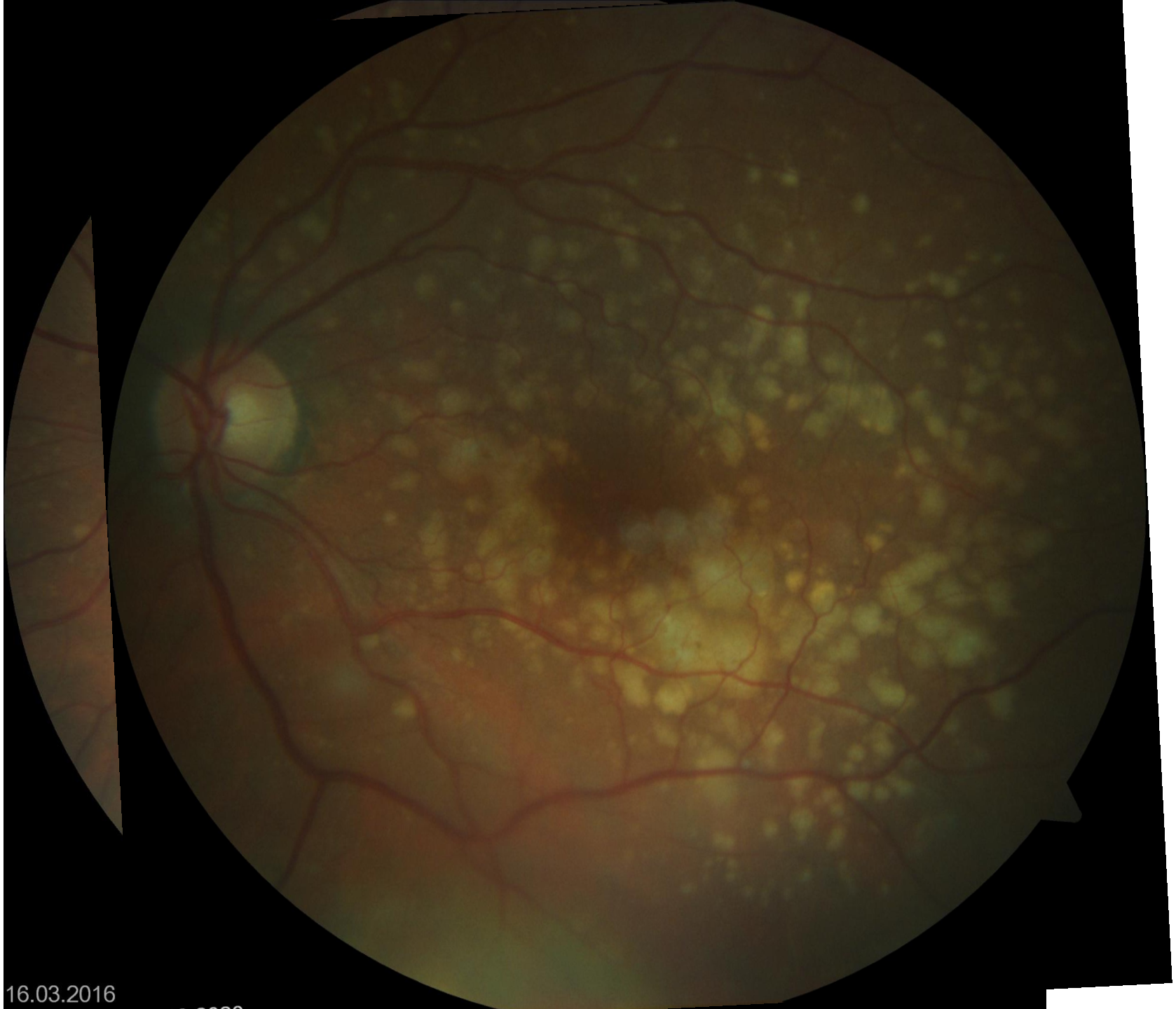
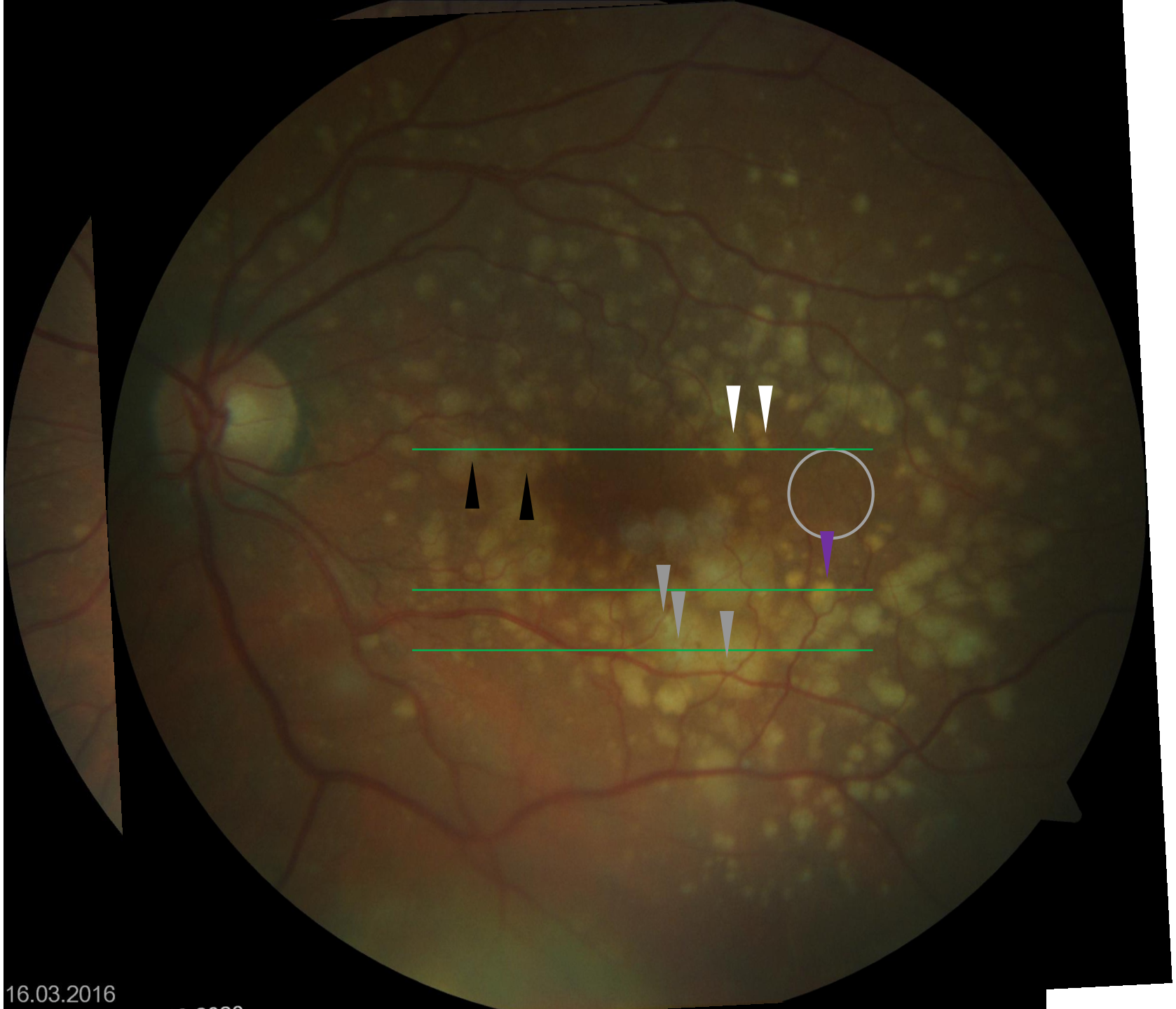


08/12/2020



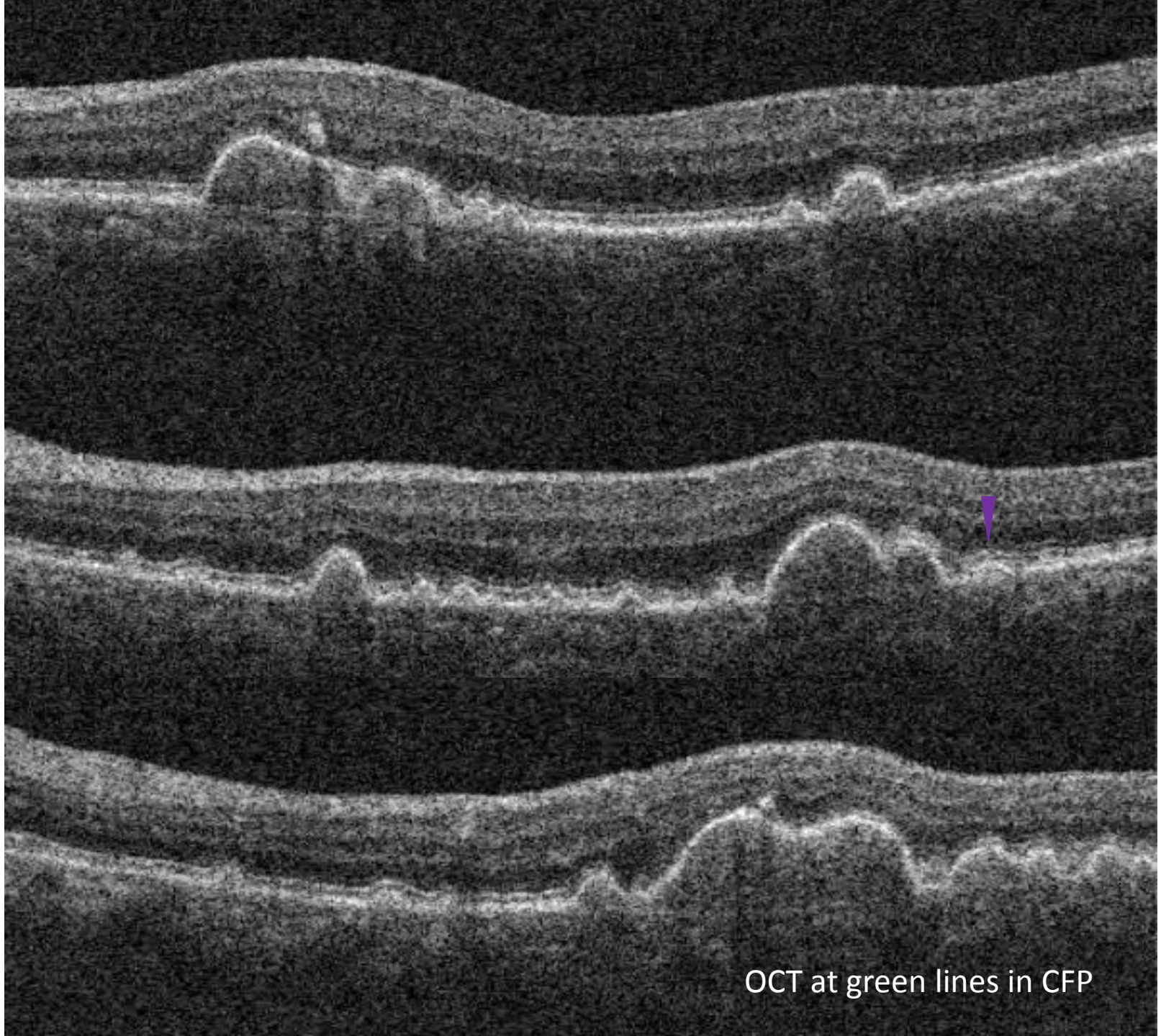
CFP

08/12/2020



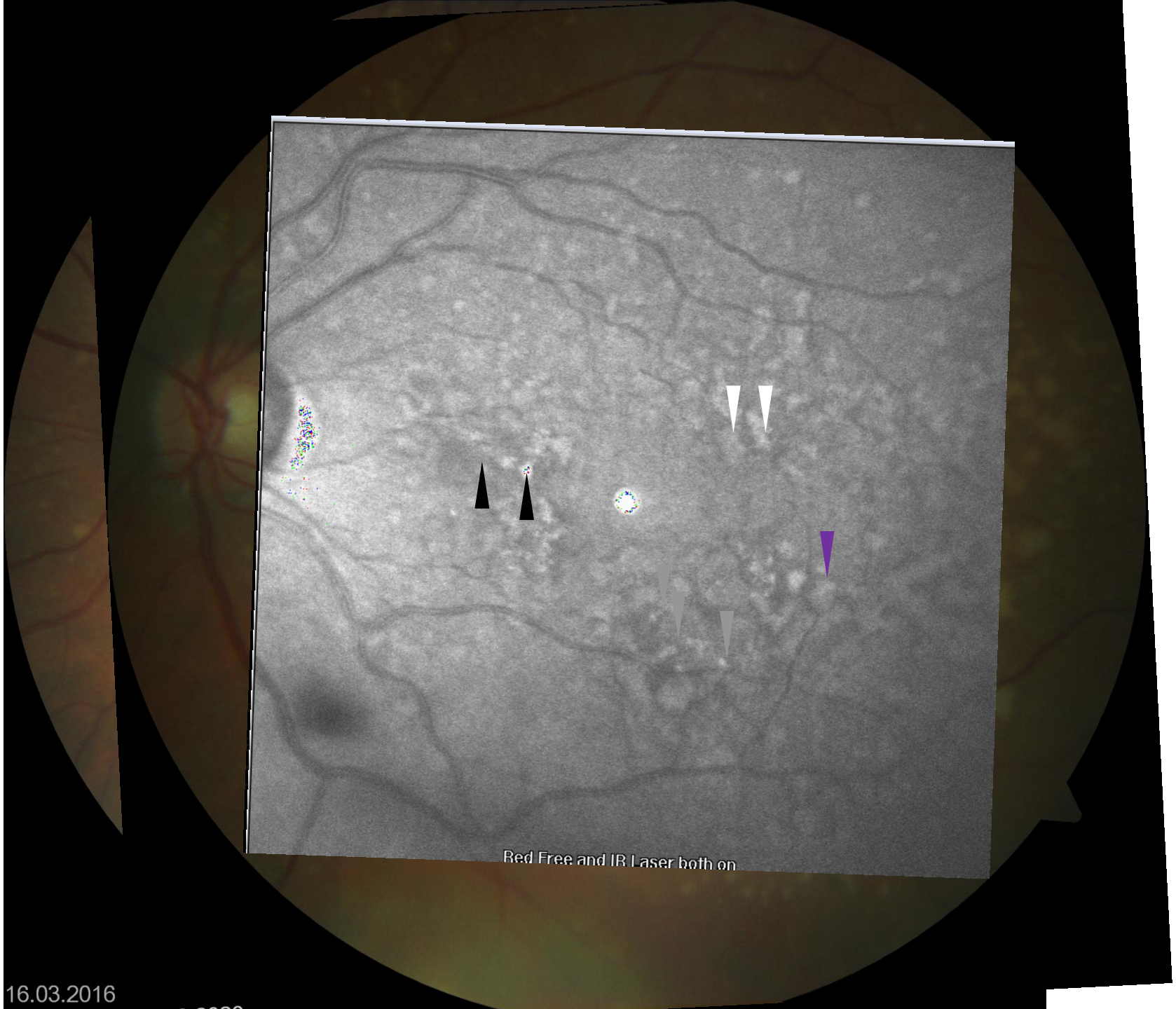
CFP

08/12/2020

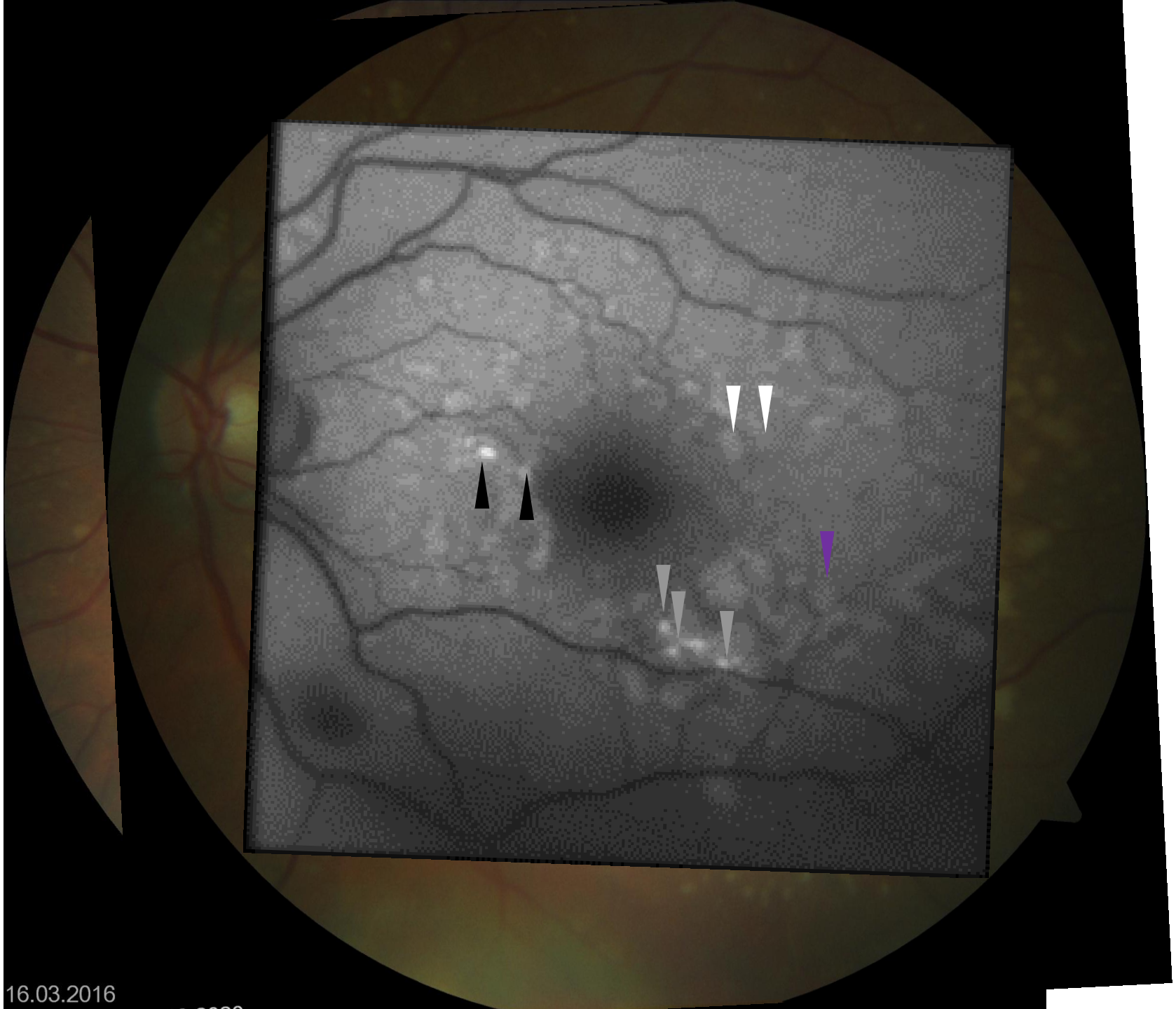


OCT at green lines in CFP

08/12/2020

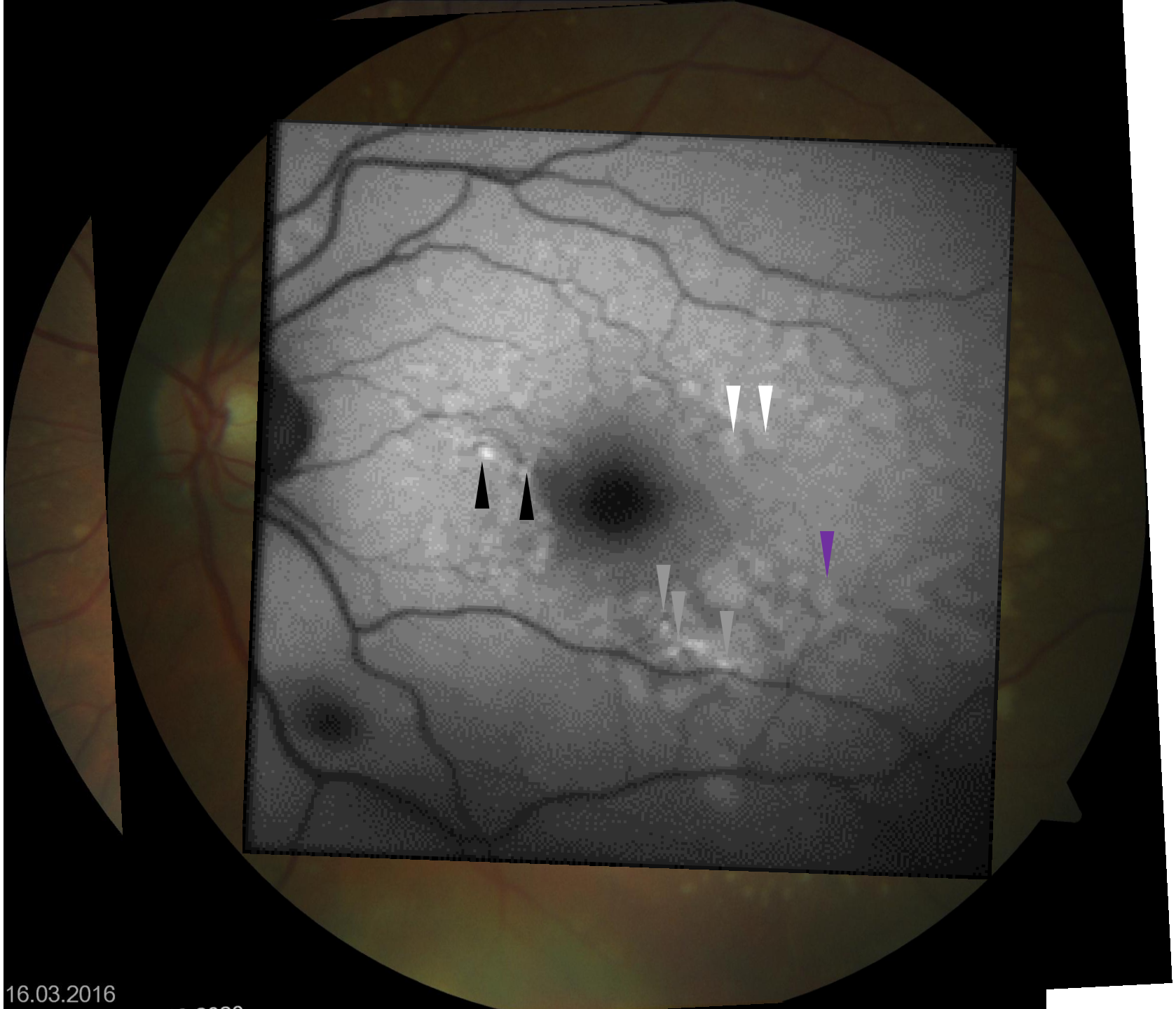


IR



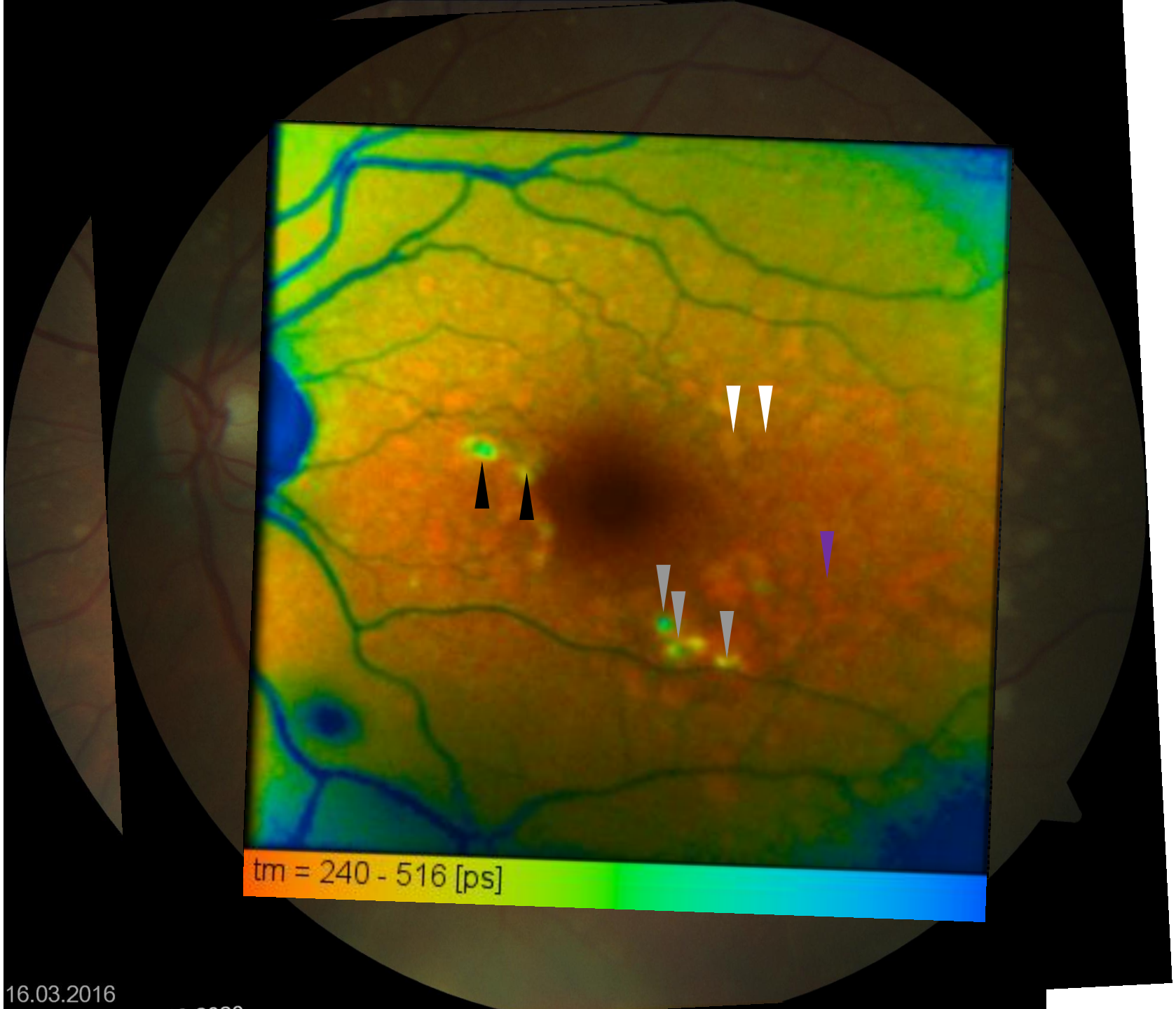
FAF SSC

08/12/2020

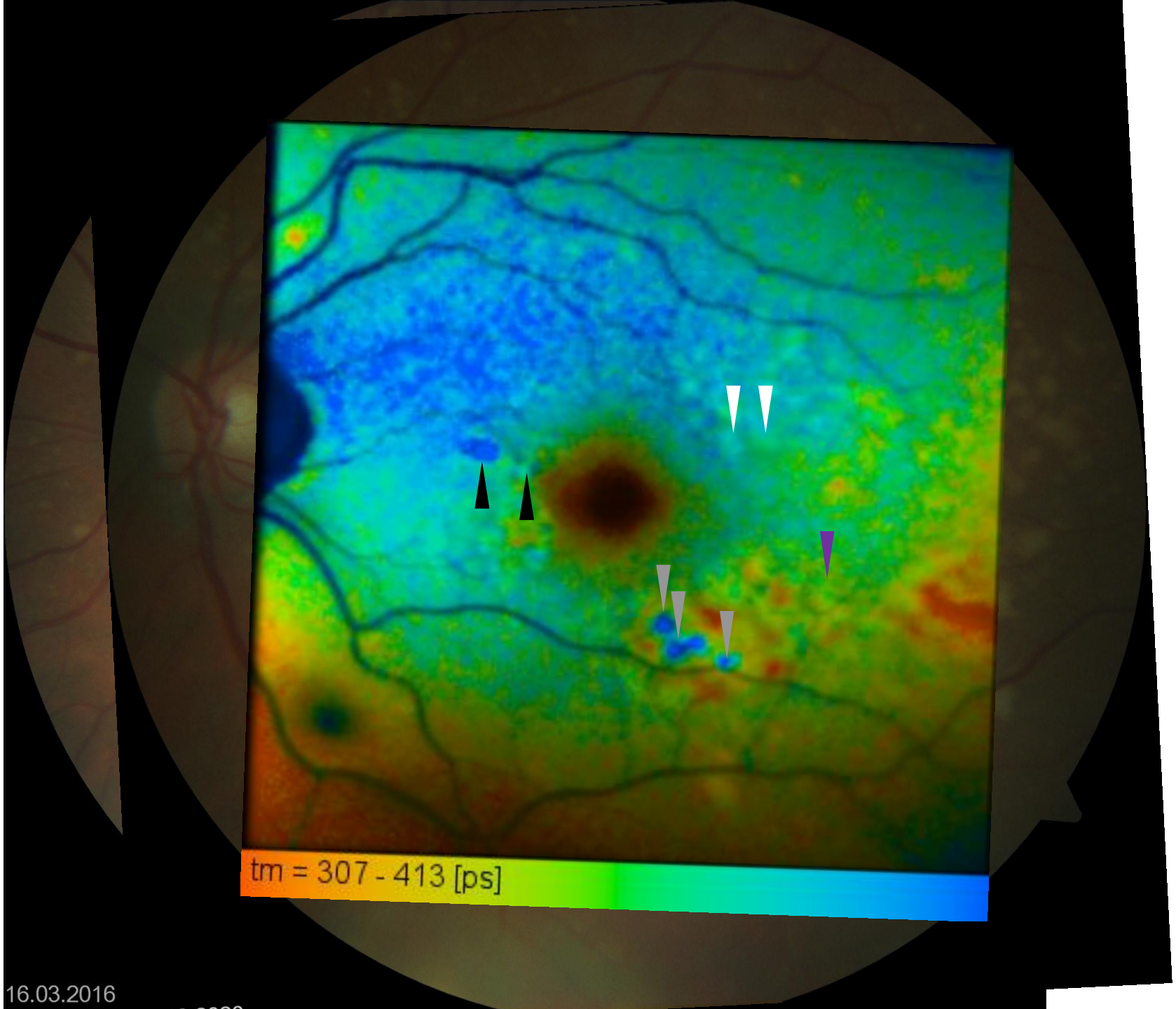


FAF LSC

08/12/2020



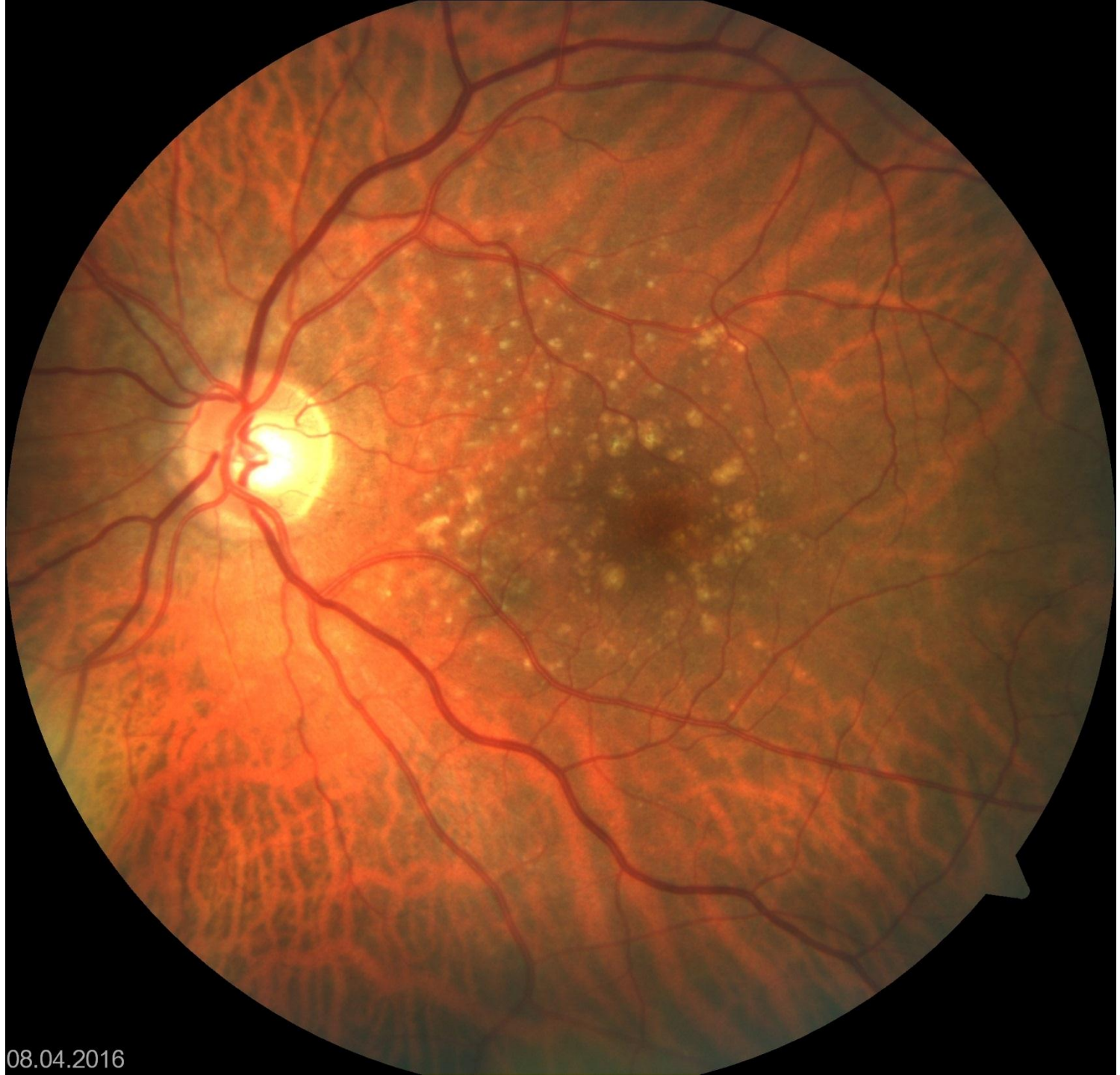
08/12/2020



AMD_39, 77 years at baseline

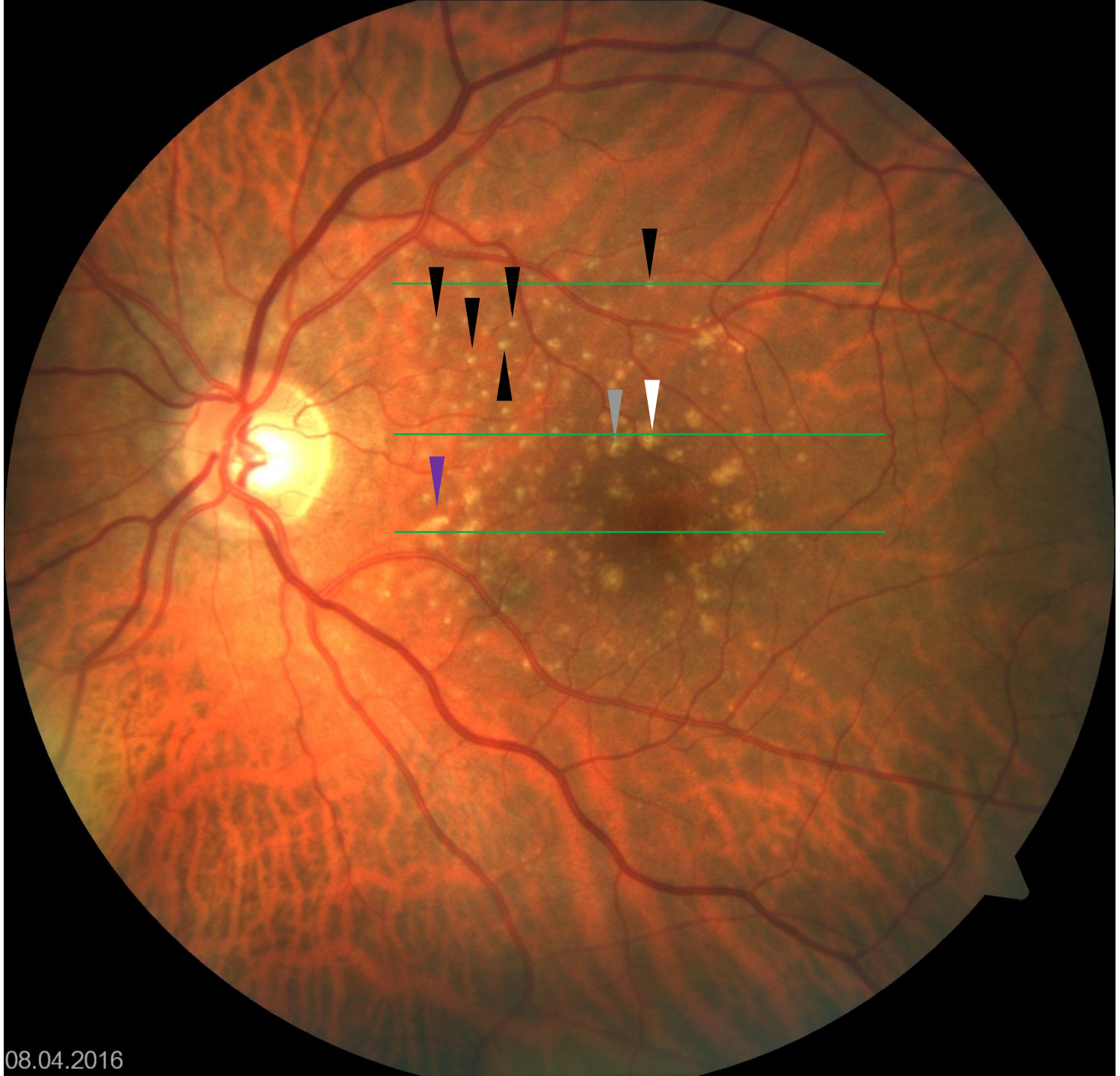
At this patient, I found the following remarkable: 1. There were SDD which were hypofluorescent at baseline (as we know them), but showed a hyperfluorescent center in the follow up (most pronounced at 19 months, black arrow heads). OCT shows SDD which disrupt the ellipsoid zone (EZ). These might be associated with hyperfluorescence, but we can't be sure because assignment of OCT scans to FAF images was difficult. **Has anyone seen that before or any explanation?** 2. There is one druse (purple arrowhead) which became hyperfluorescent in the follow ups only, however, showed long liftimes without hyperfluorescence at baseline already. **As there was no "additional FAF", does the long lifetime point to alterations in the RPE?** 3. At the last follow up, 52 months after baseline, there were massive hyperpigmentations showing long lifetimes as we found before. One druse, topped with hyperpigmentation (grey arrowhead, green in OCT), however, turned from long lifetime at basline and first follow up to short lifetime at 52 months. In contrast, a neighboring druse (white arrowhead) showed very long lifetime although no hyperpigmentation is seen in CFP. In general, drusen had long lifetimes in this case. **Is that due to drusen content or does it, eventually, indicate RPE changes prior to migration?**

04/08/2016



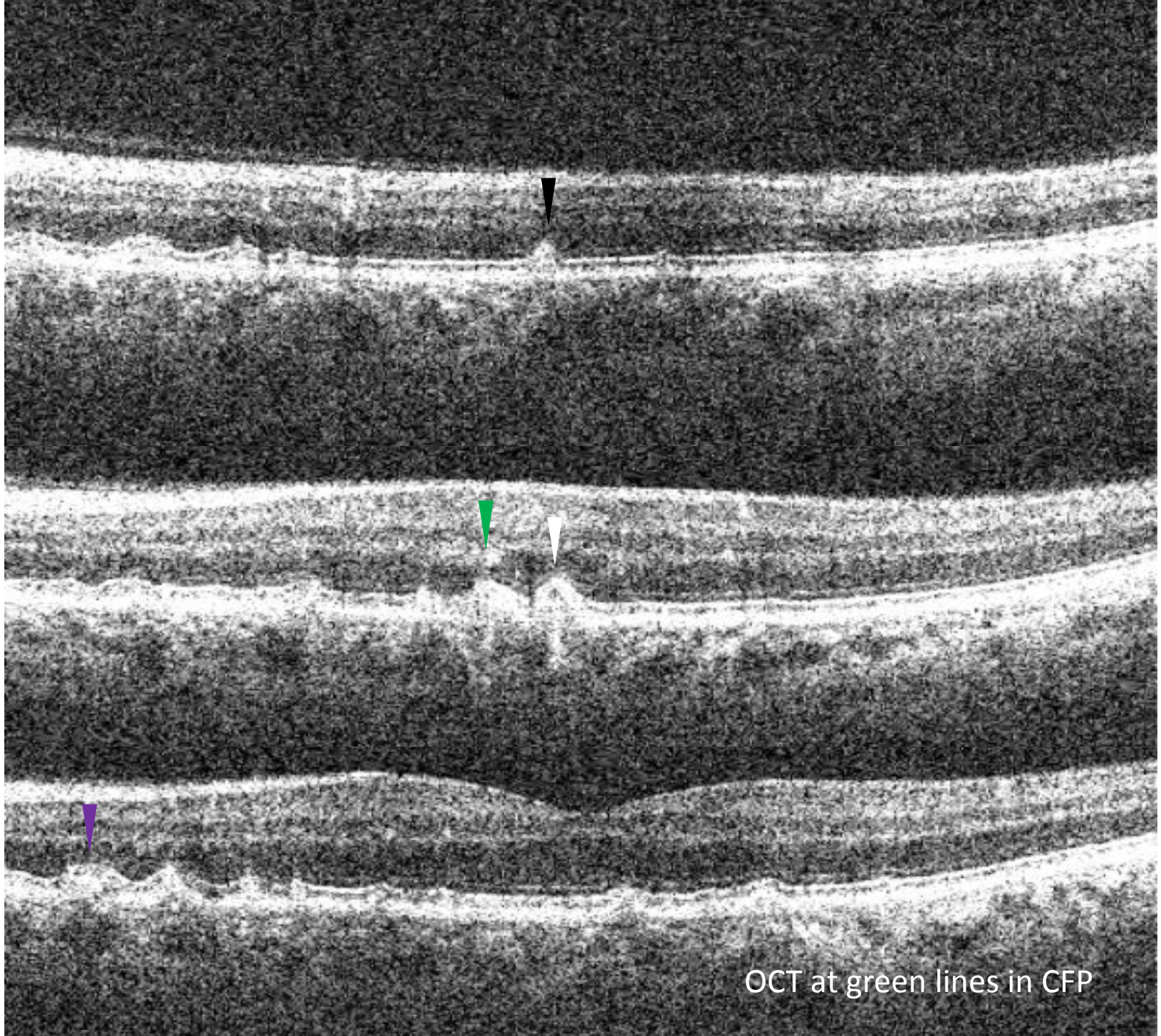
CFP

04/08/2016



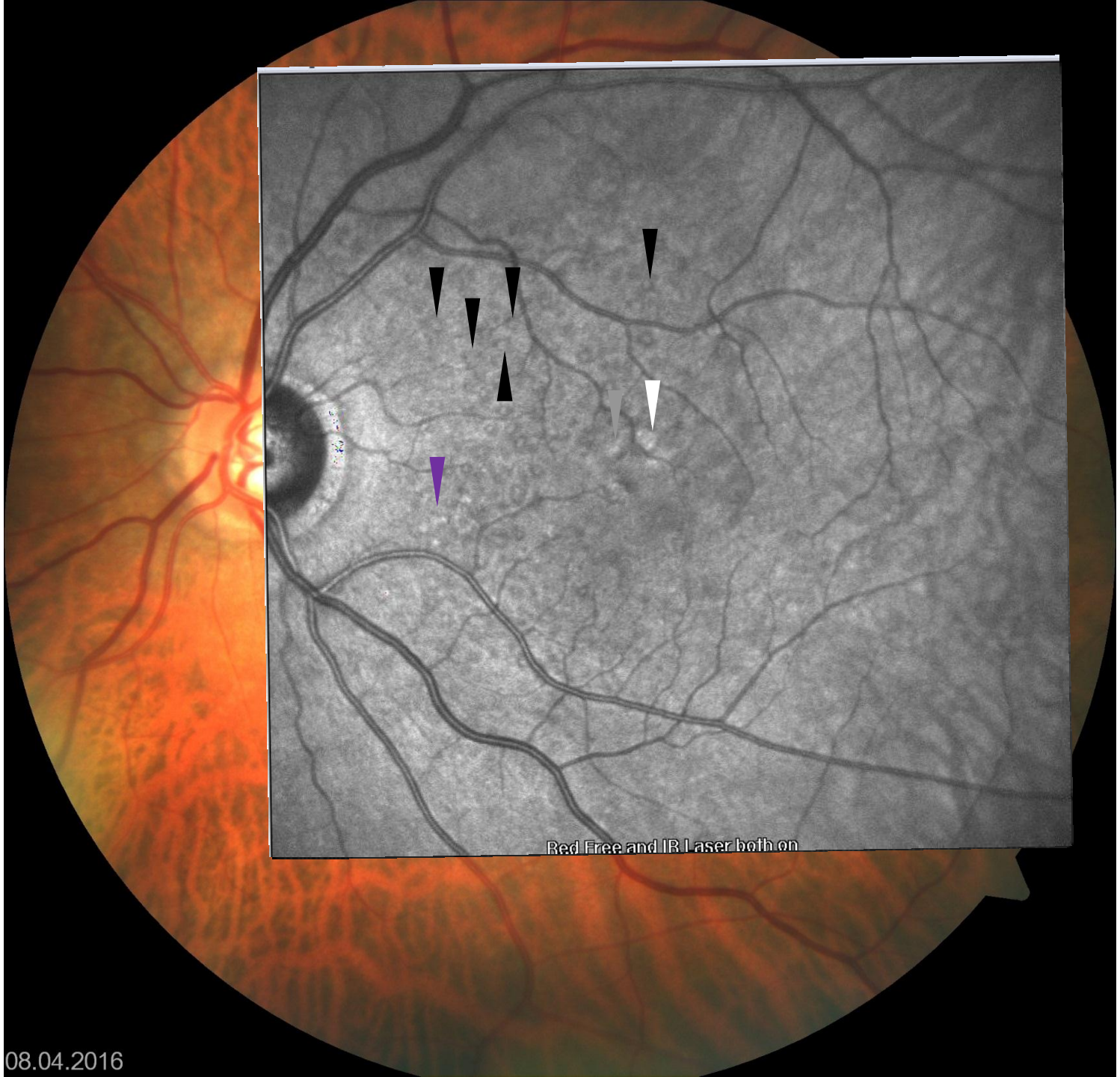
CFP

04/08/2016



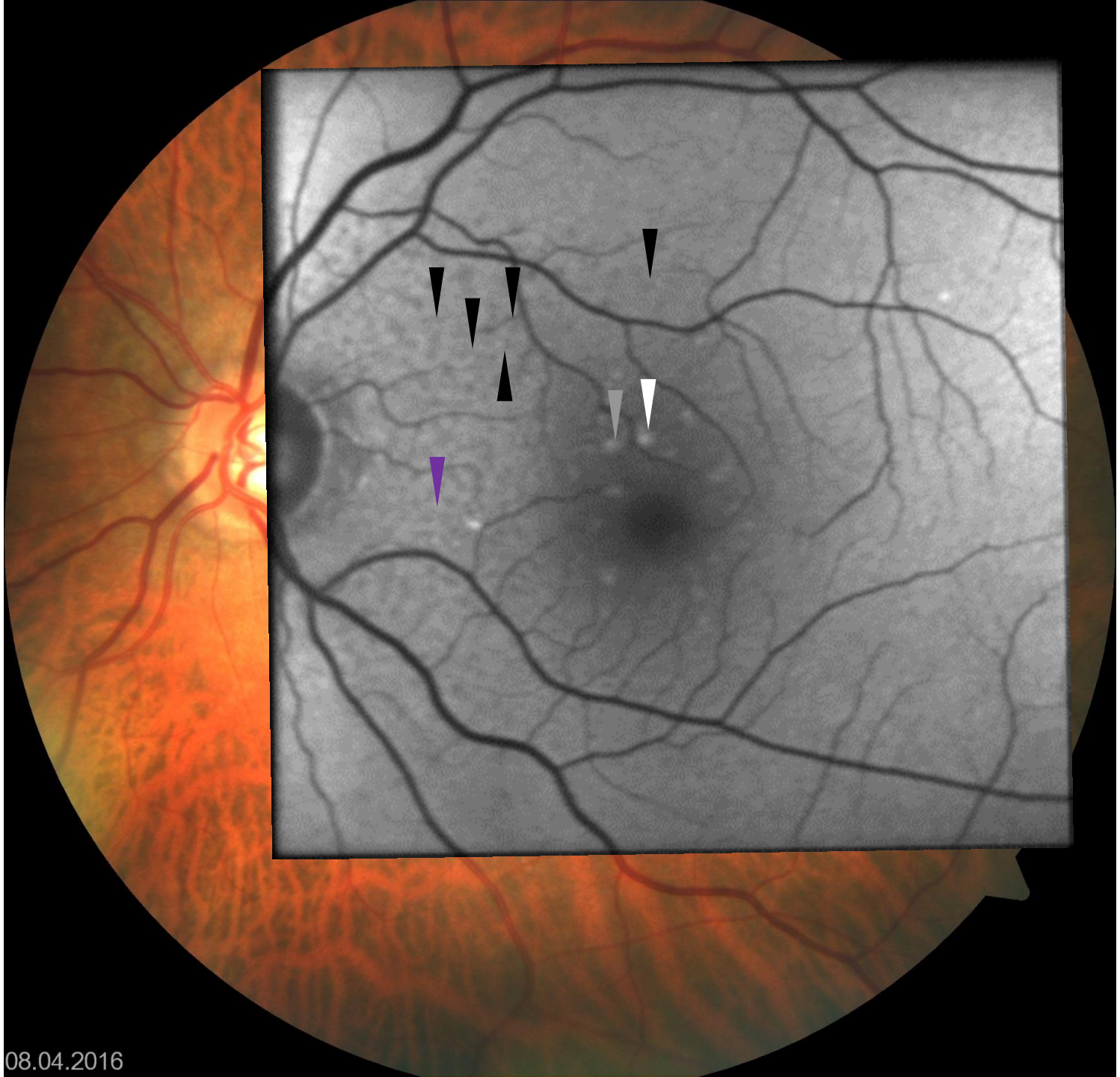
OCT at green lines in CFP

04/08/2016



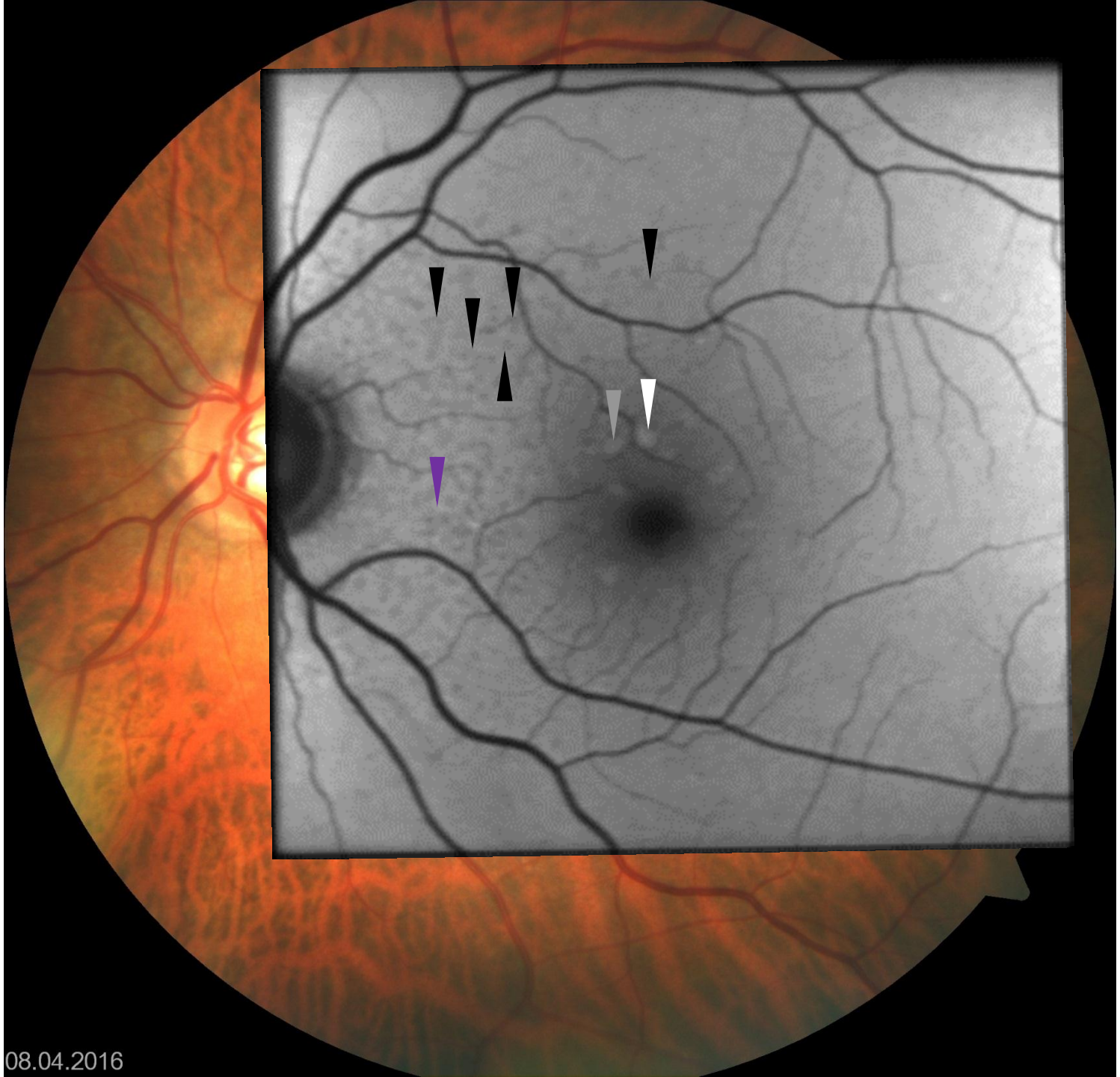
IR

04/08/2016



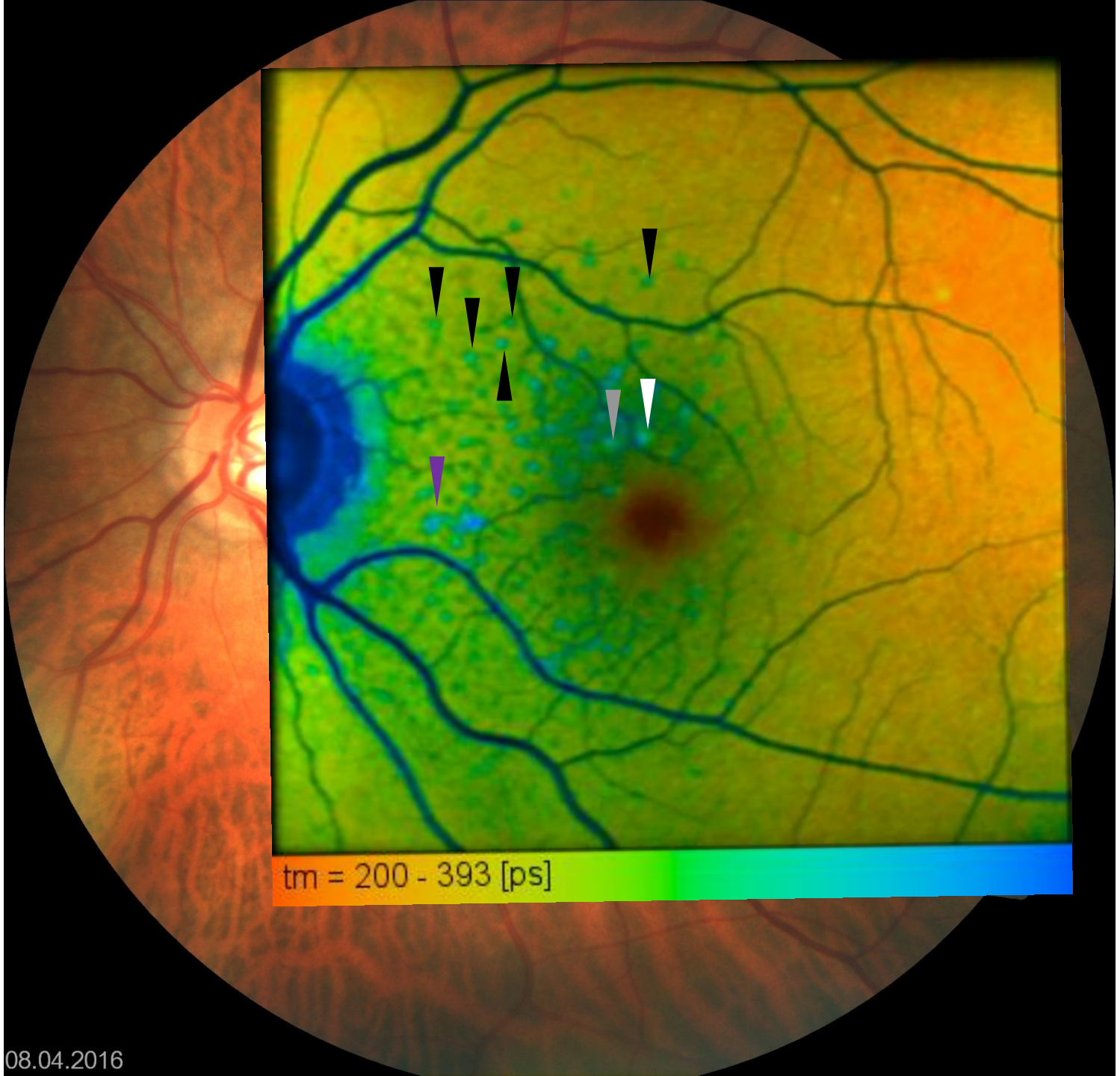
FAF SSC

04/08/2016



FAF LSC

04/08/2016



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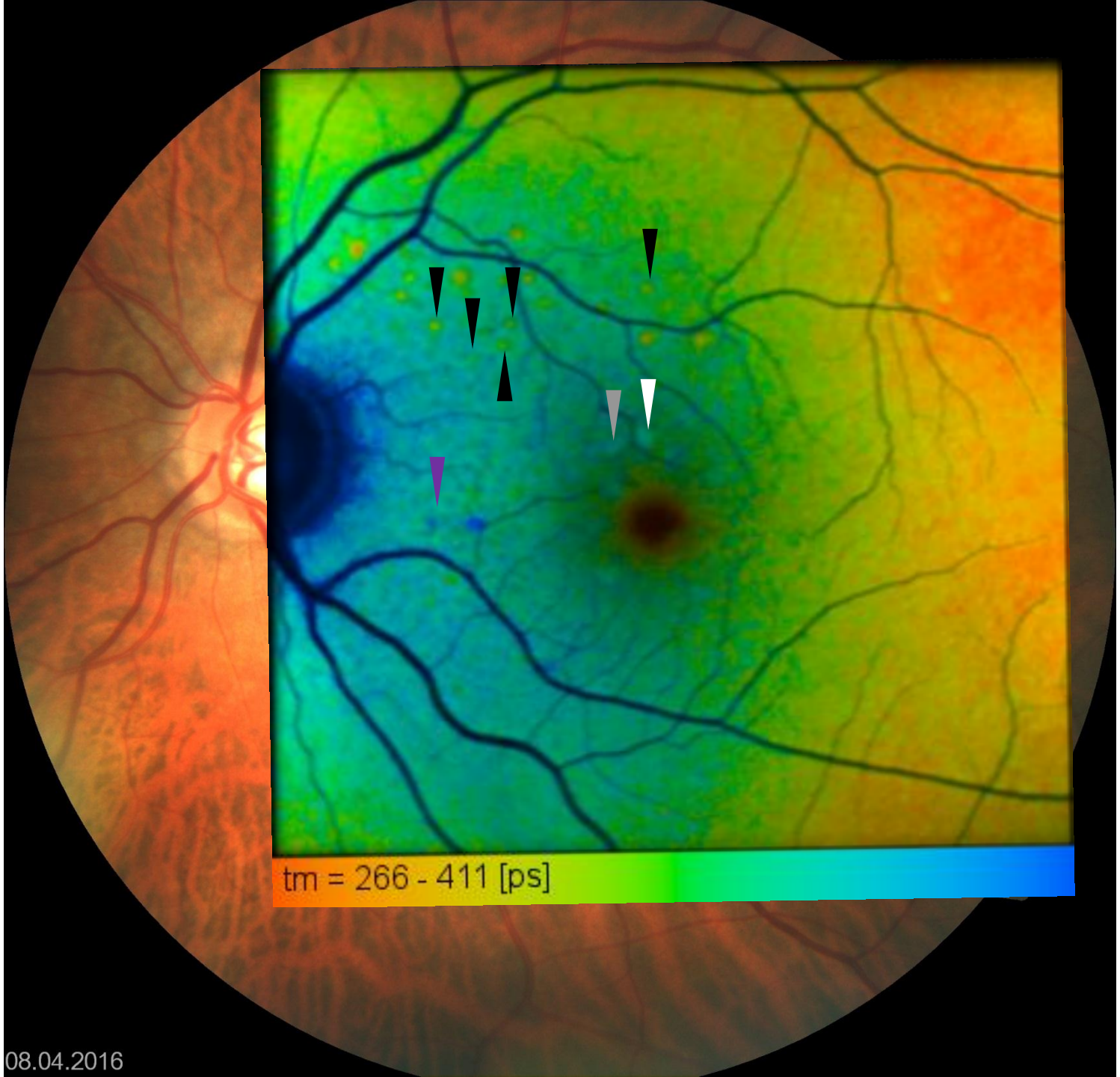


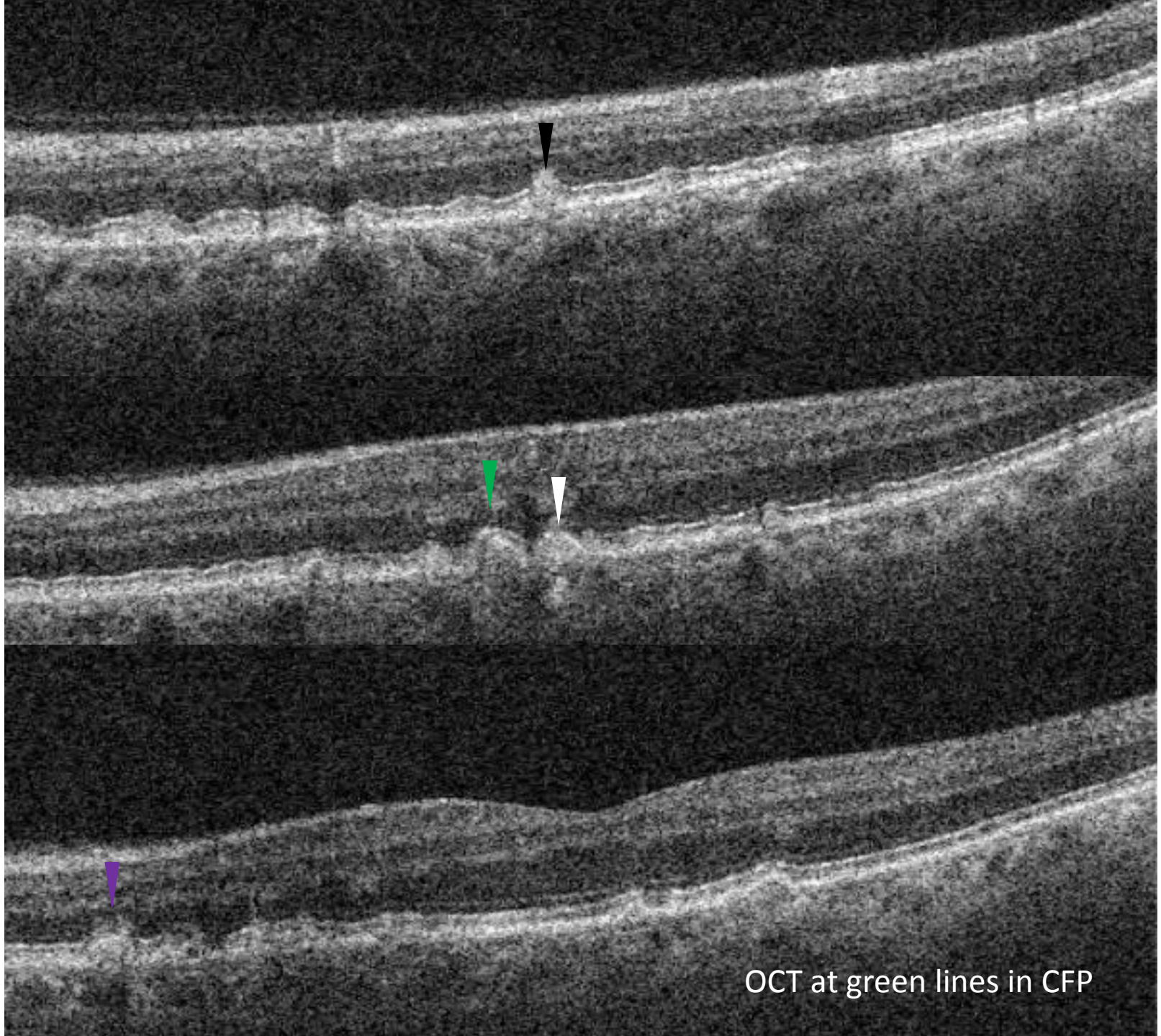
11/23/2017

CFP

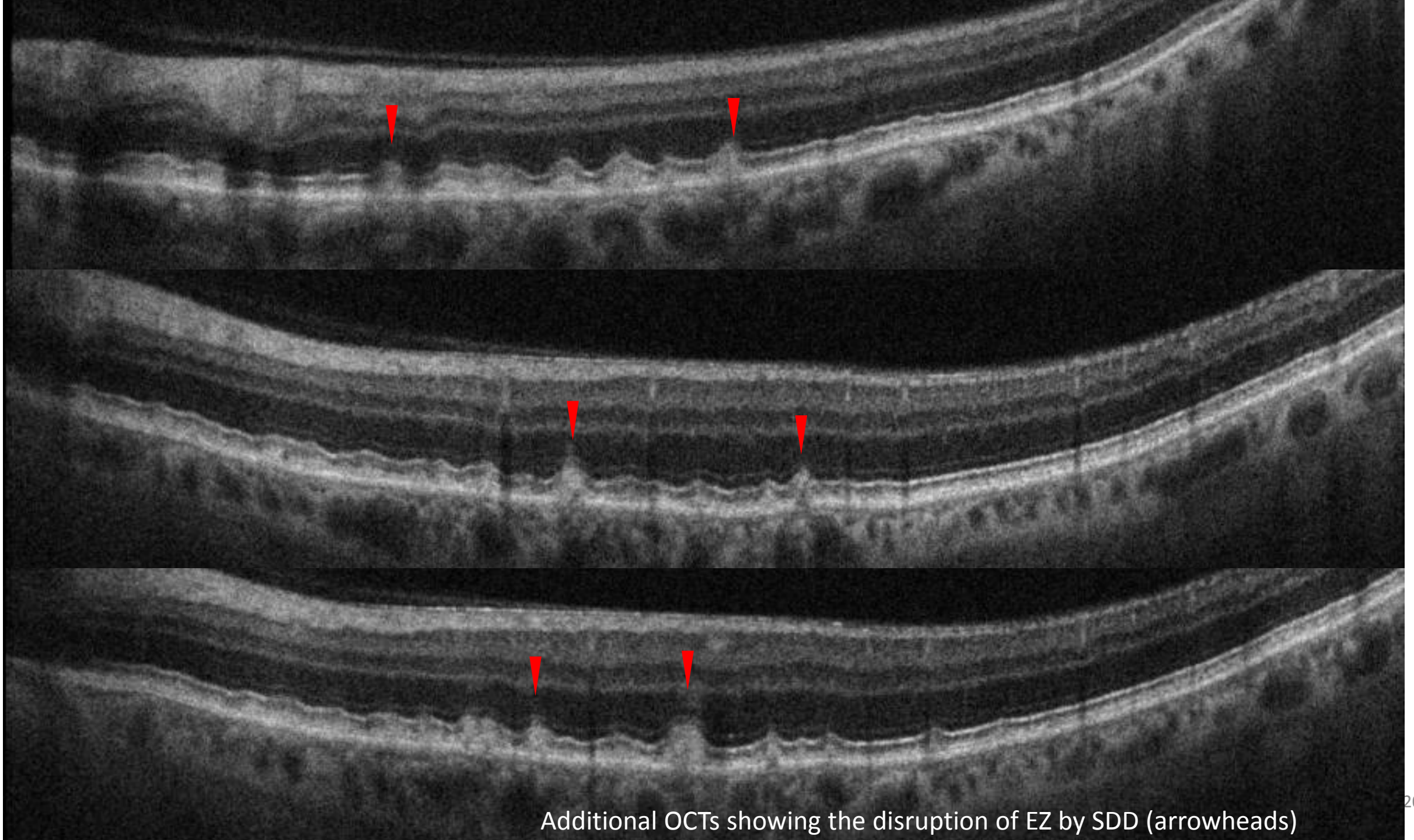
11/23/2017

CFP

11/23/2017

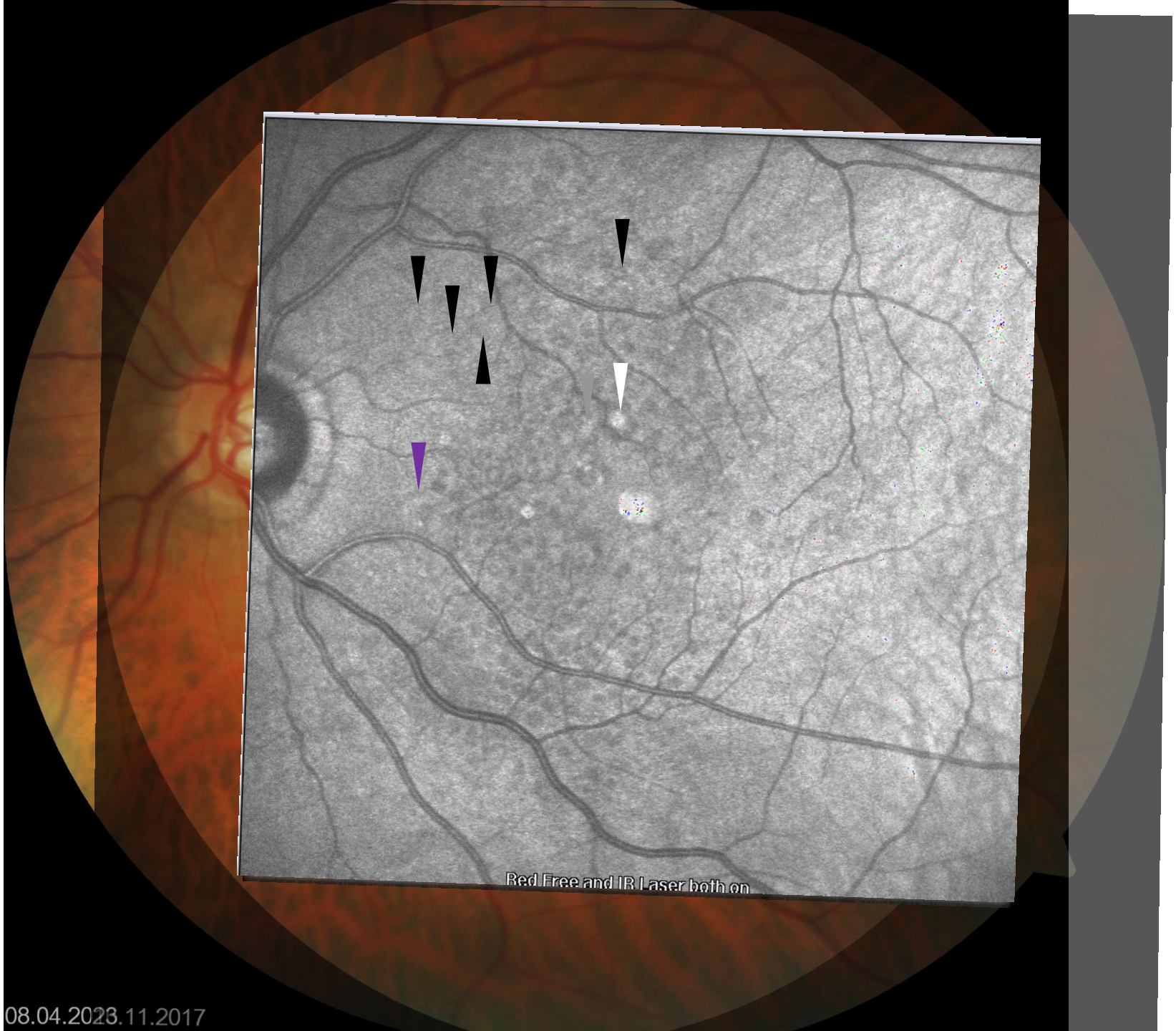


OCT at green lines in CFP



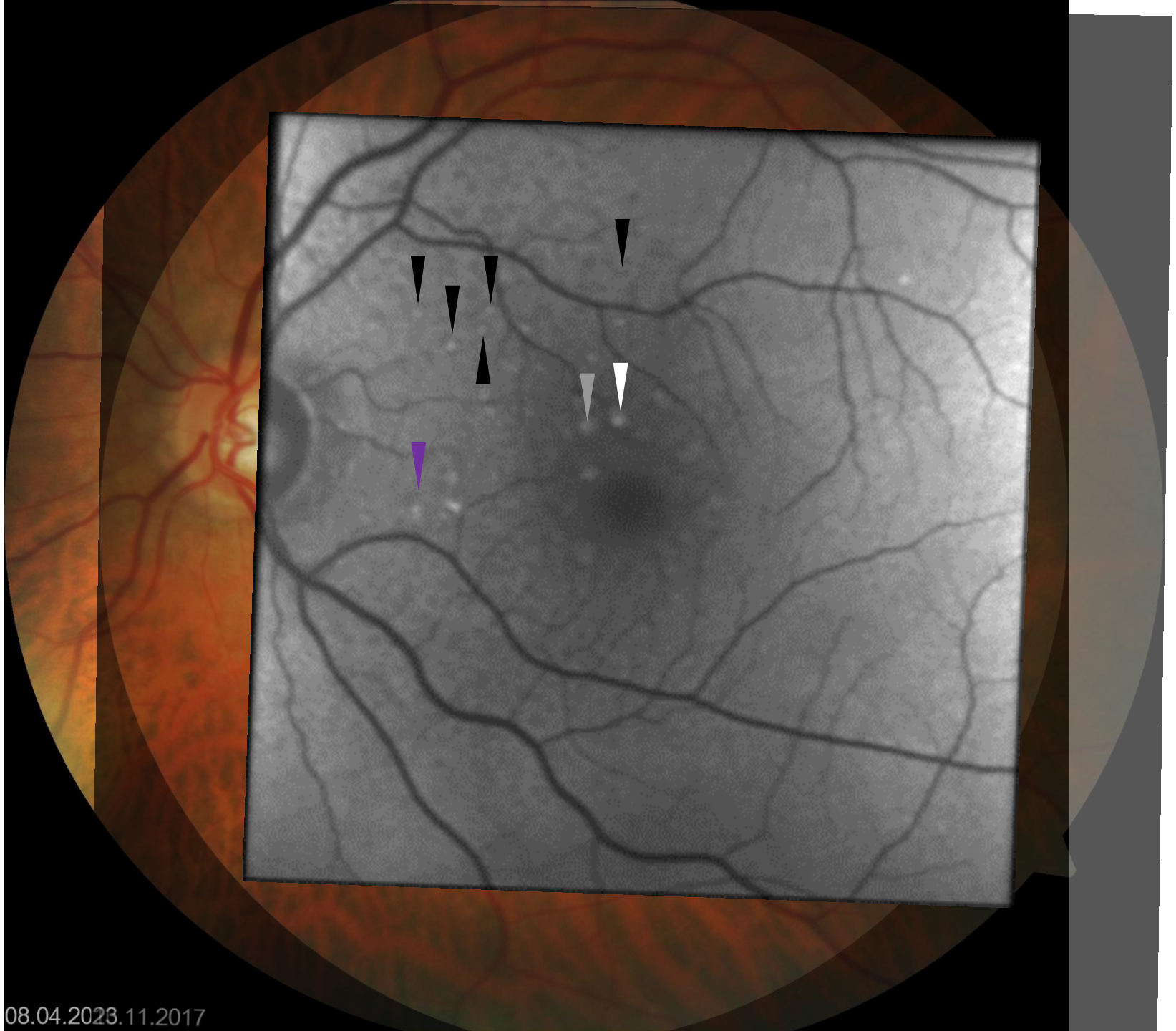
Additional OCTs showing the disruption of EZ by SDD (arrowheads)

11/23/2017



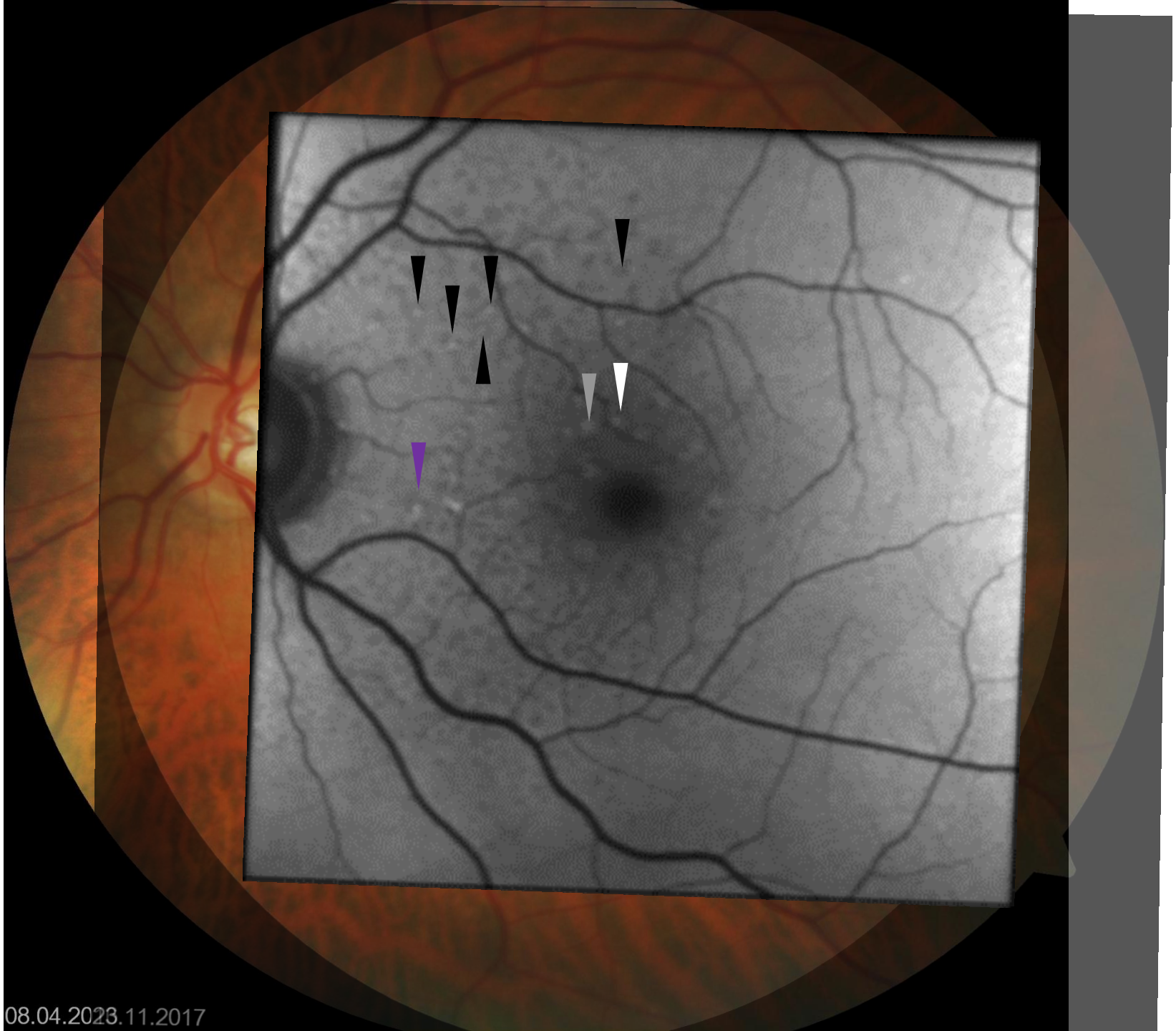
IR

11/23/2017



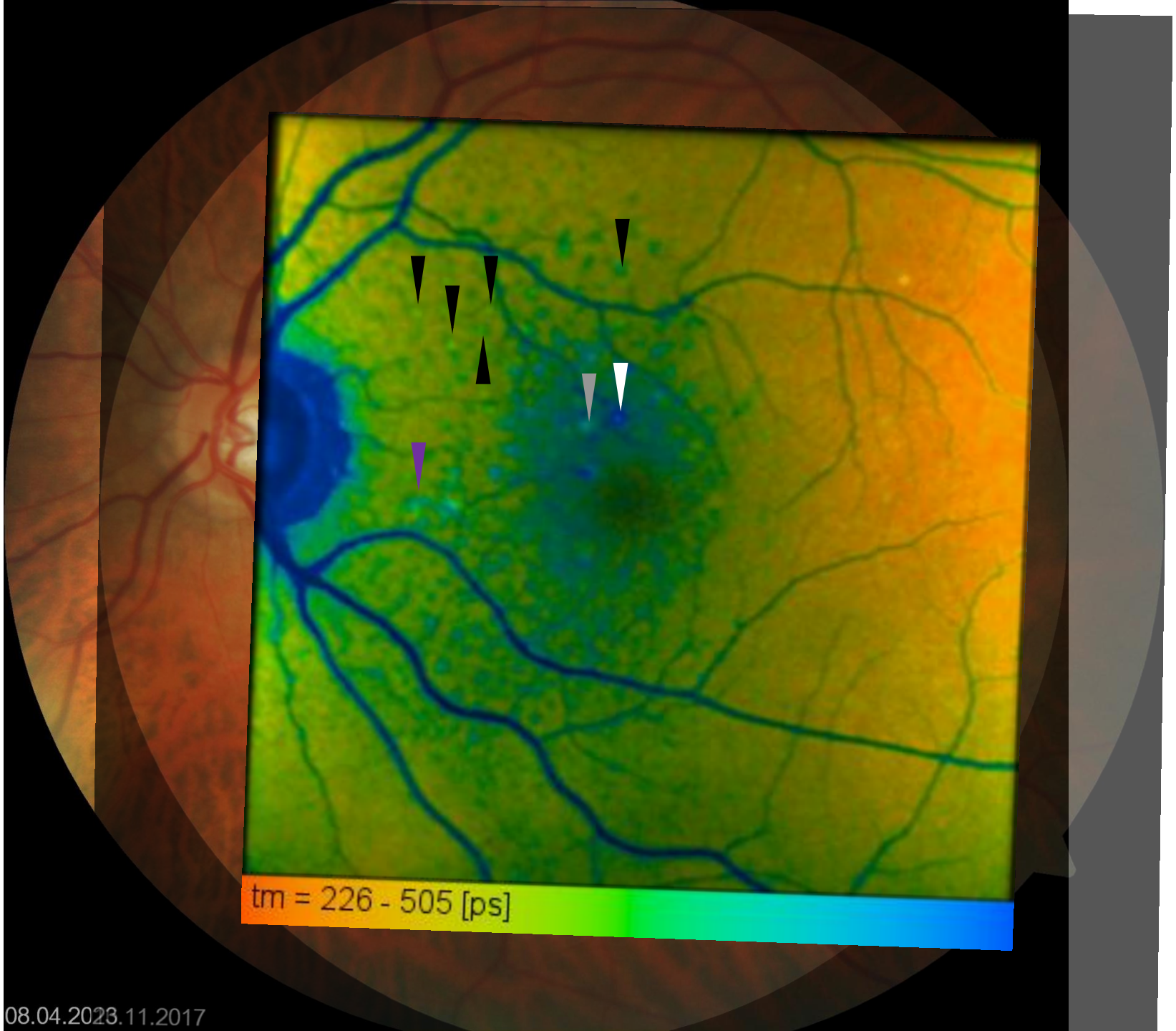
FAF SSC

11/23/2017

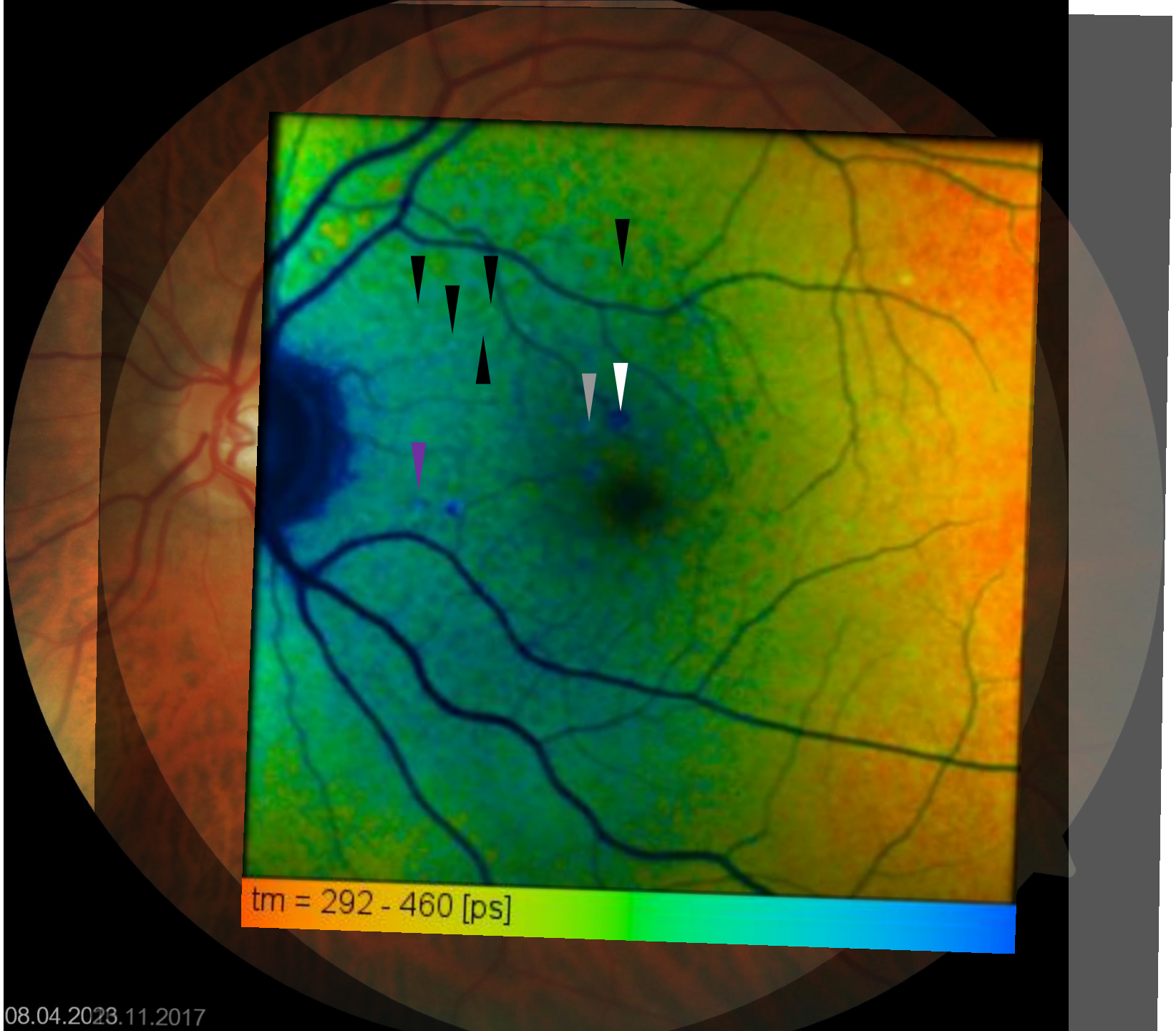


FAF LSC

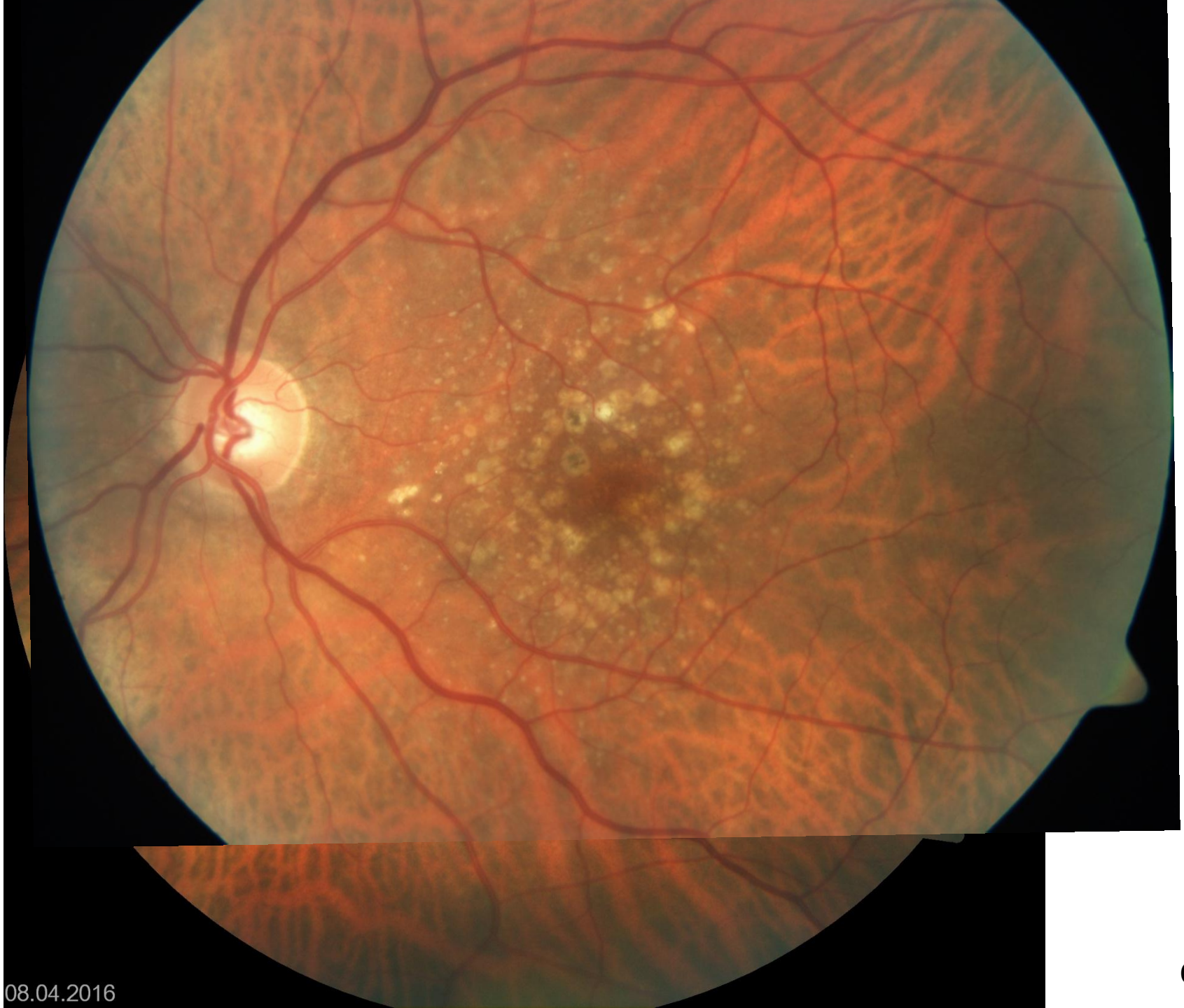
11/23/2017



11/23/2017

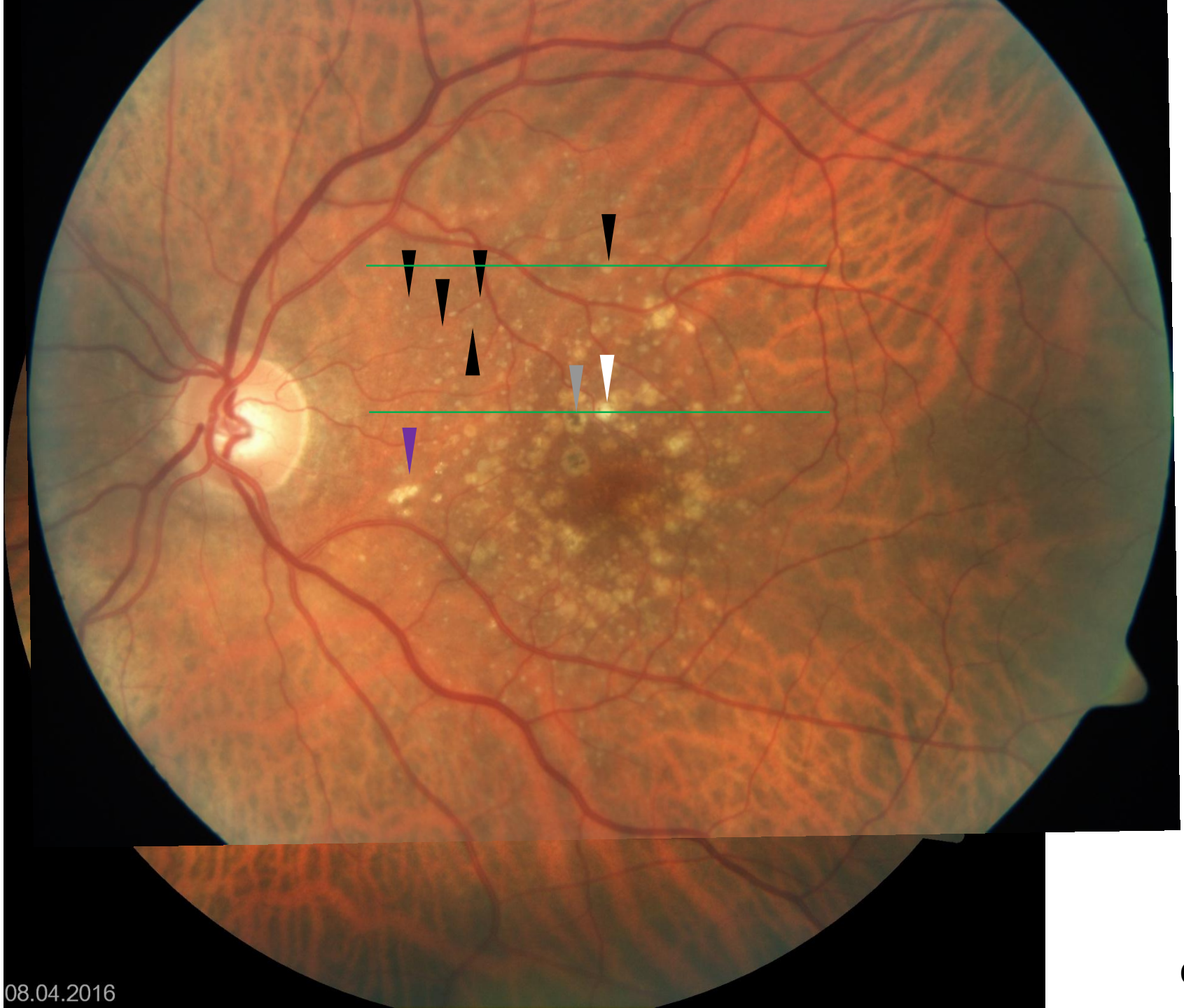


08/11/2020



CFP

08/11/2020



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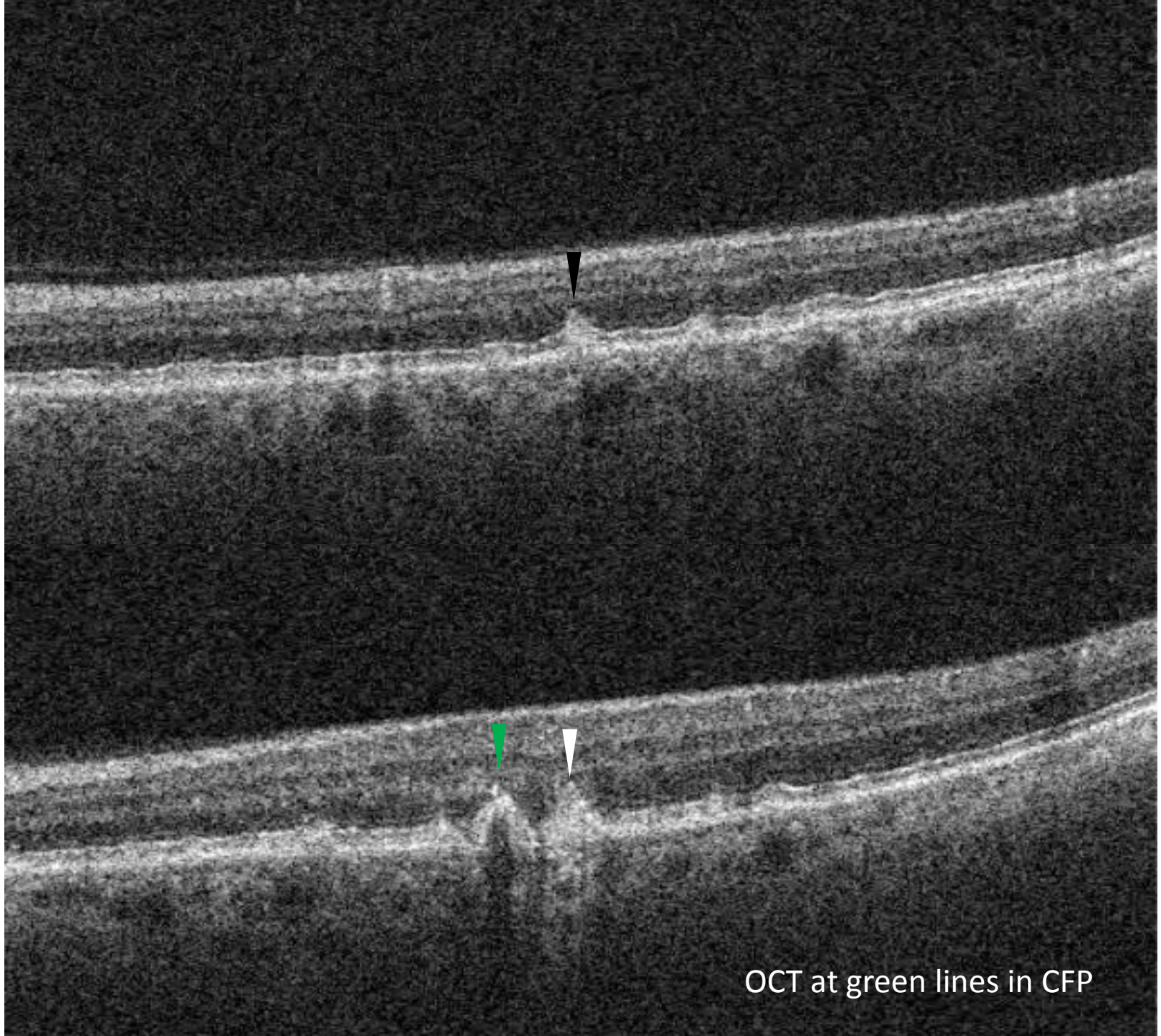
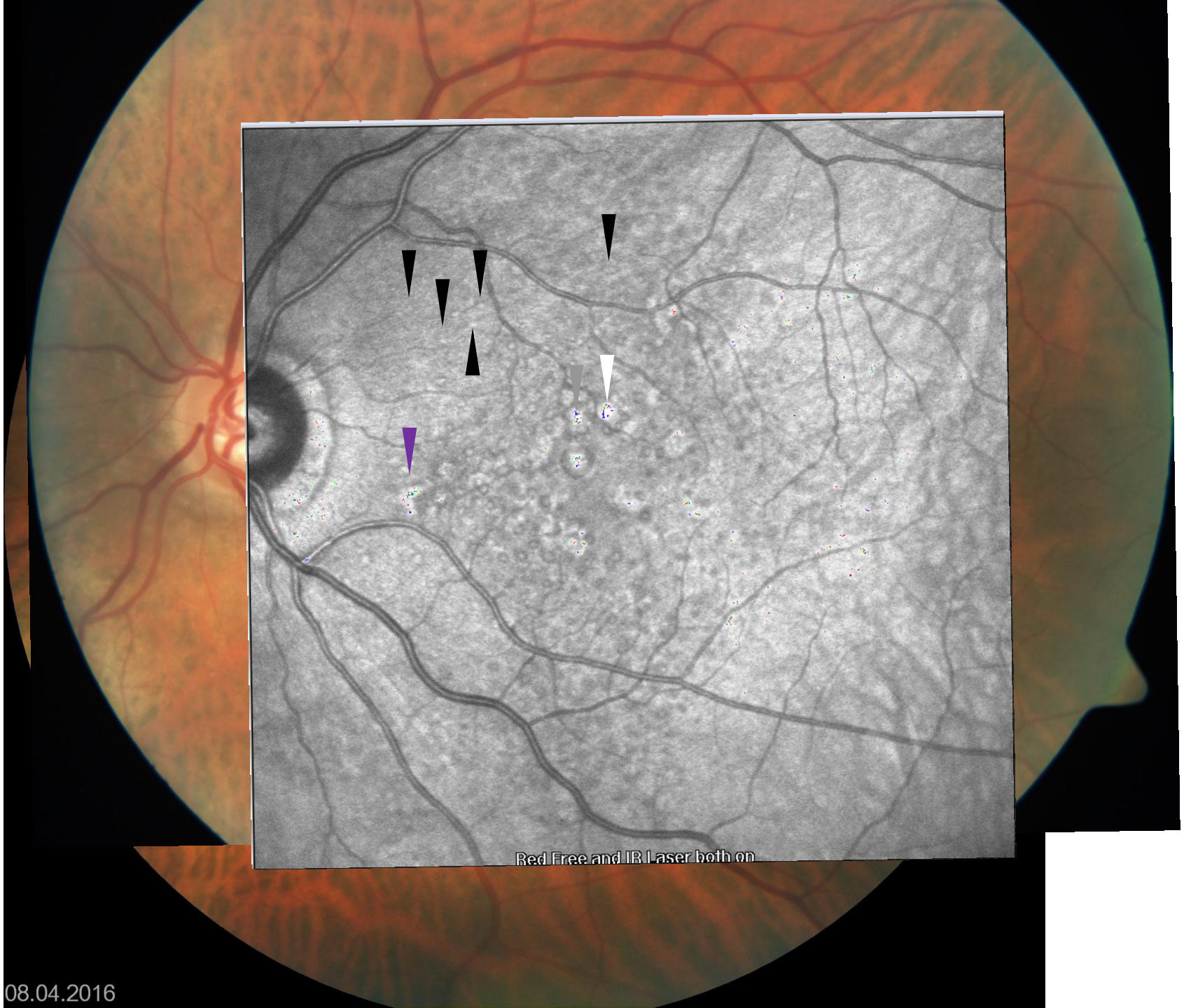
08/11/2020



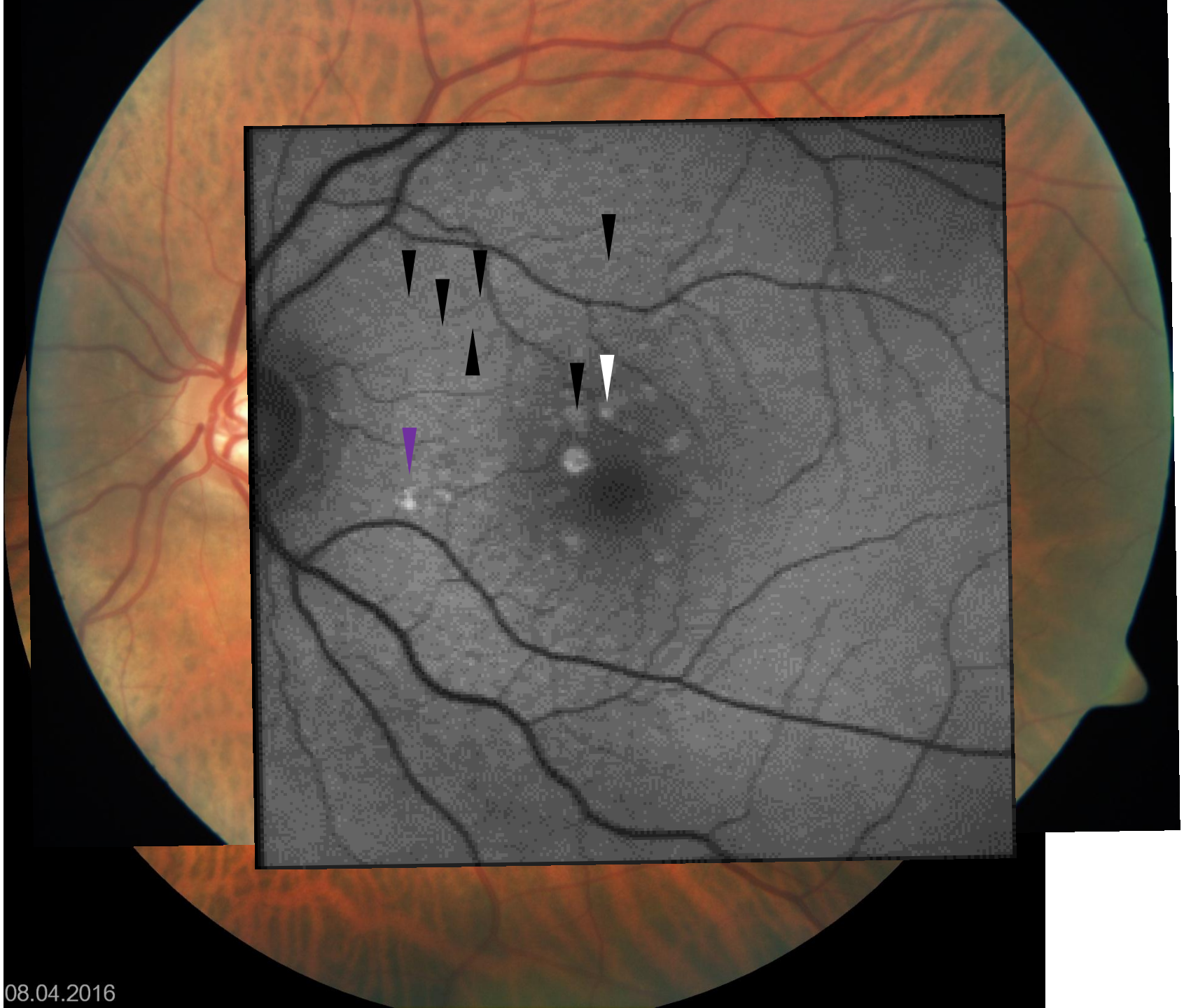
OCT at green lines in CFP

08/11/2020



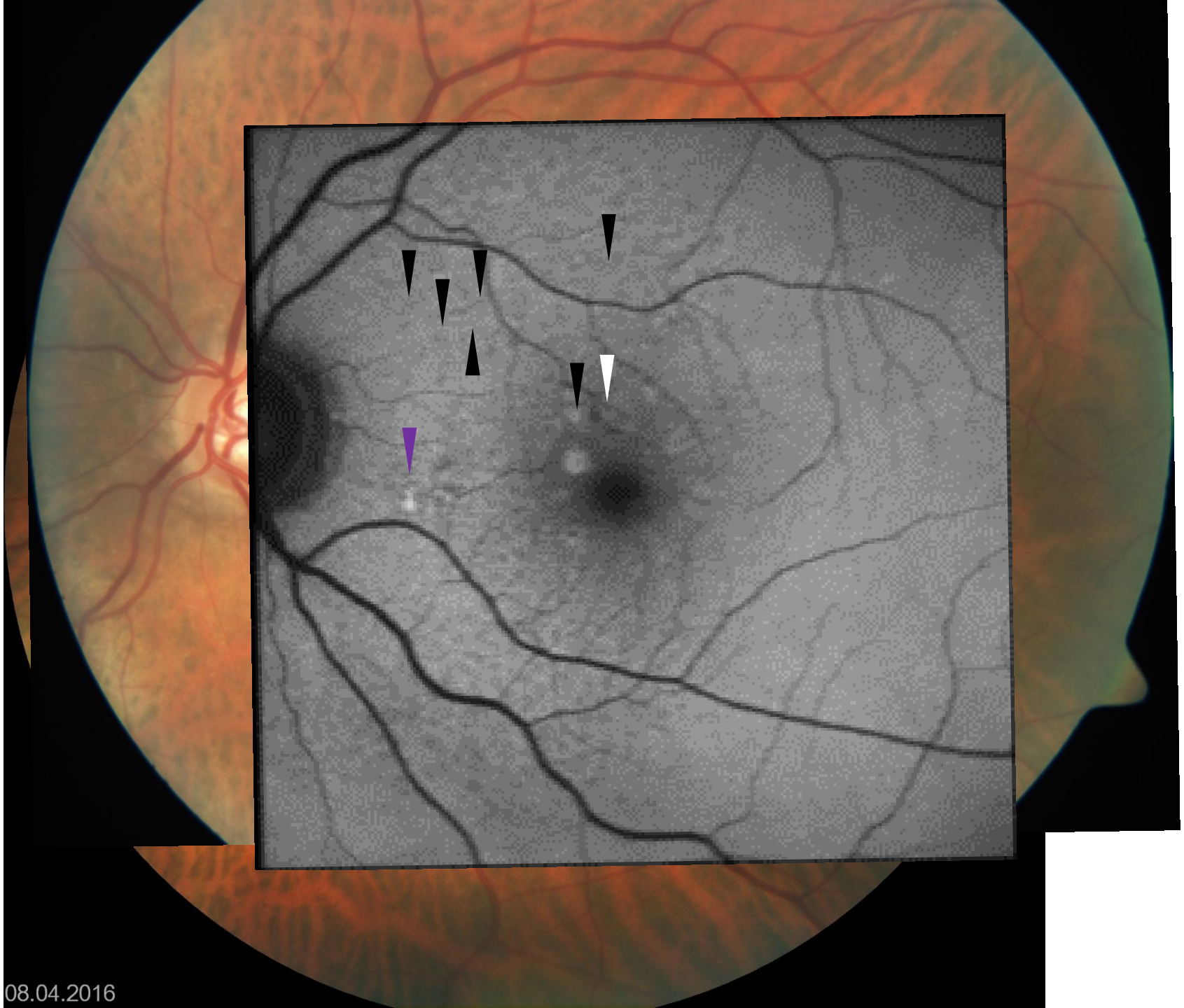
IR

08/11/2020



FAF SSC

08/11/2020



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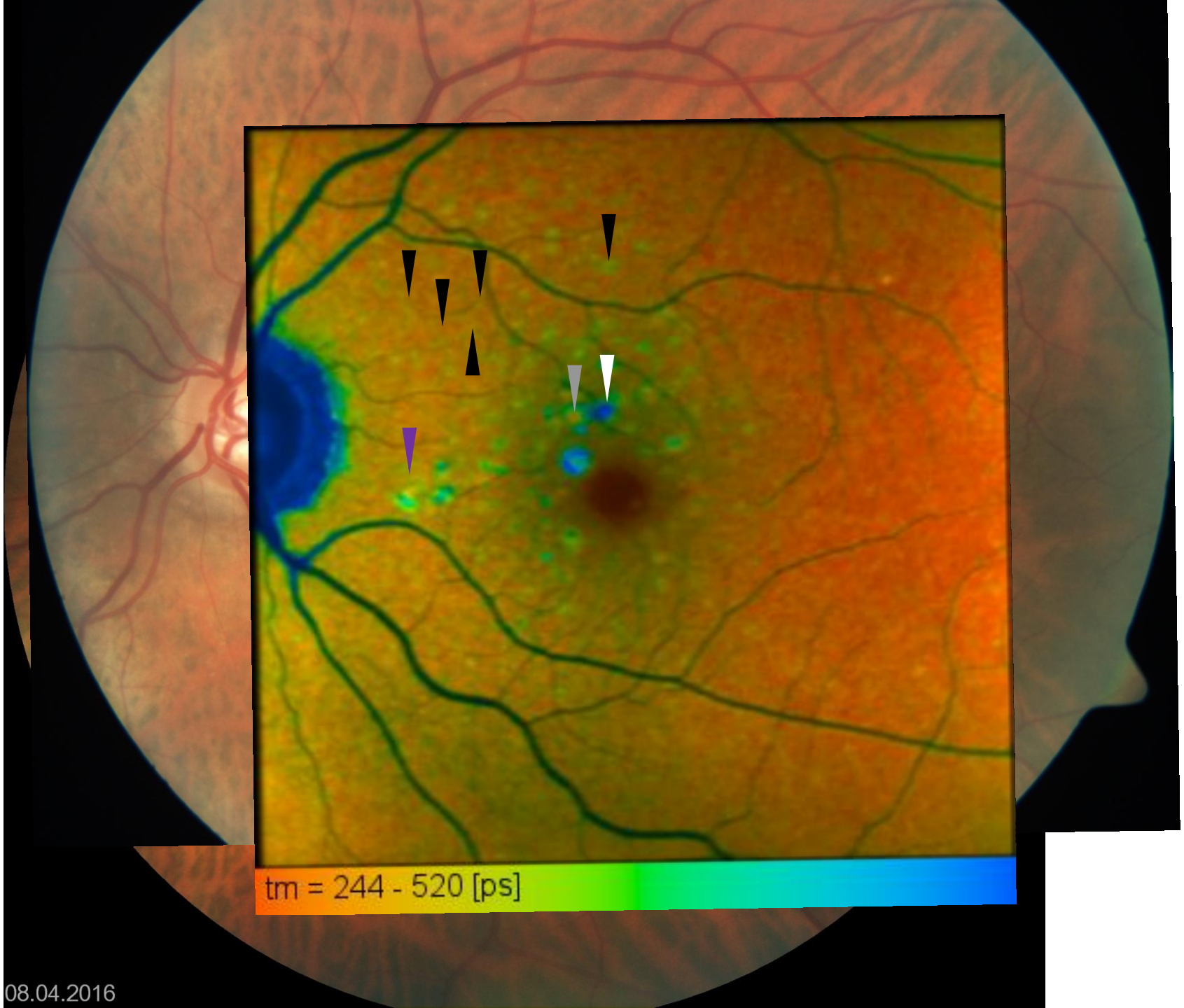

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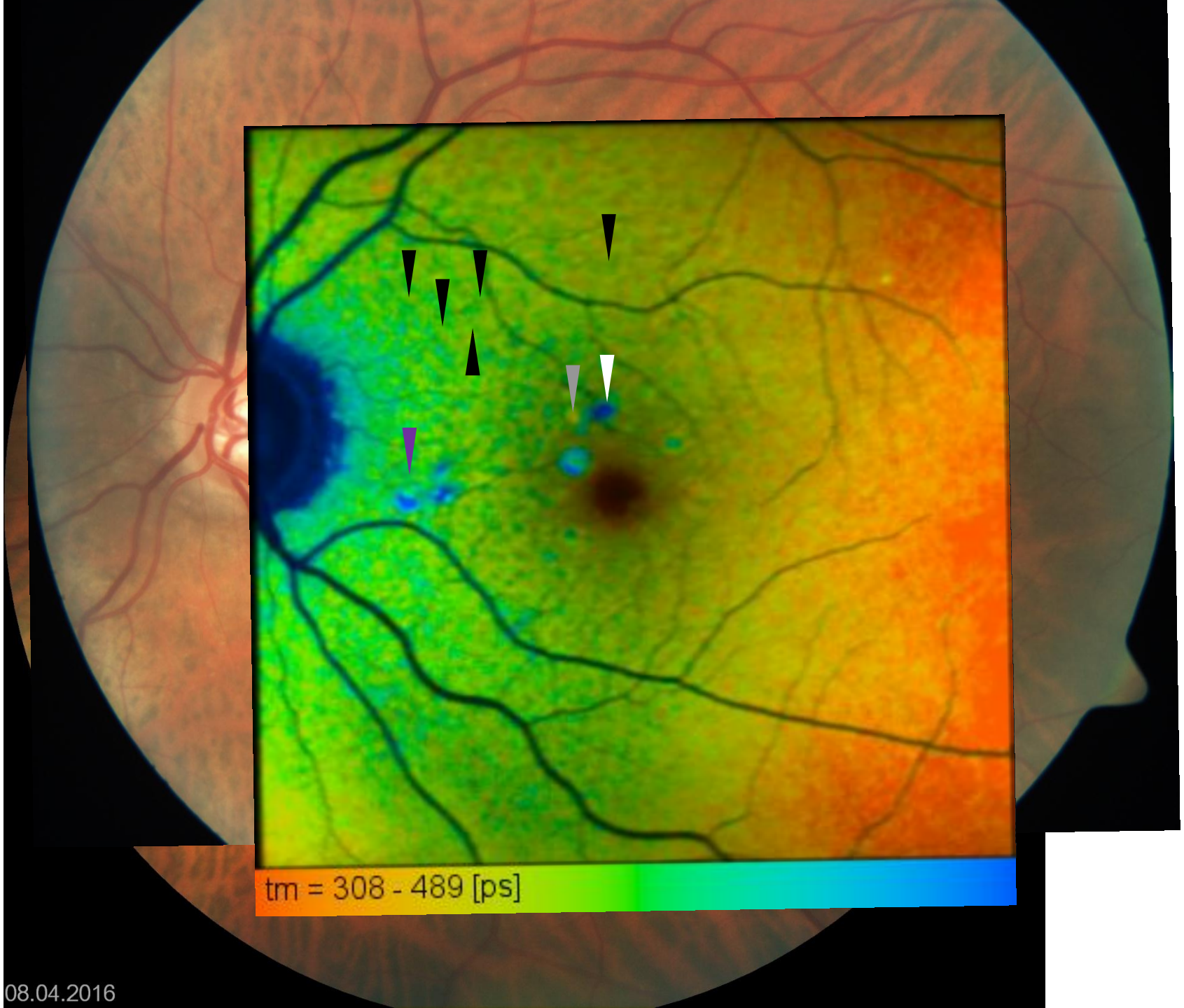
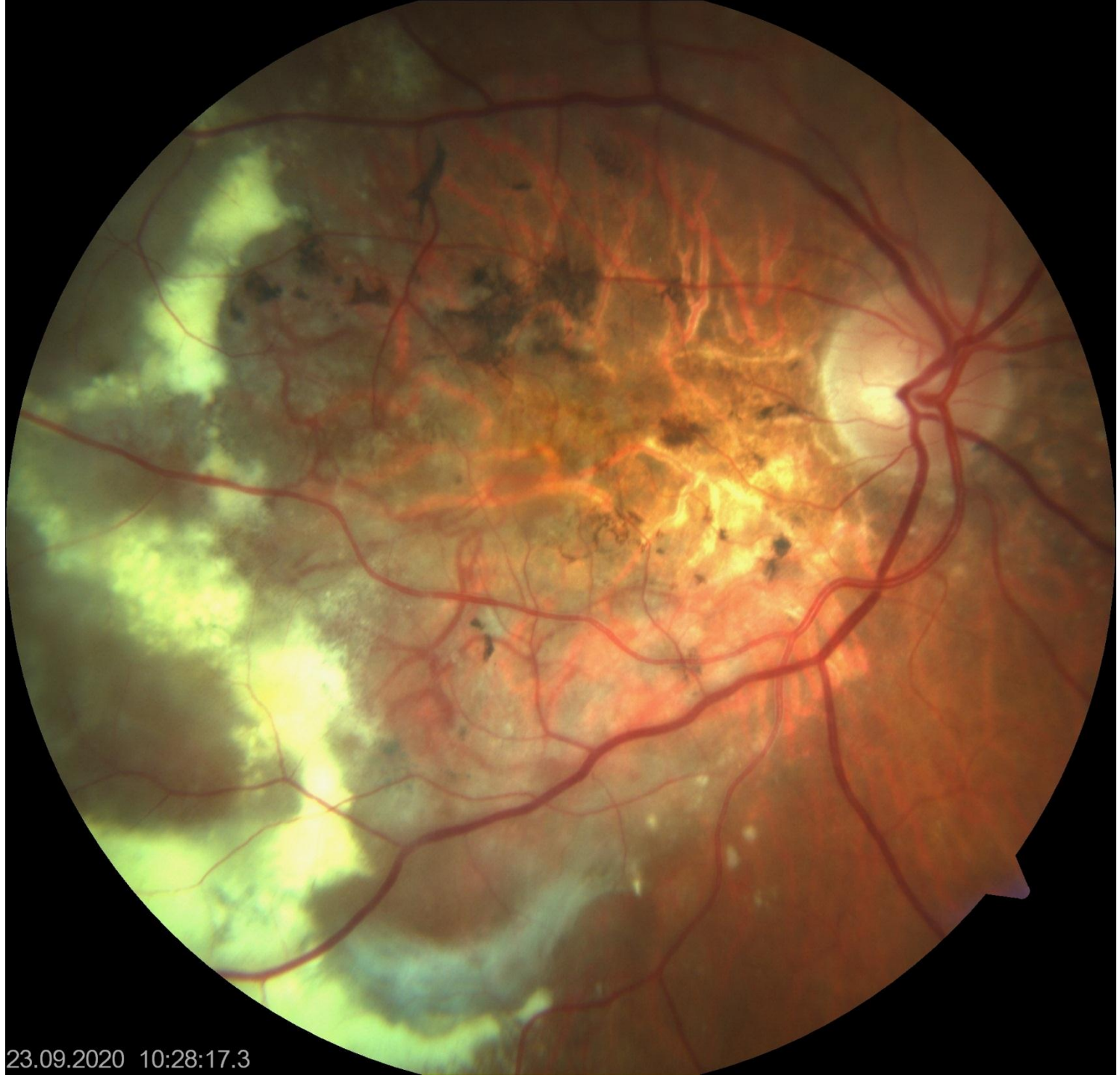


AMD_46, 79 years

What do we see in this GA? Purple arrowhead: remaining druse? Black arrowhead: residual RPE (short lifetime)**? White arrowhead: fibrotic scar** (having long FAF lifetimes from collagen what holds for the white arrowhwead)? **But what makes the short FAF lifetime at the grey arrowhed (green in OCT)? Could be macular pigment, but what is the thick, hyperreflective, subretinal material (fibrotic tissue or BlamD?) and what contribution could it make to FAF?**

09/23/2020



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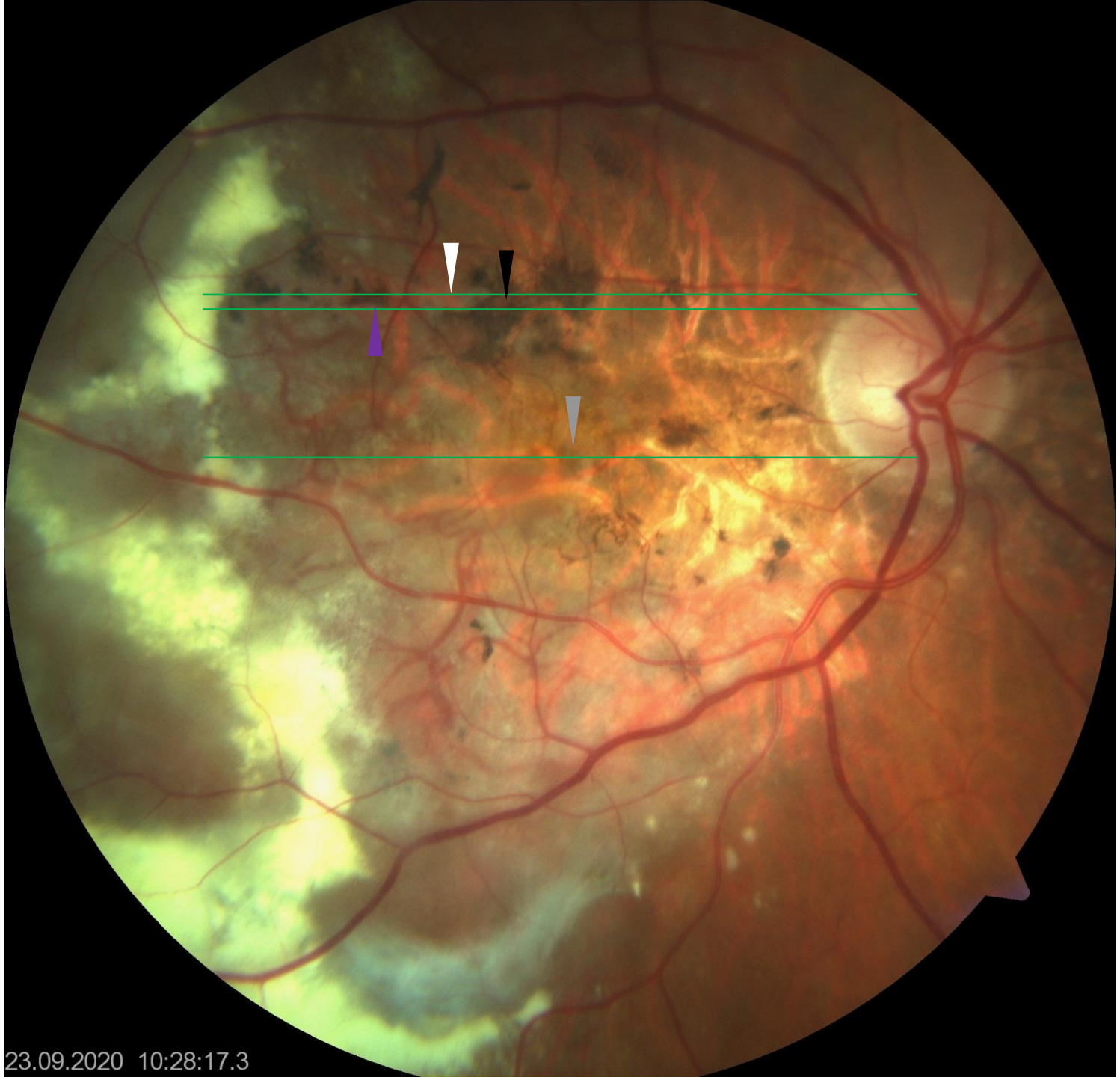
09/23/2020



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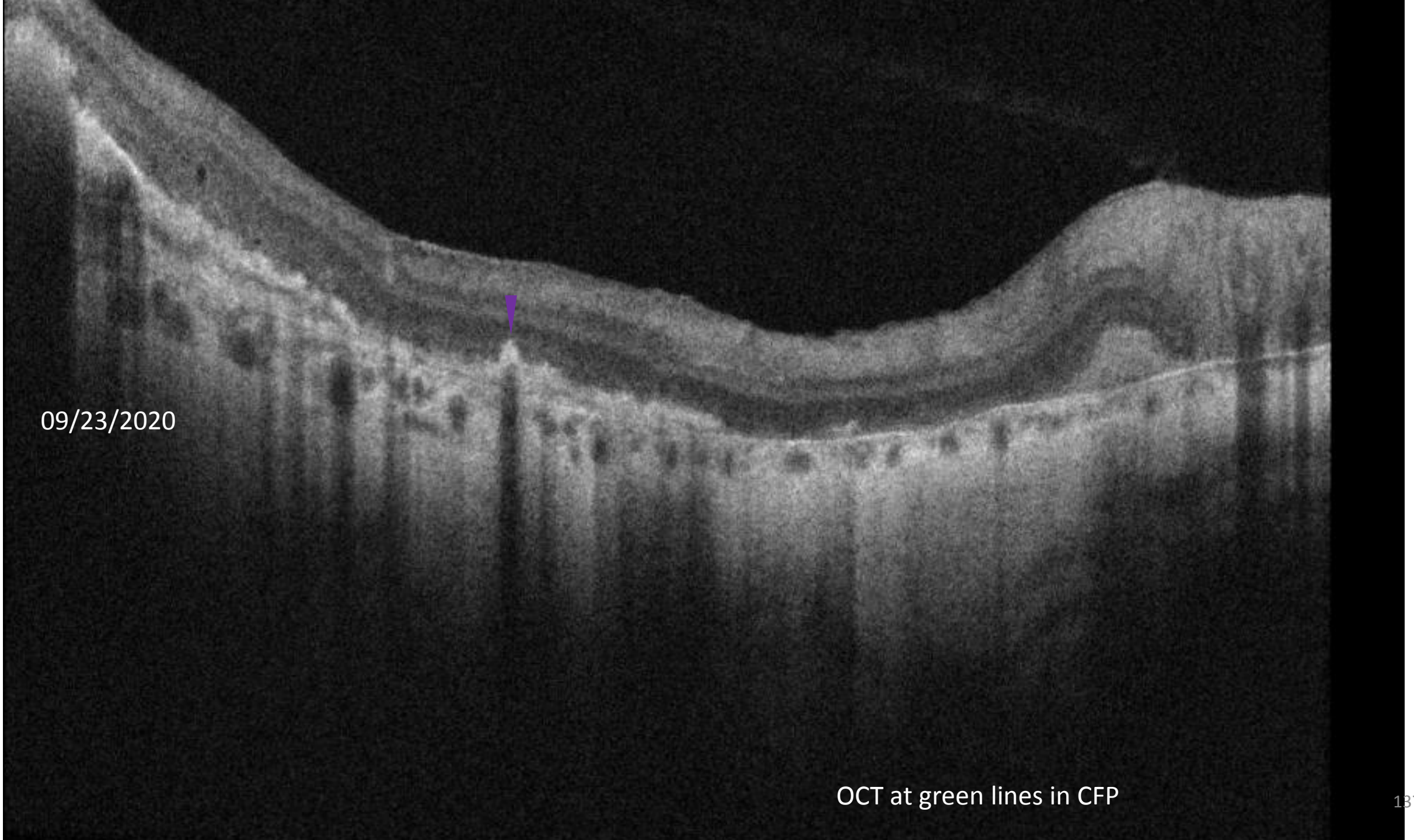
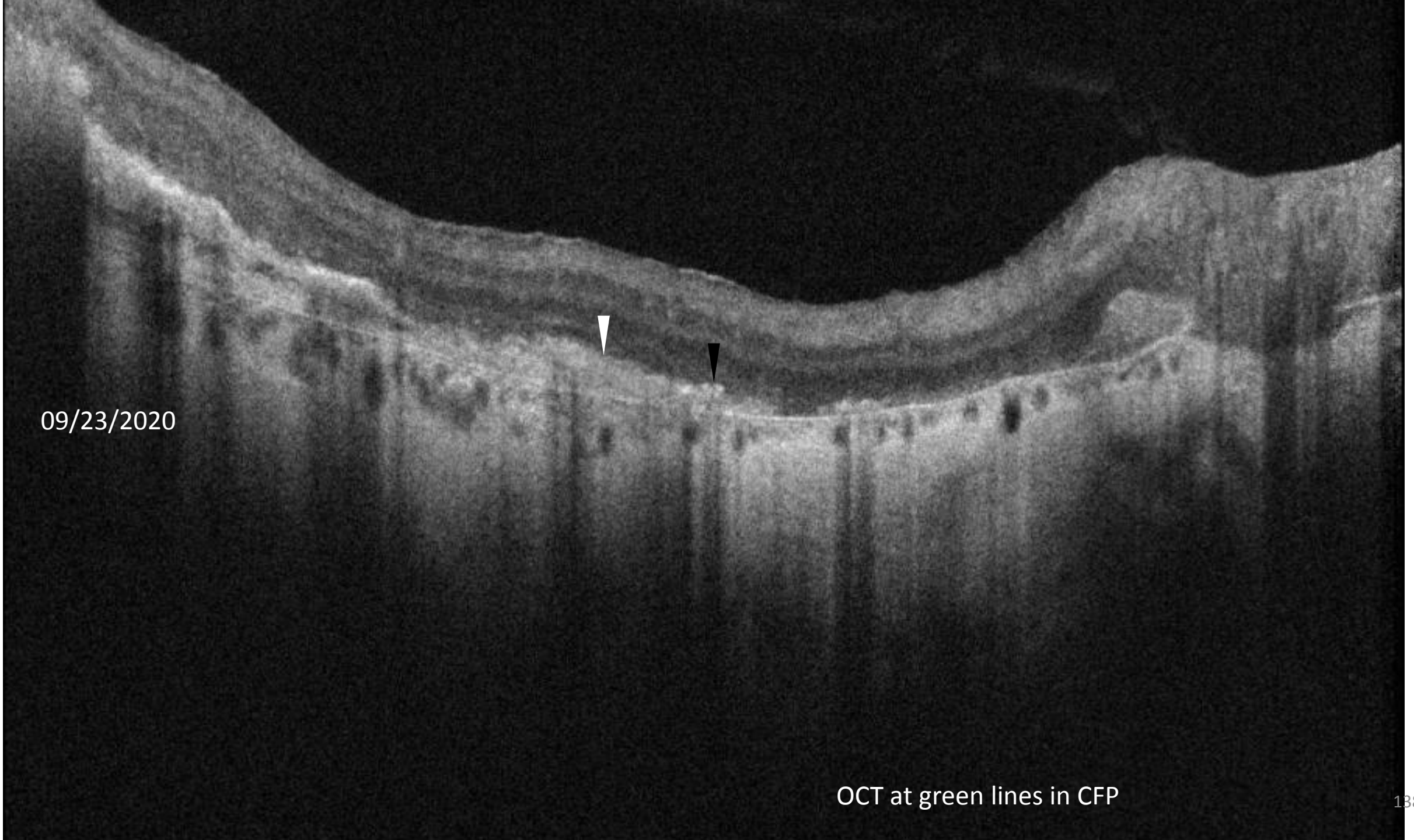
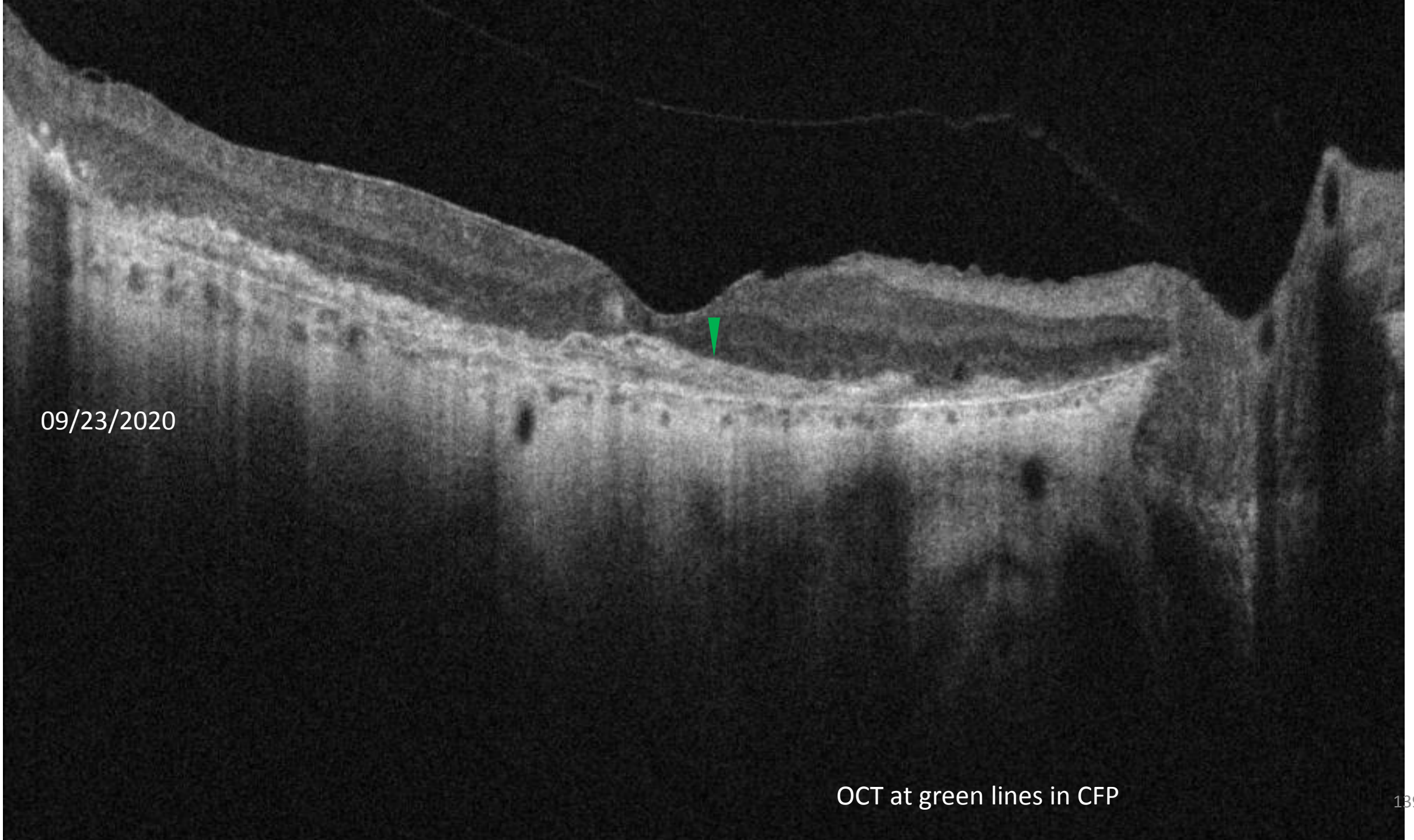


OCT at green lines in CFP

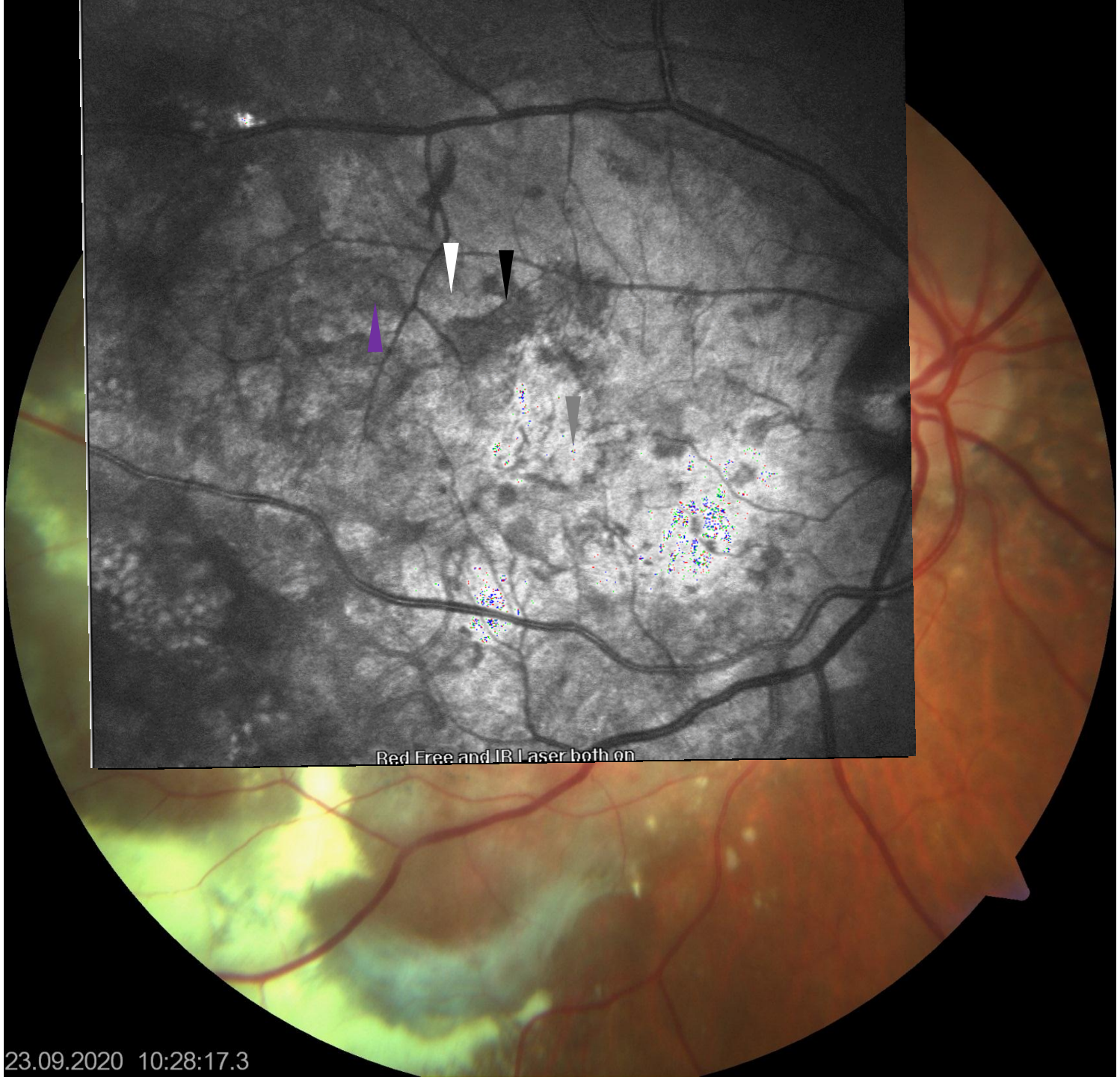


OCT at green lines in CFP



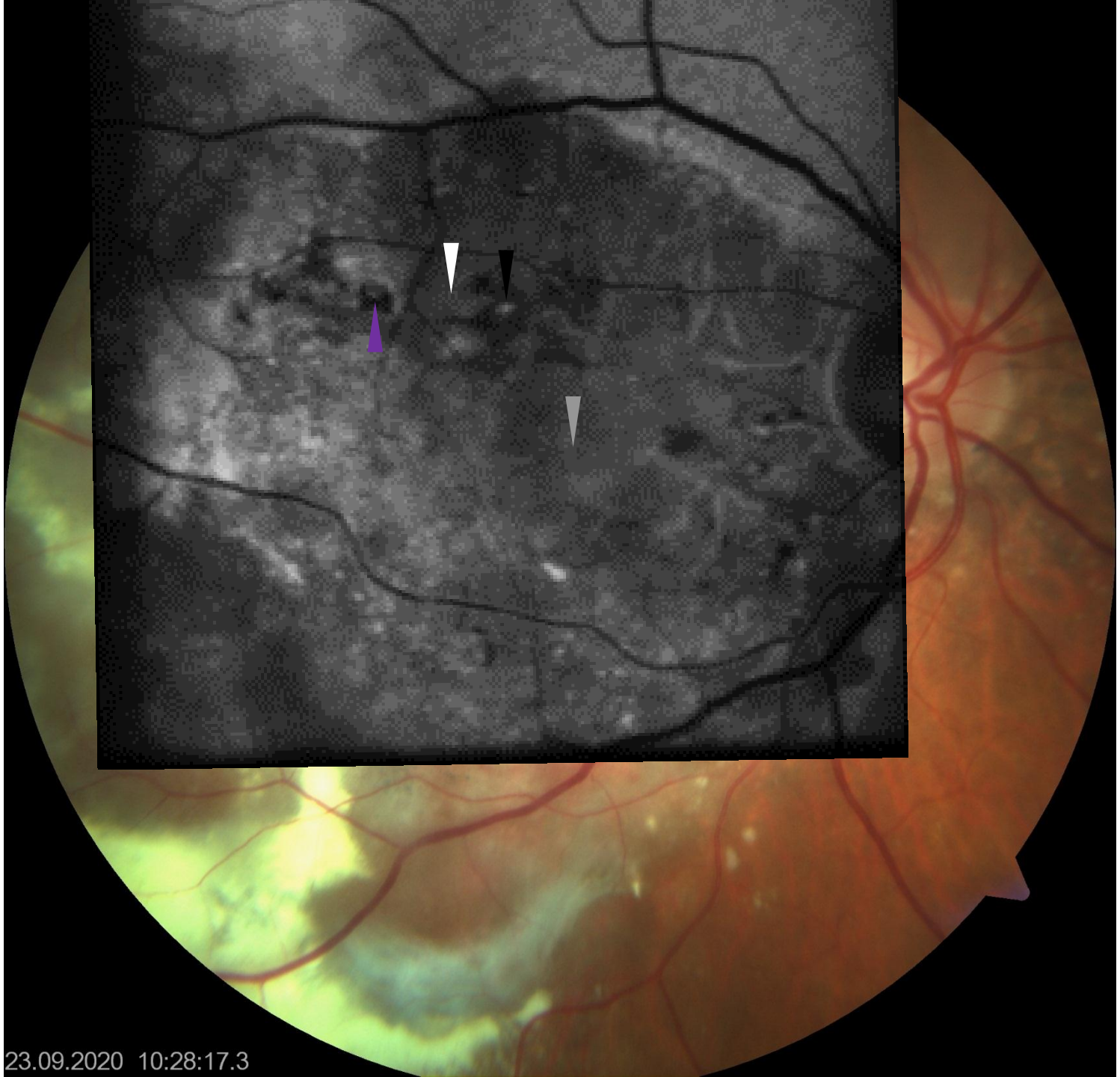
OCT at green lines in CFP

09/23/2020

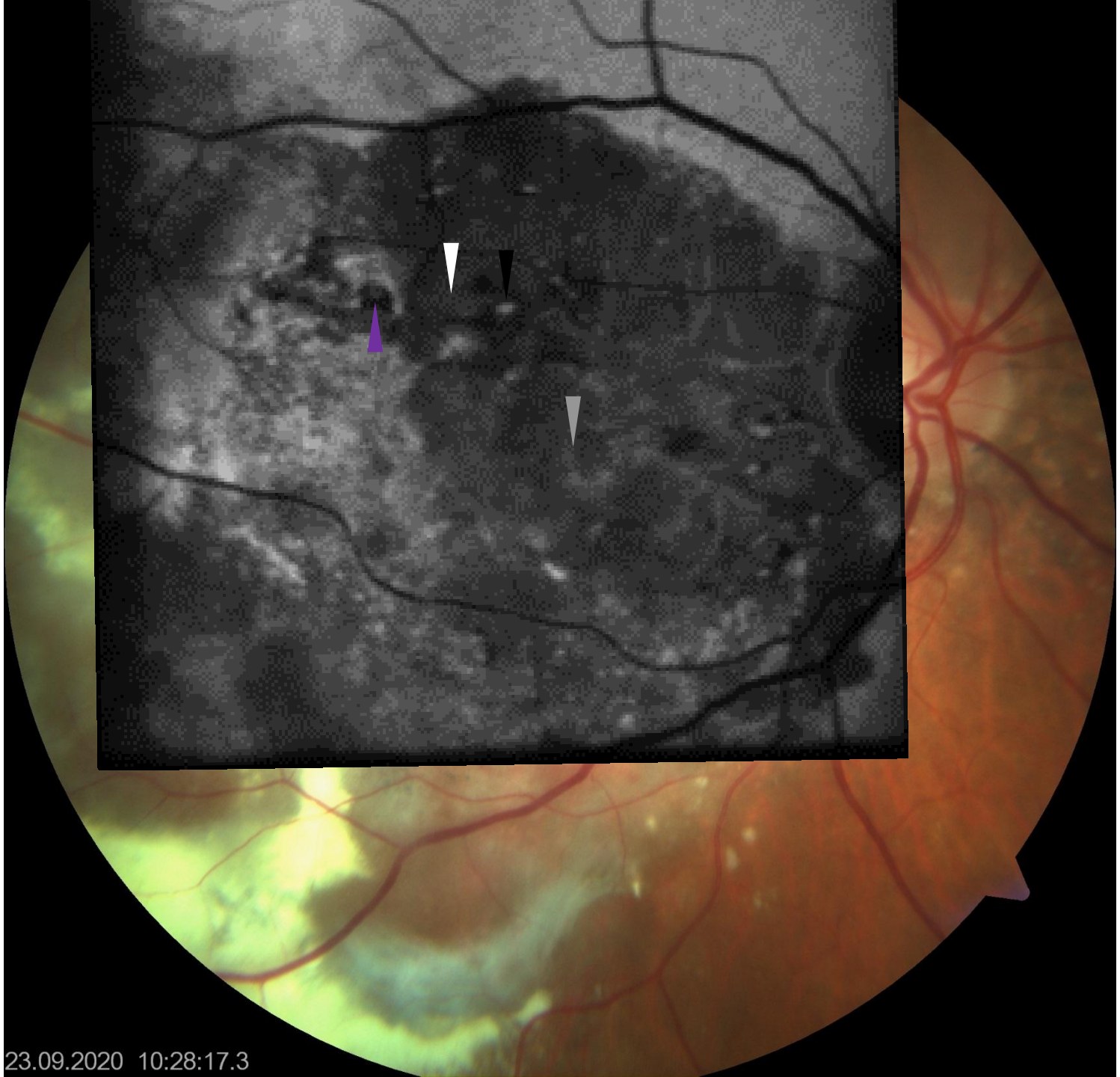


IR

09/23/2020

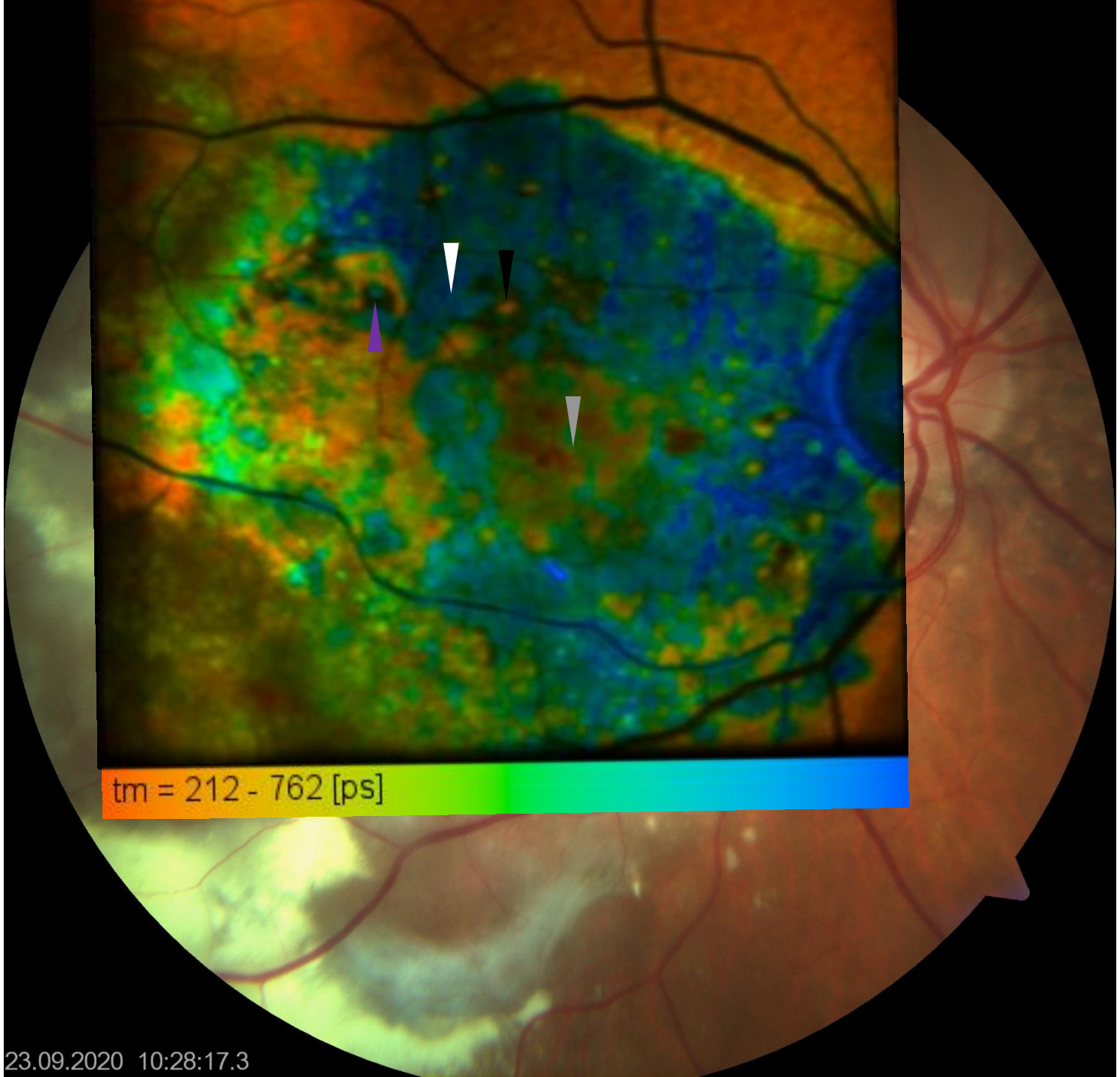


09/23/2020

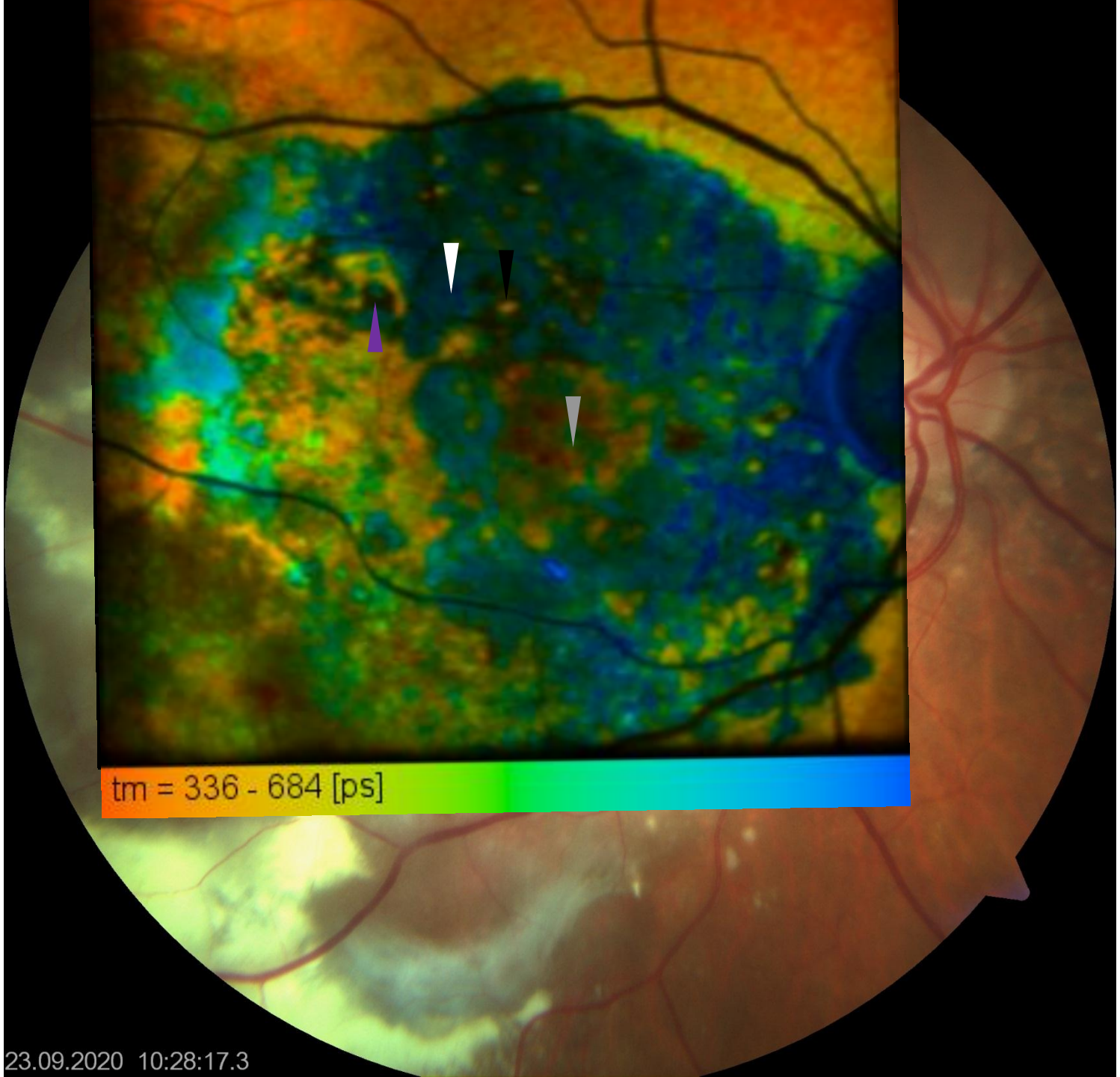


FAF LSC

09/23/2020



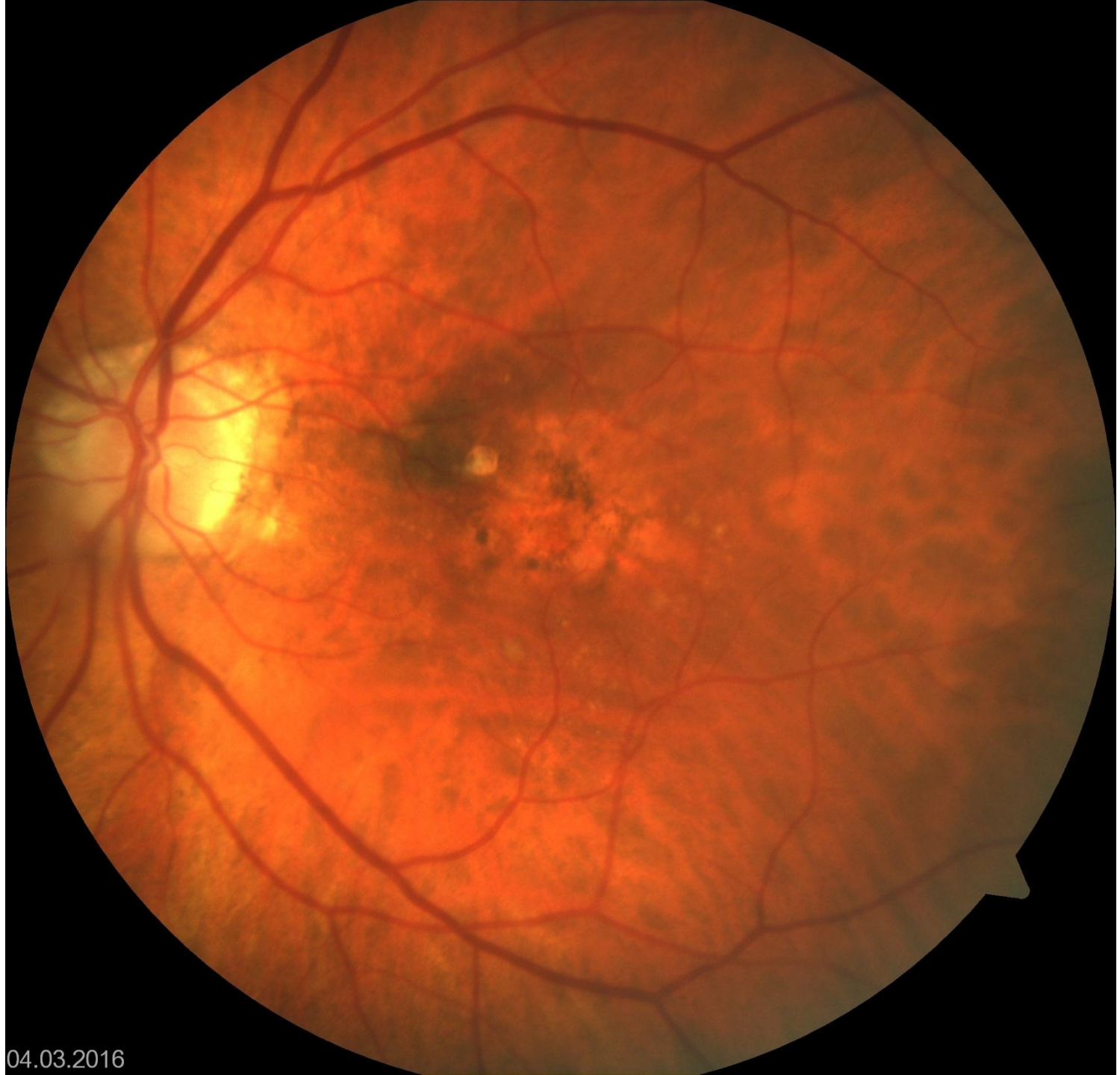
09/23/2020



AMD_54, 75 years at baseline

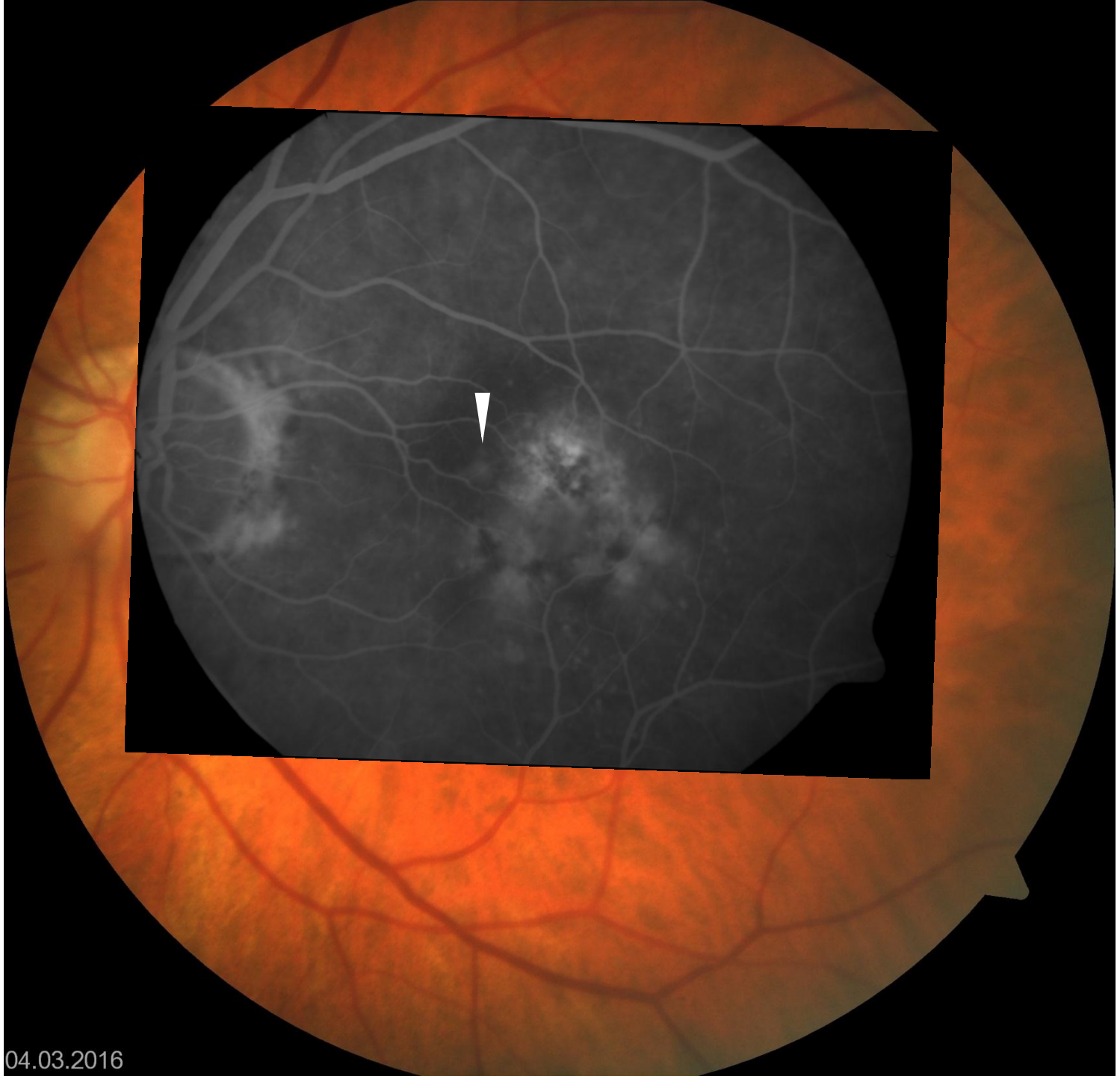
In this patient, a CNV was found at baseline in FAG as well as OCT. Furthermore, there was a hyperreflective spot in CFP (white arrowhead) which did not show fluorescein leakage at baseline (however at follow up) and was hyperfluorescent in SSC but hypofluorescent in LSC (FAF, not FAG). Long FAF lifetimes, along with a loss of RPE on top of an RPE elevation (druse or RPE detachment?), non-reflective in OCT, indicate a beginning atrophy. 20 months later, OCT, FAF, and FLIO clearly show the atrophy. Also the macula starts to become atrophic (RPE migration and penetration of the laser into the choroid in OCT). FAG shows persisting leakage and this eye is under treatment with Ranibizumab (28 injetions so far). Most interestingly, the leakage is surrounded by a hyperfluorescent area which shows absence of the outer retina in OCT (called the “floodpain” phenomenon by Zanzottera et al., Retina 36 (2016) S12-S25) **.** This outer retinal atrophy (ORA) showed short FAF lifetimes. **As RPE and sub-RPE structures were intact (more or less), might it be that the long lifetimes, typical for AMD, are related to fluorophores in the outer retina?**

04/03/2016



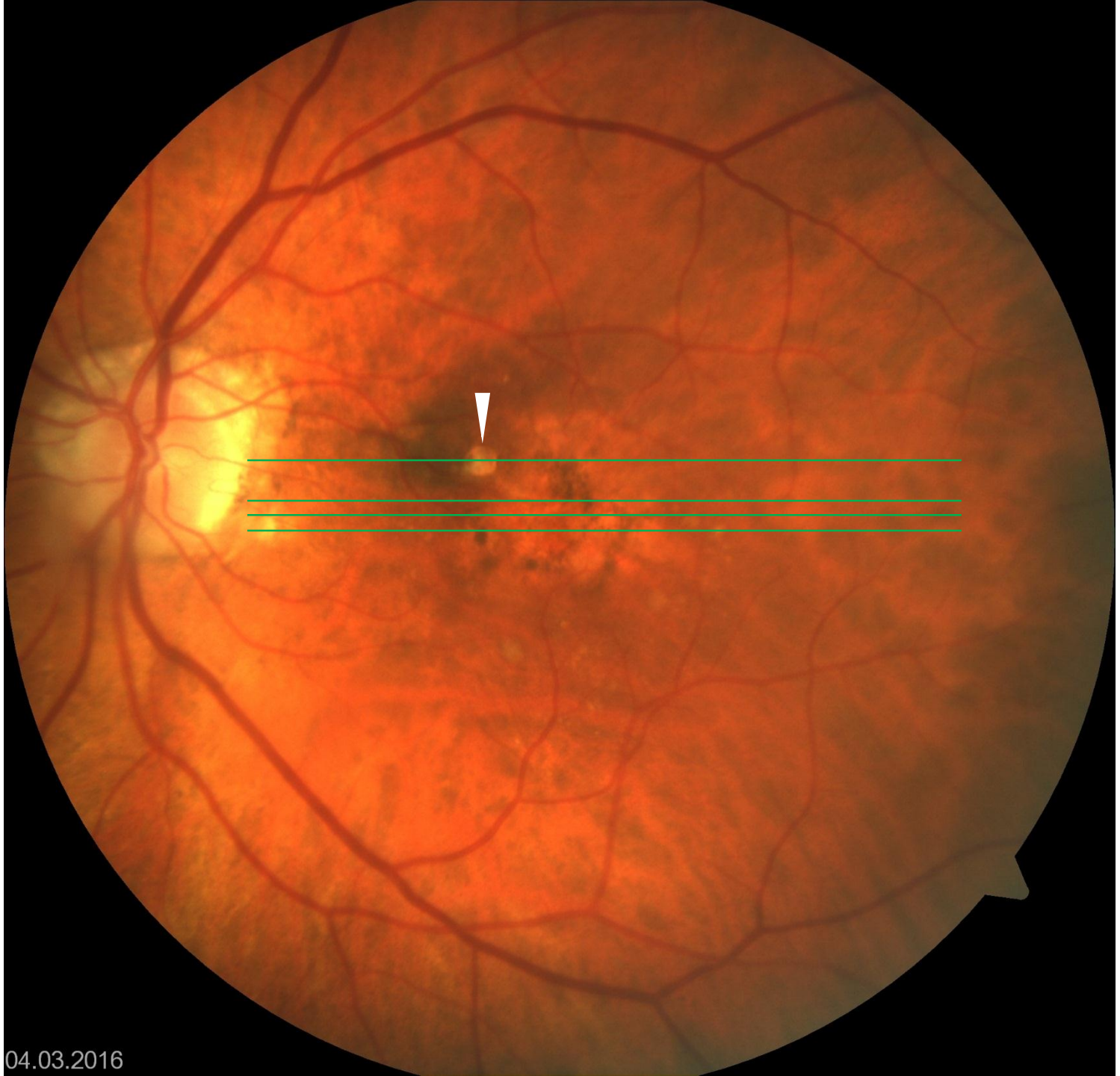
CFP

01/23/2015

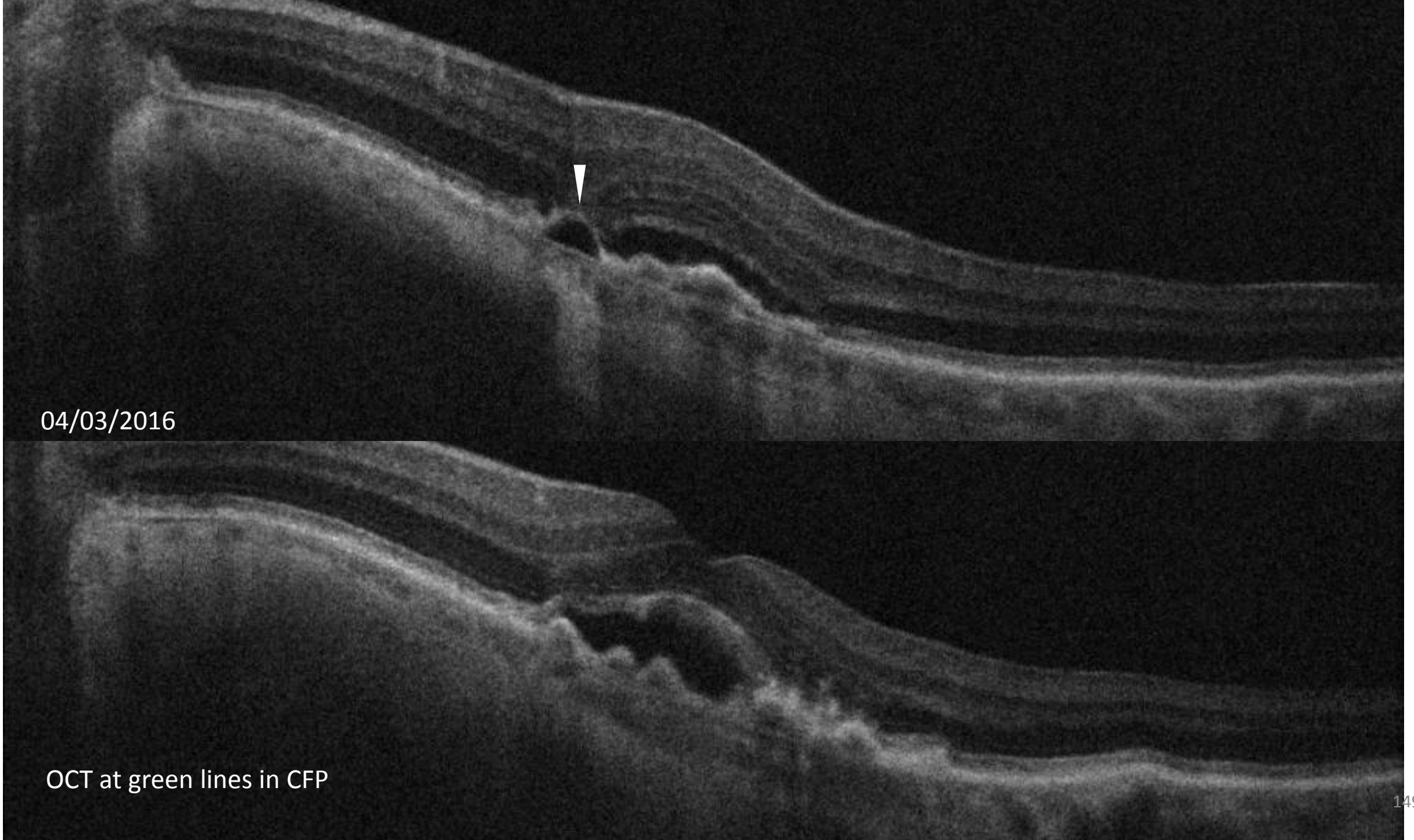


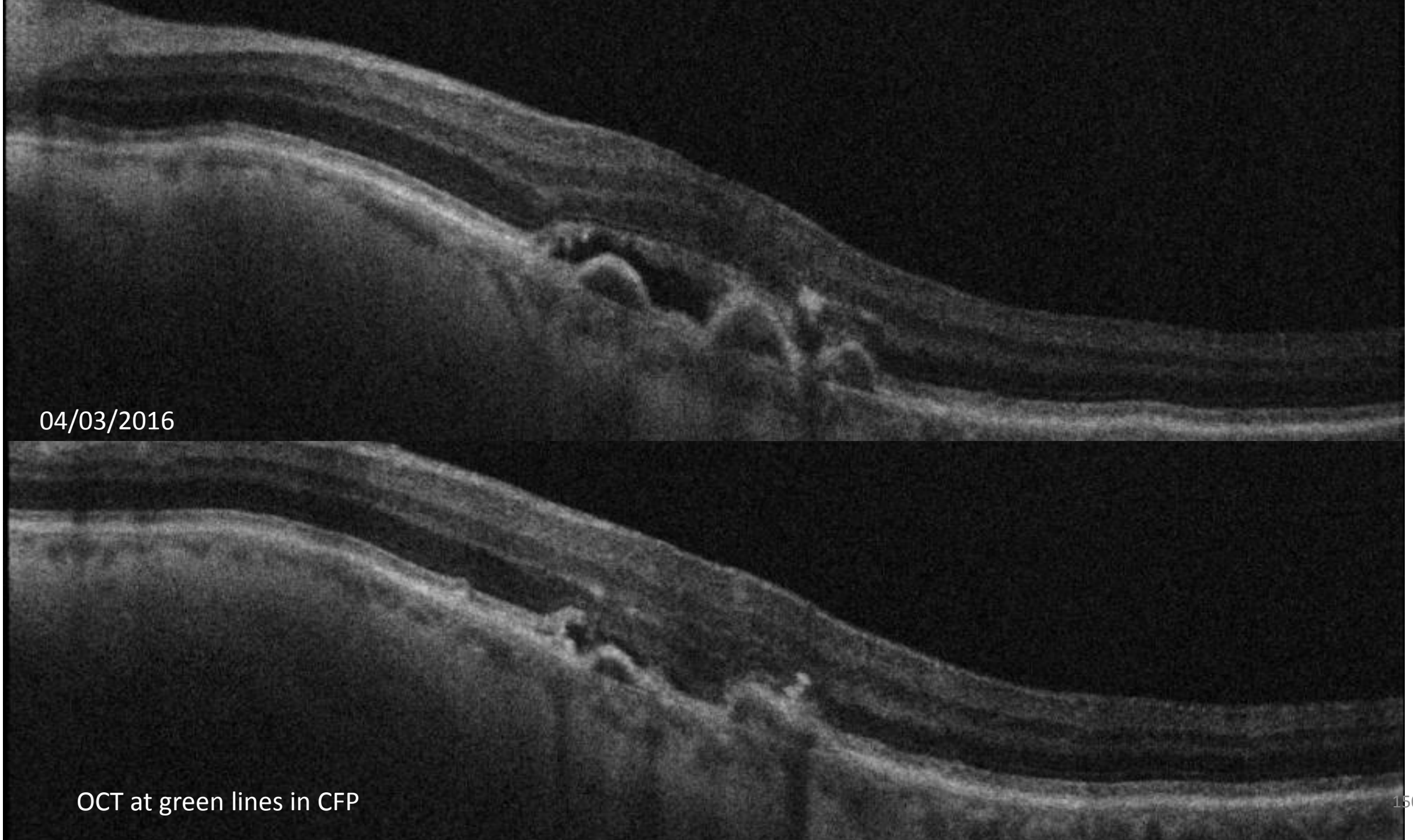
FAG late phase

04/03/2016

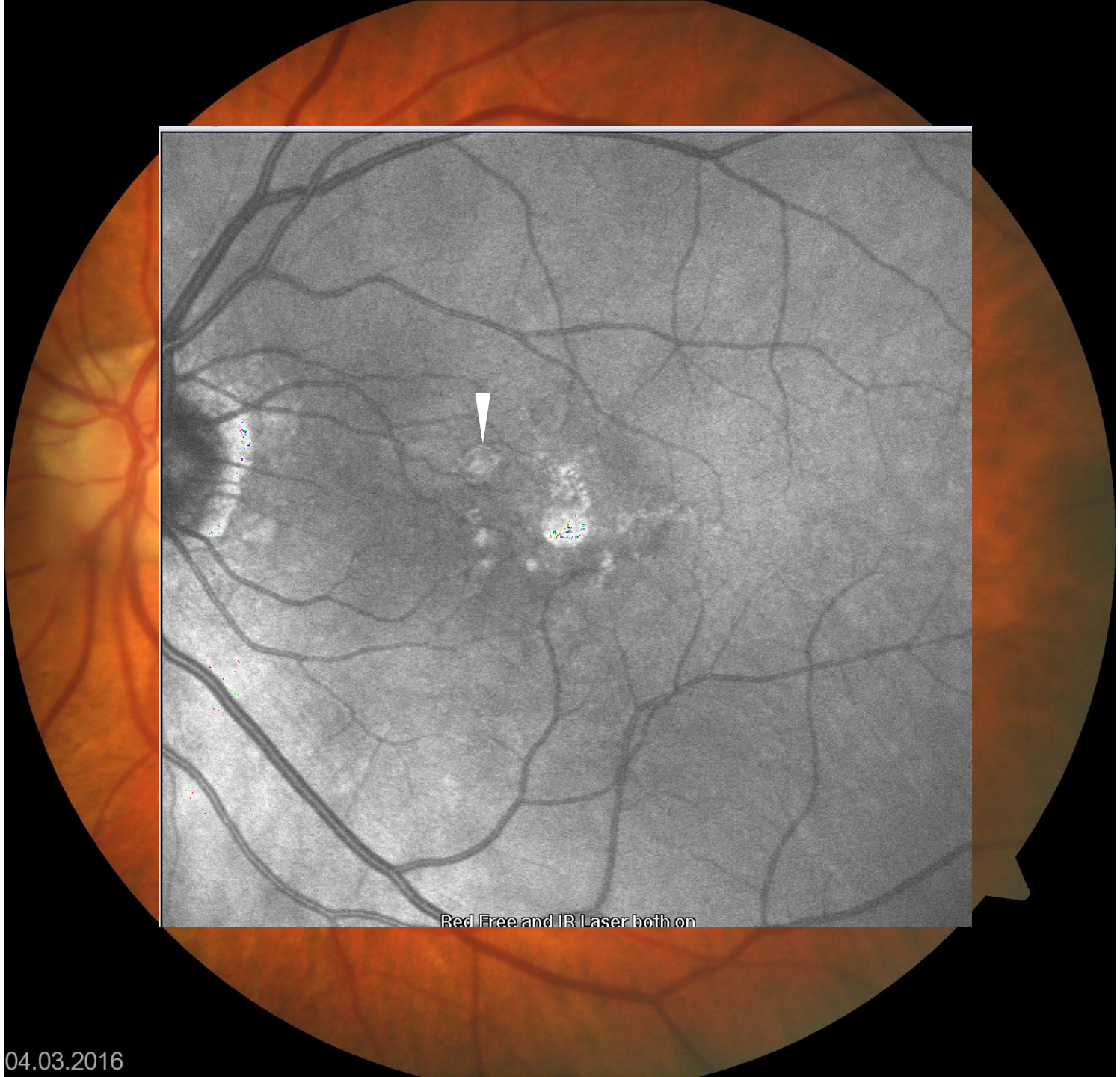


CFP


04/03/2016
OCT at green lines in CFP

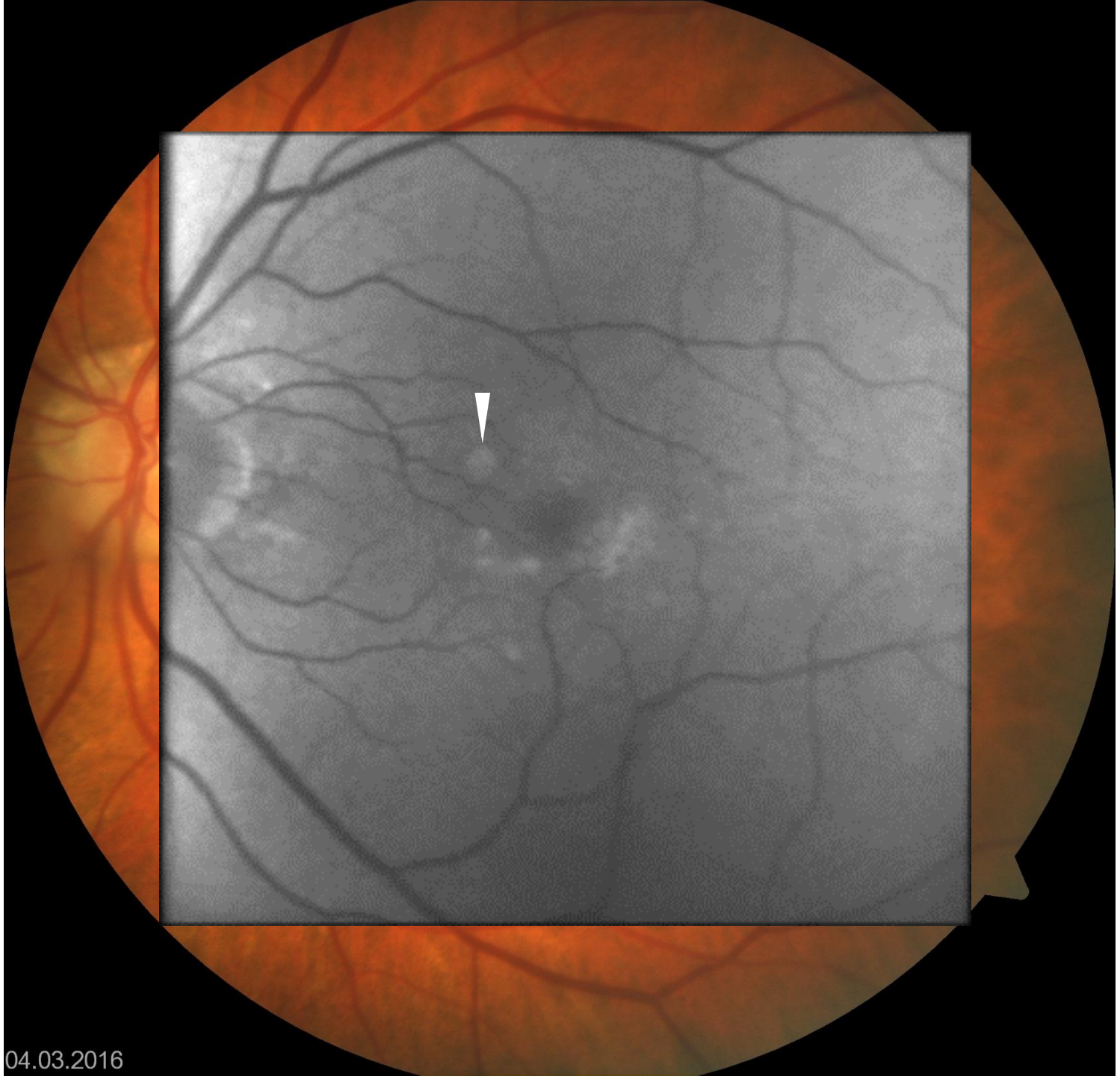

04/03/2016
OCT at green lines in CFP

04/03/2016



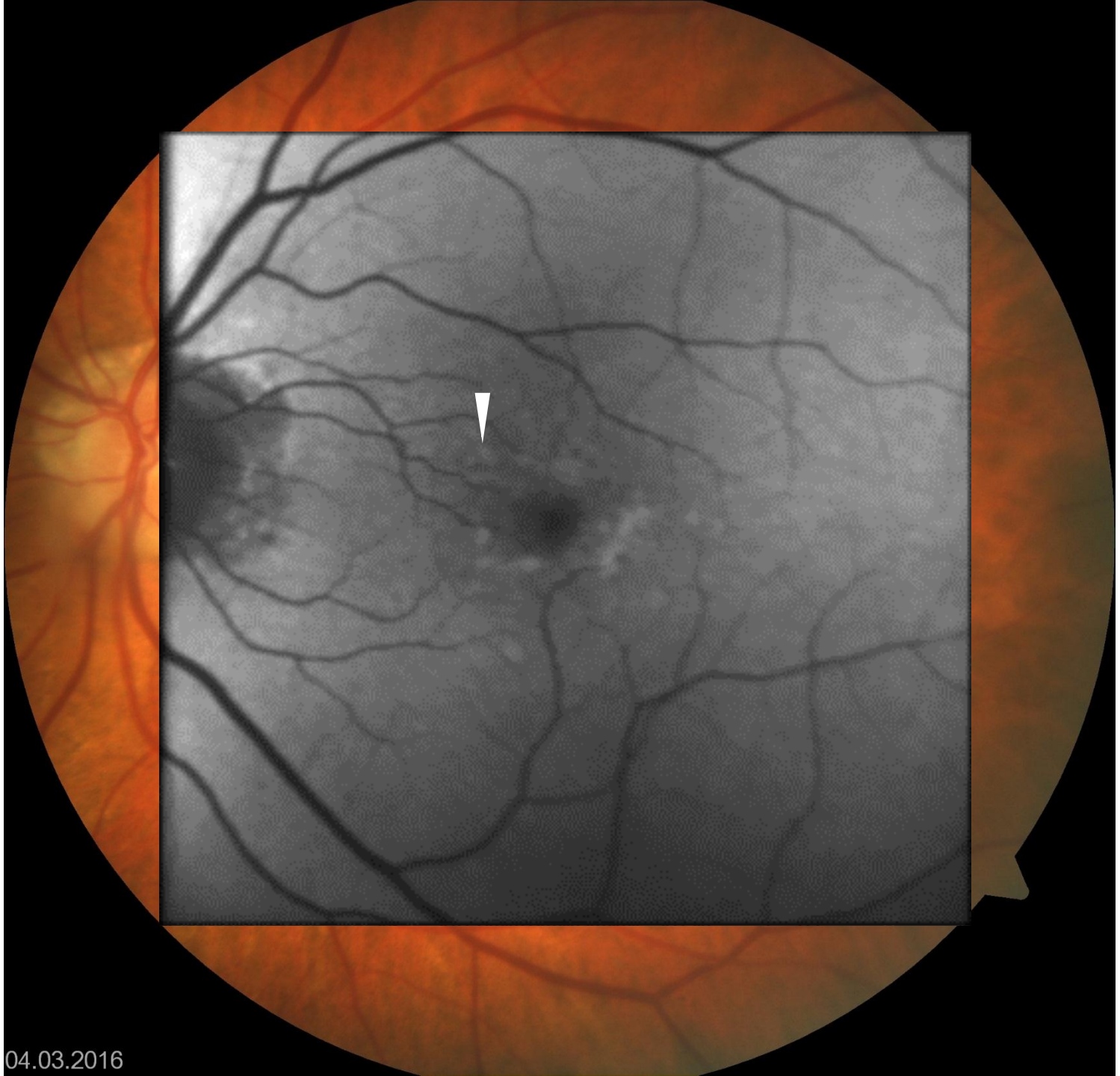
IR

04/03/2016



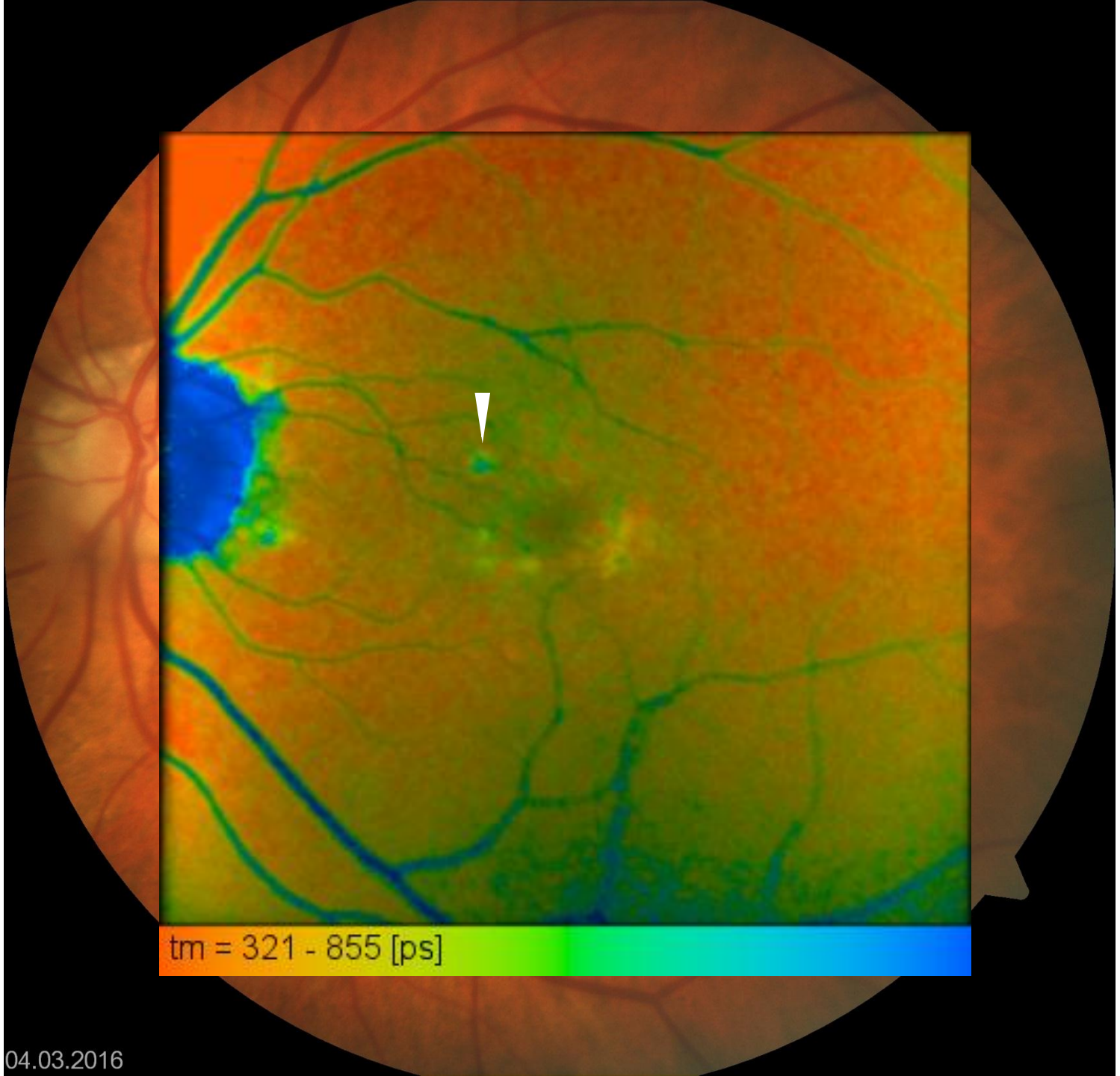
FAF SSC

04/03/2016



FAF LSC

04/03/2016



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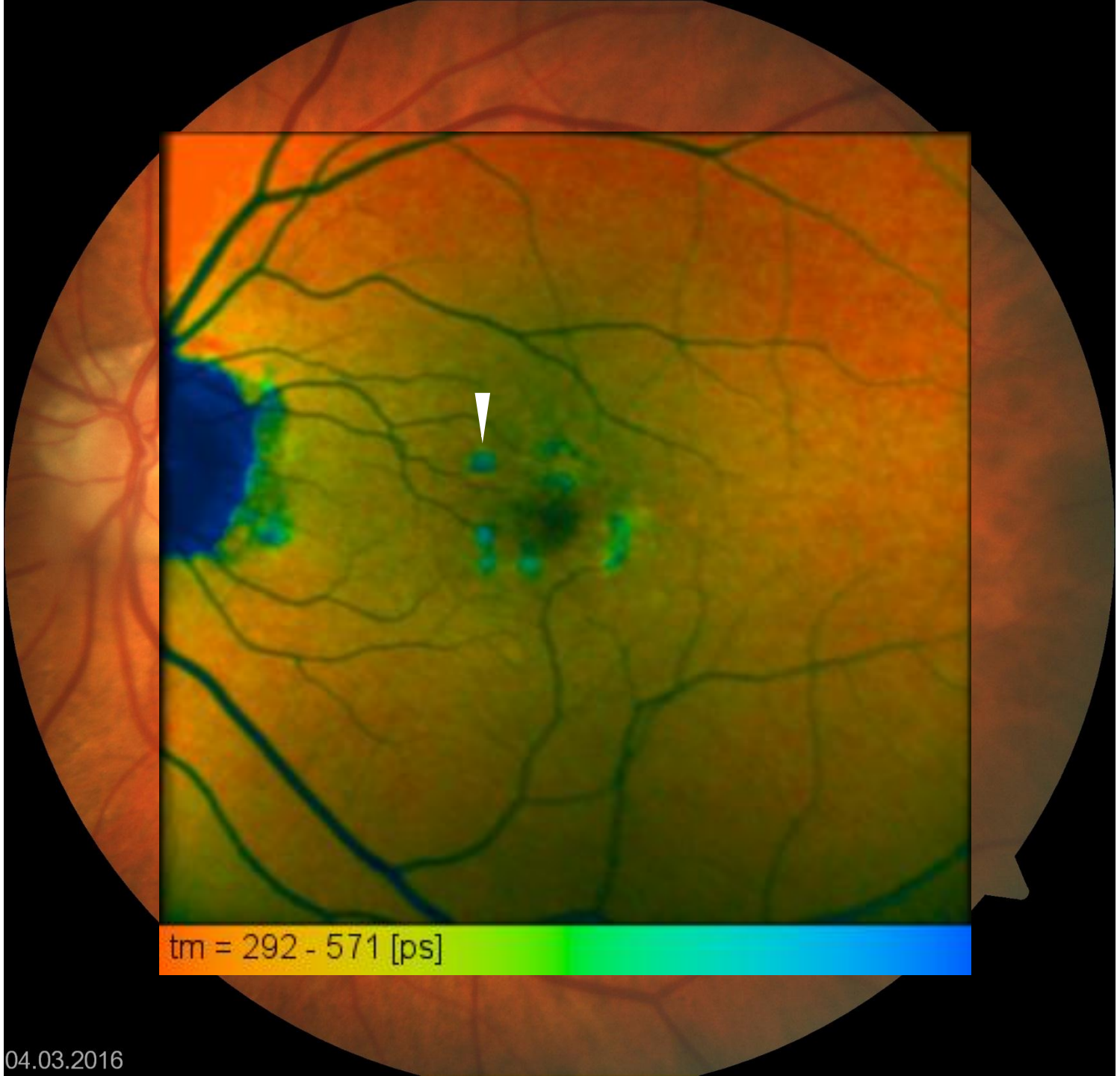
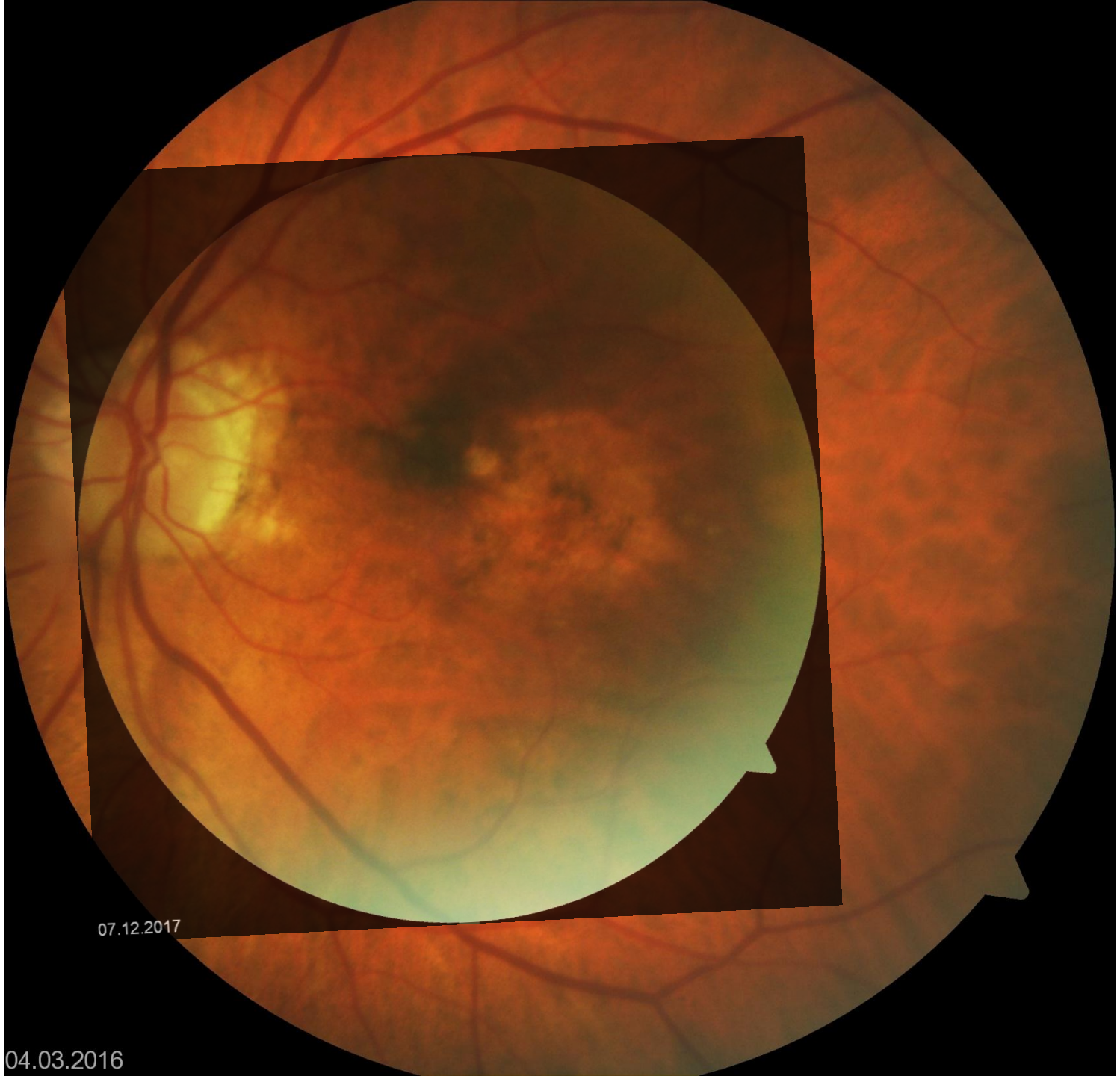
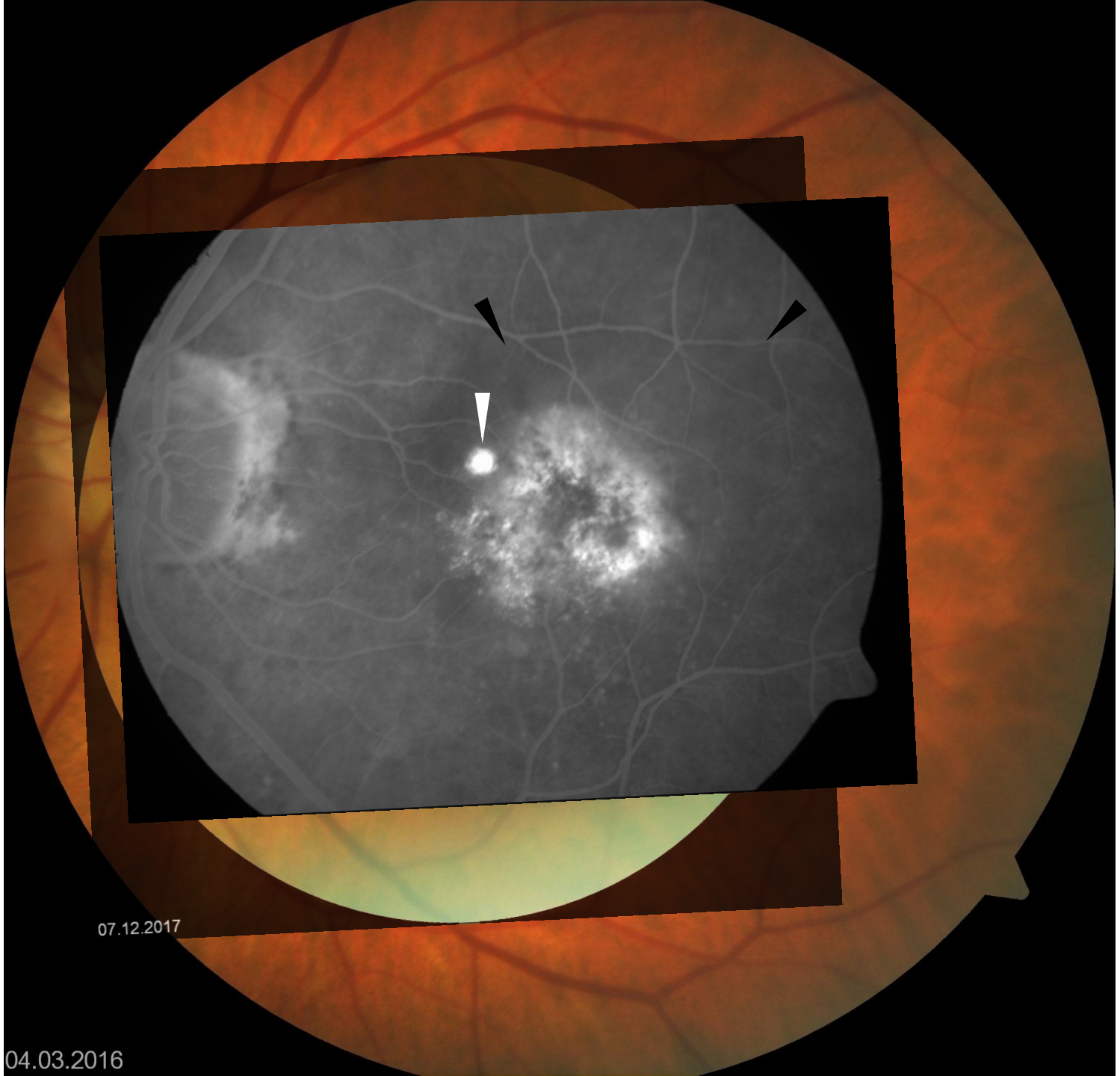


12/07/2017



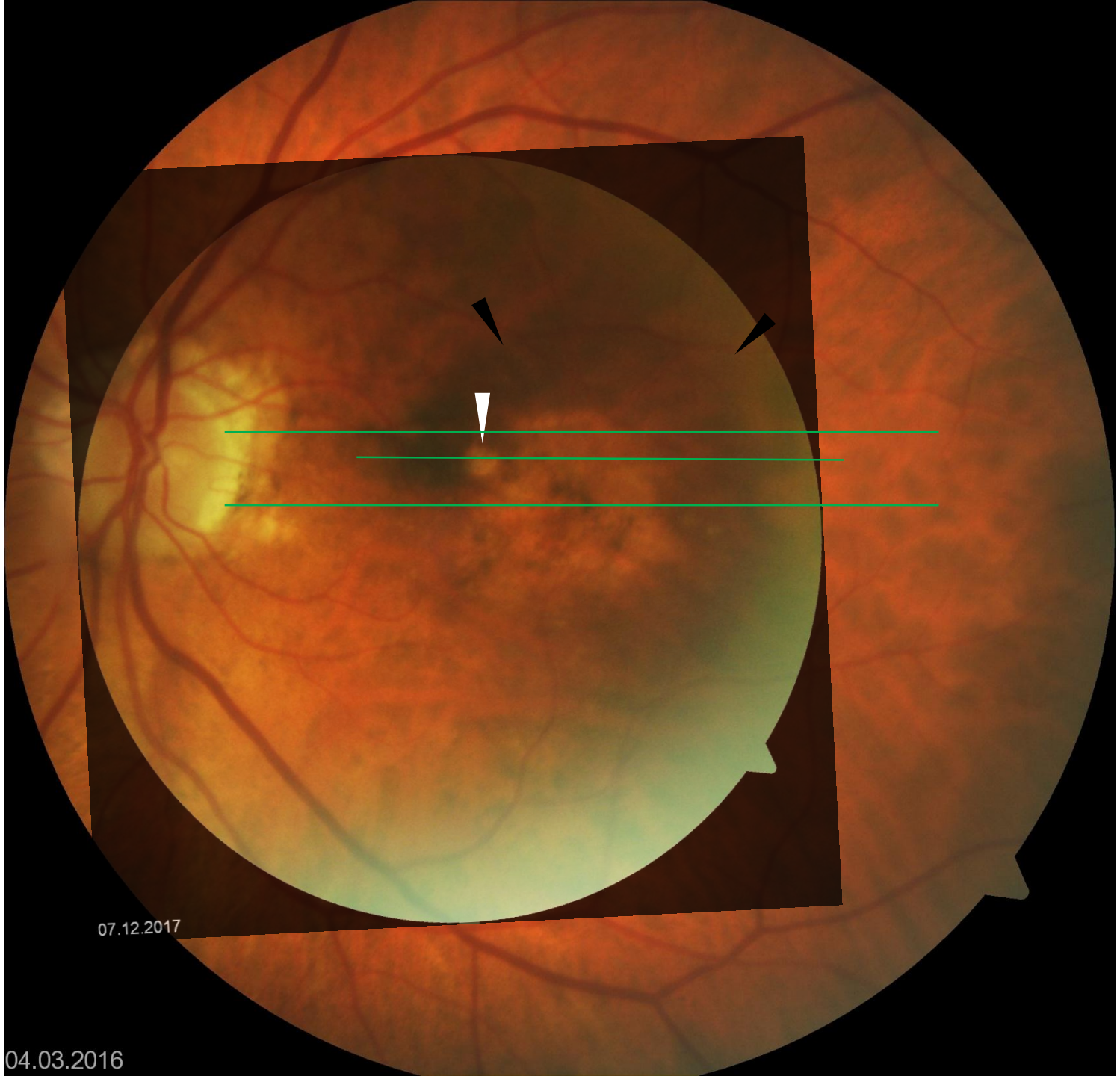
CFP

07/28/2017

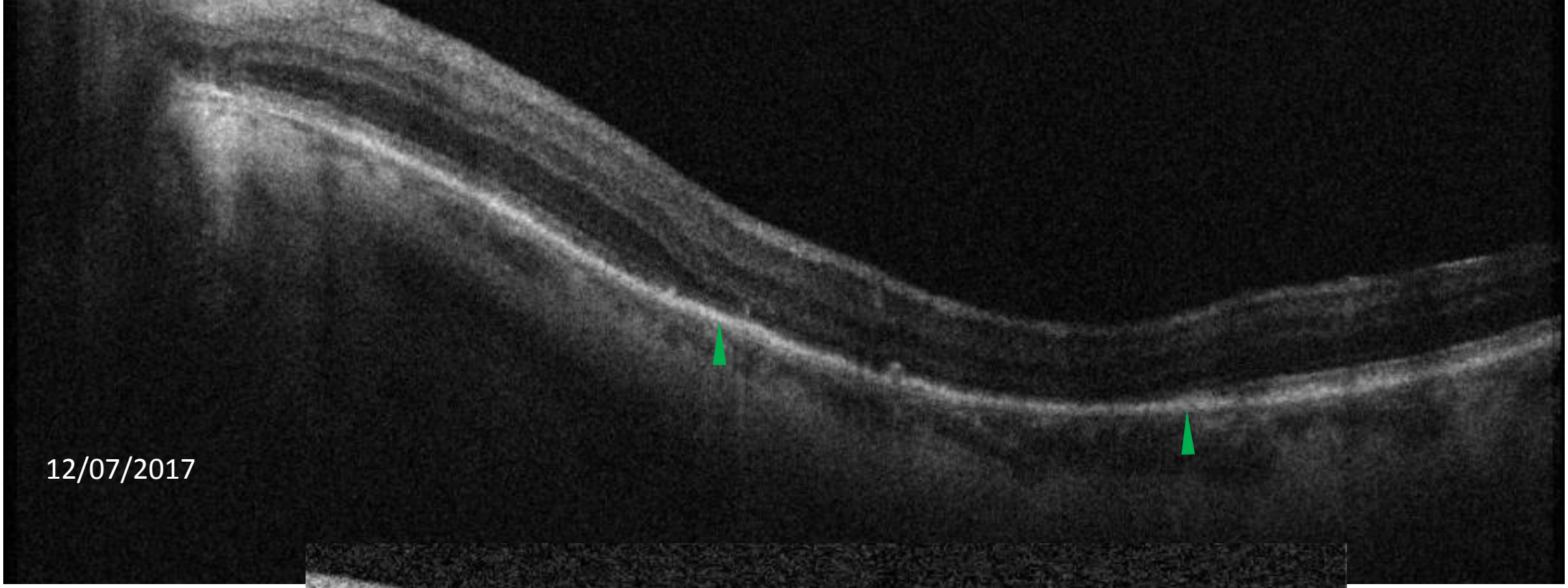


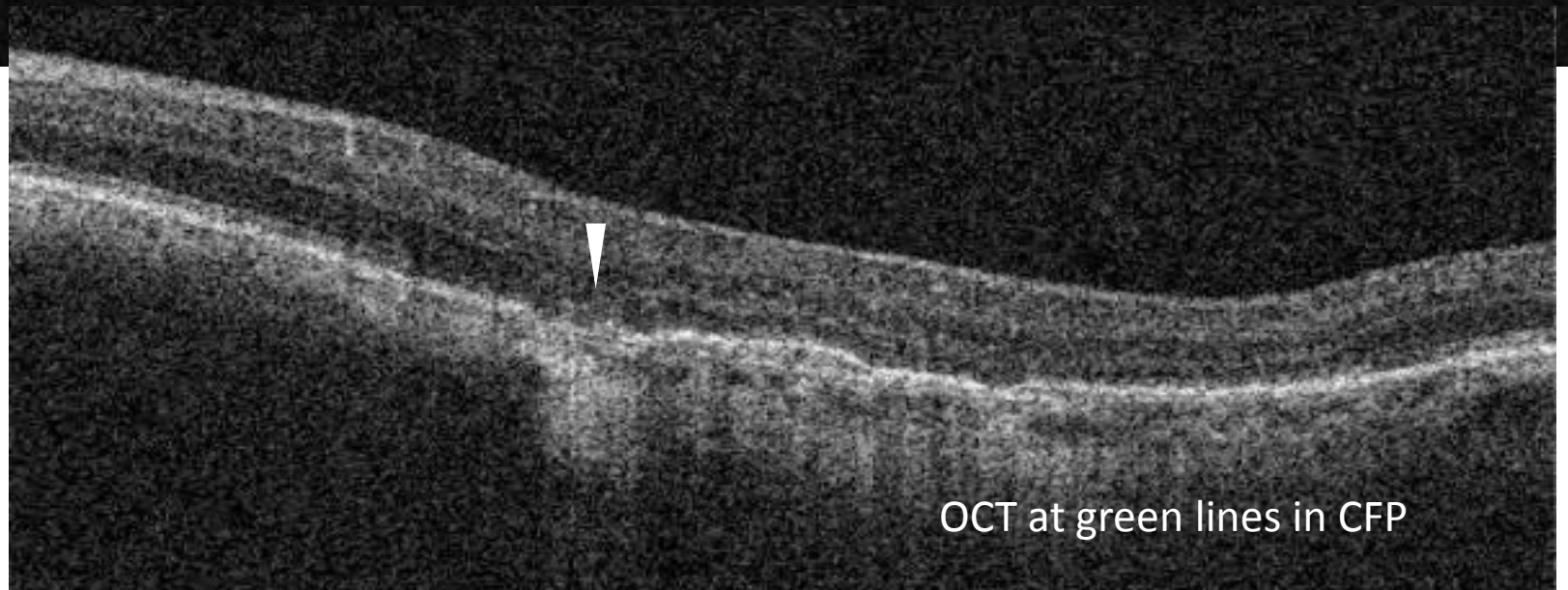
FAG late phase

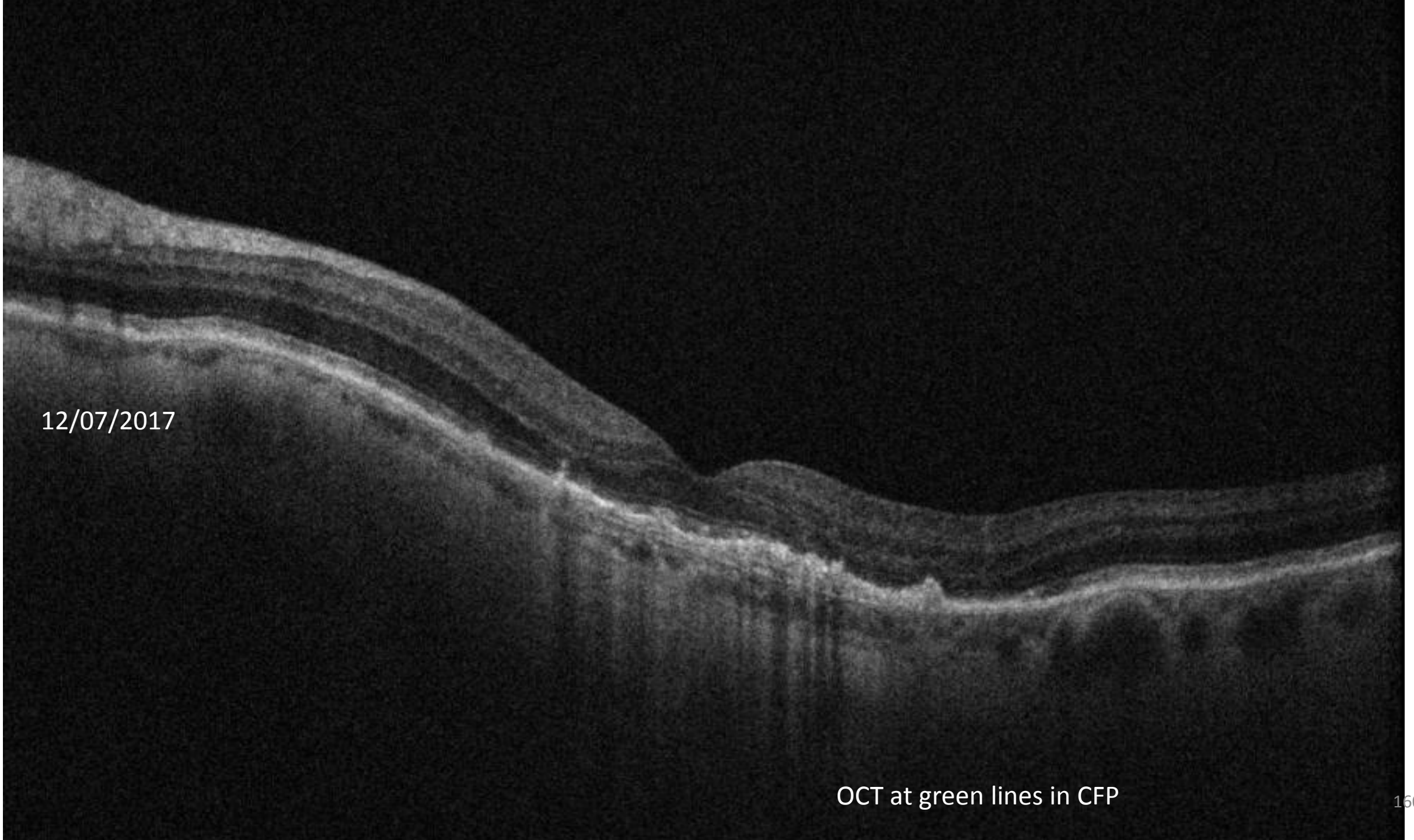
12/07/2017



CFP

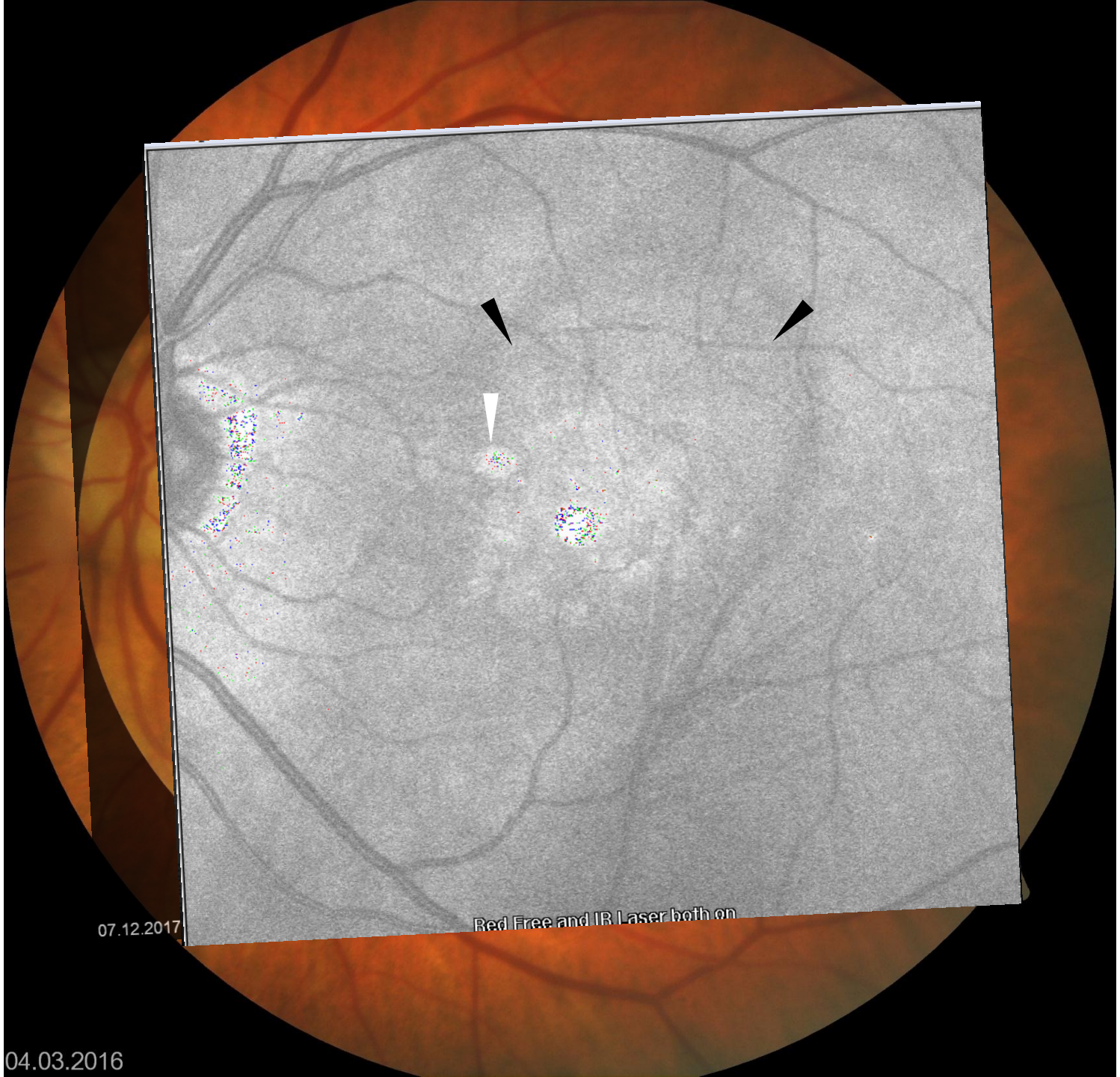

12/07/2017


OCT at green lines in CFP



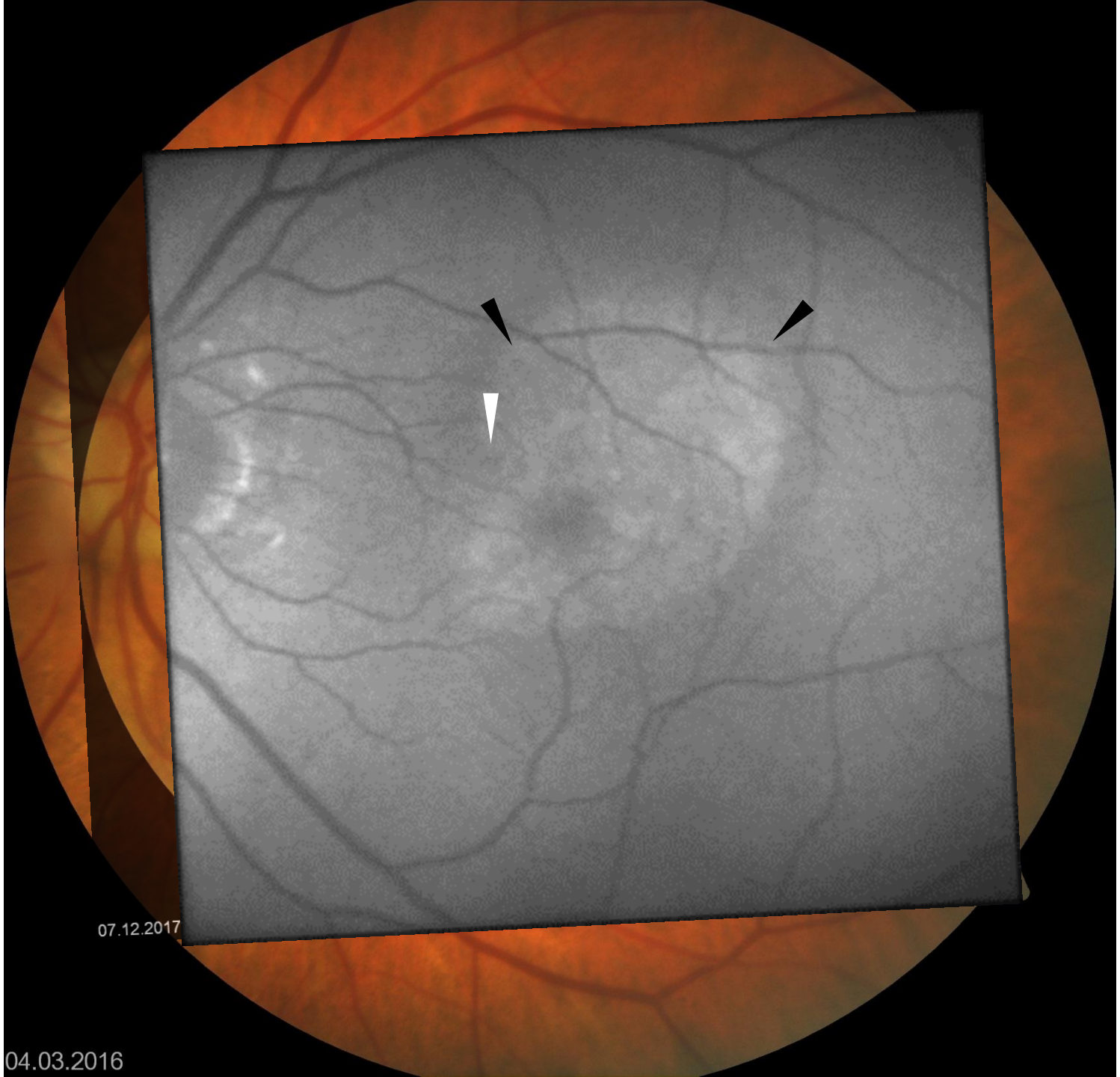
OCT at green lines in CFP

12/07/2017



IR

12/07/2017



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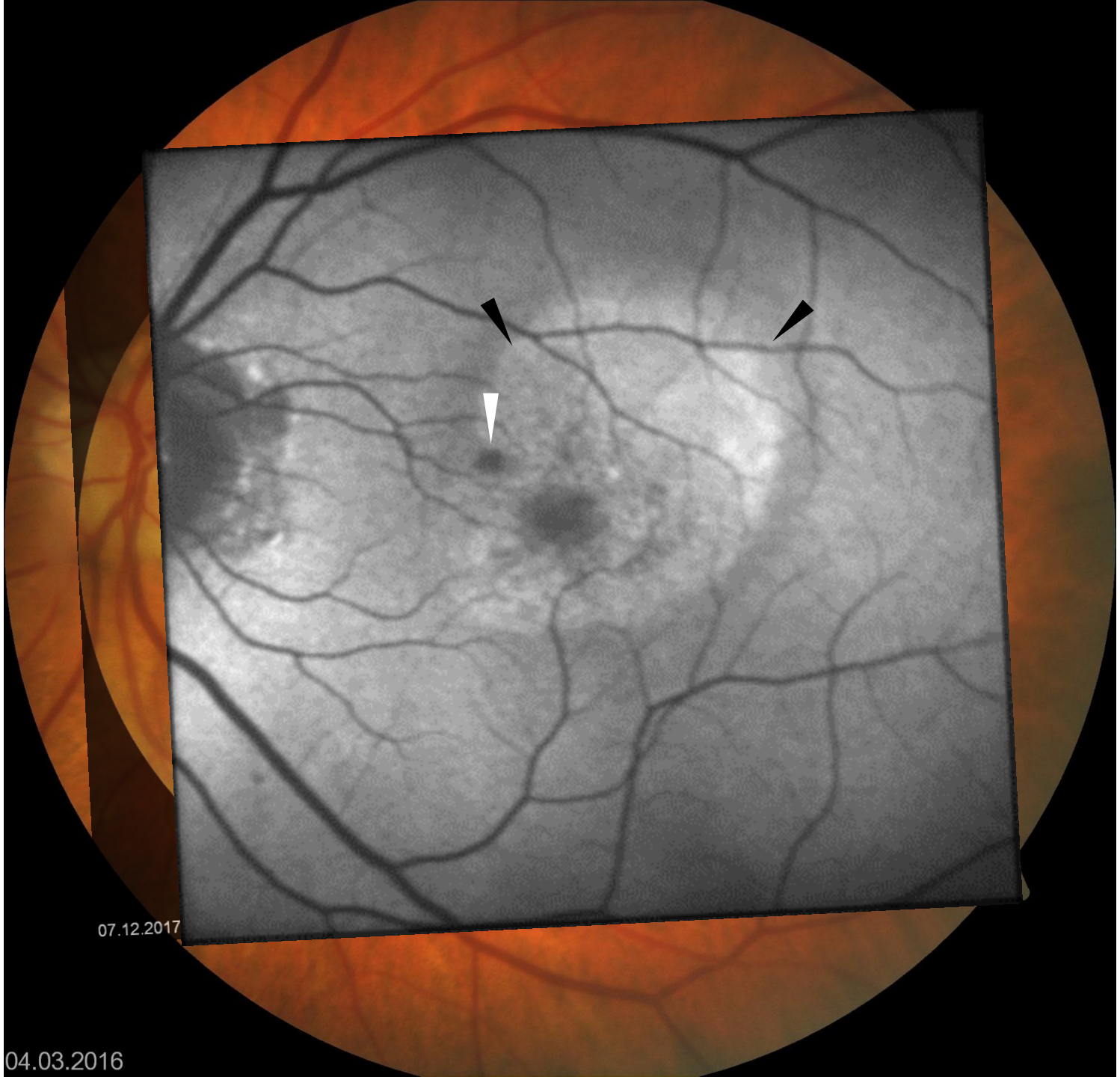
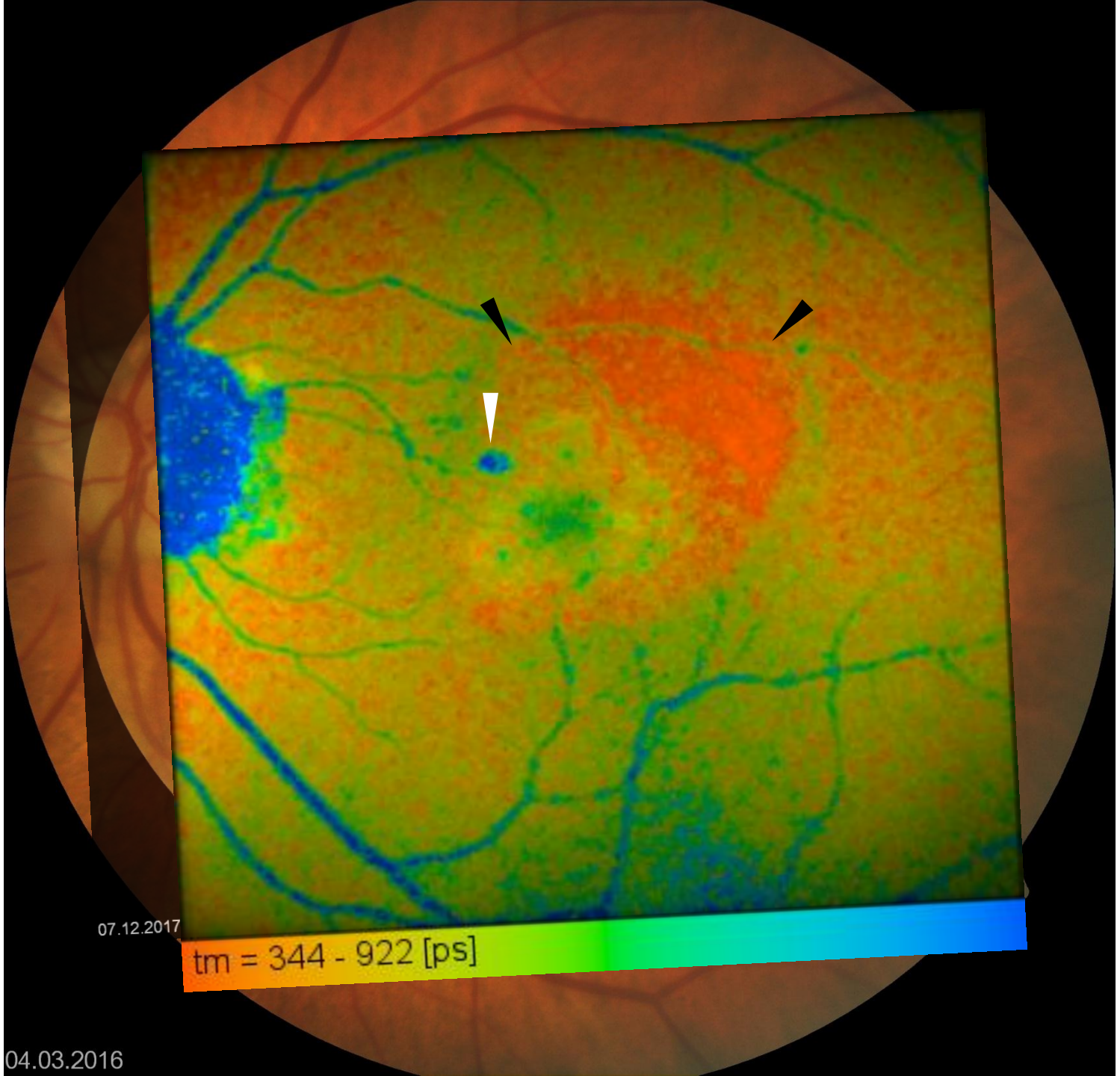
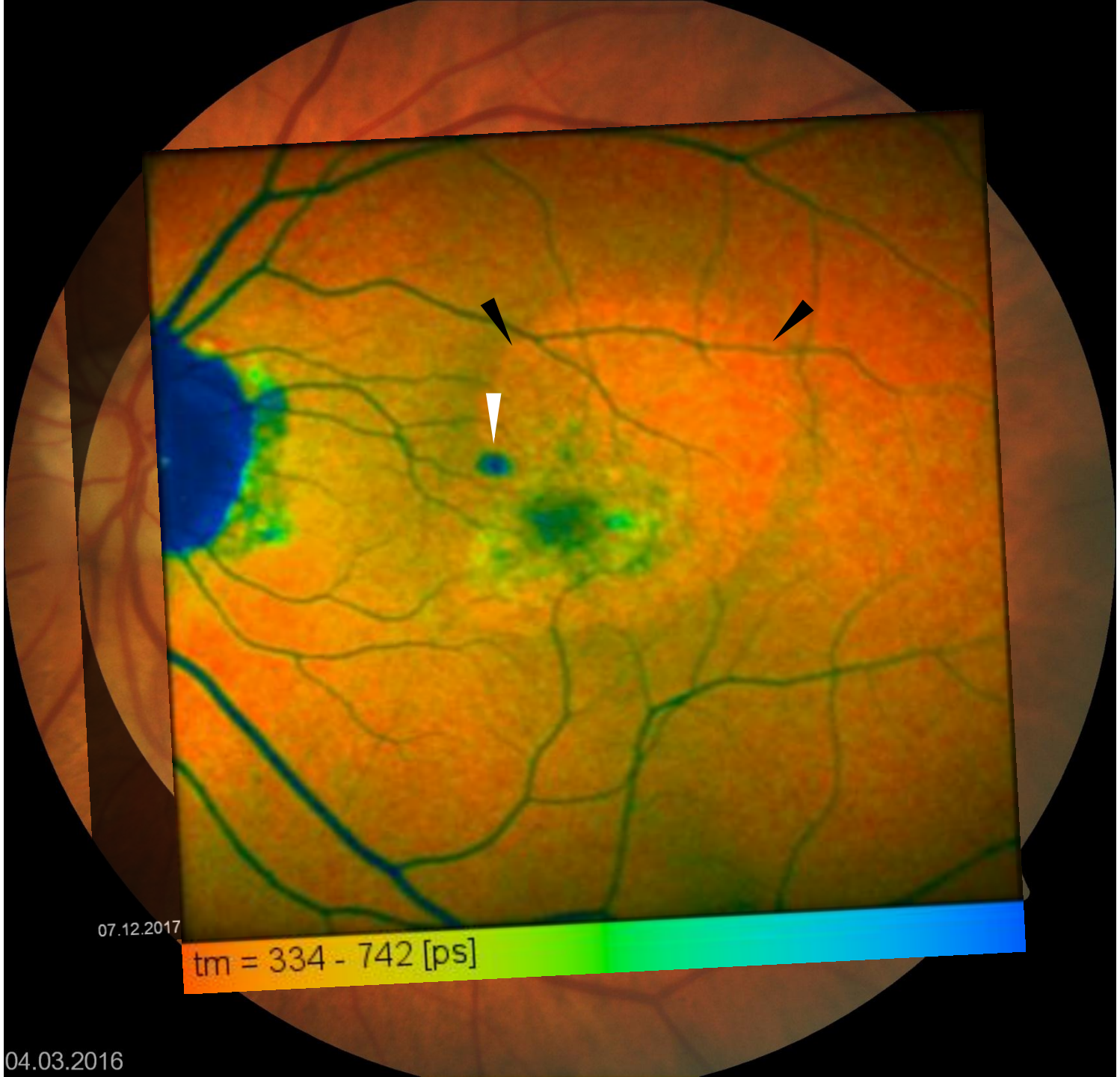


FAF LSC

12/07/2017



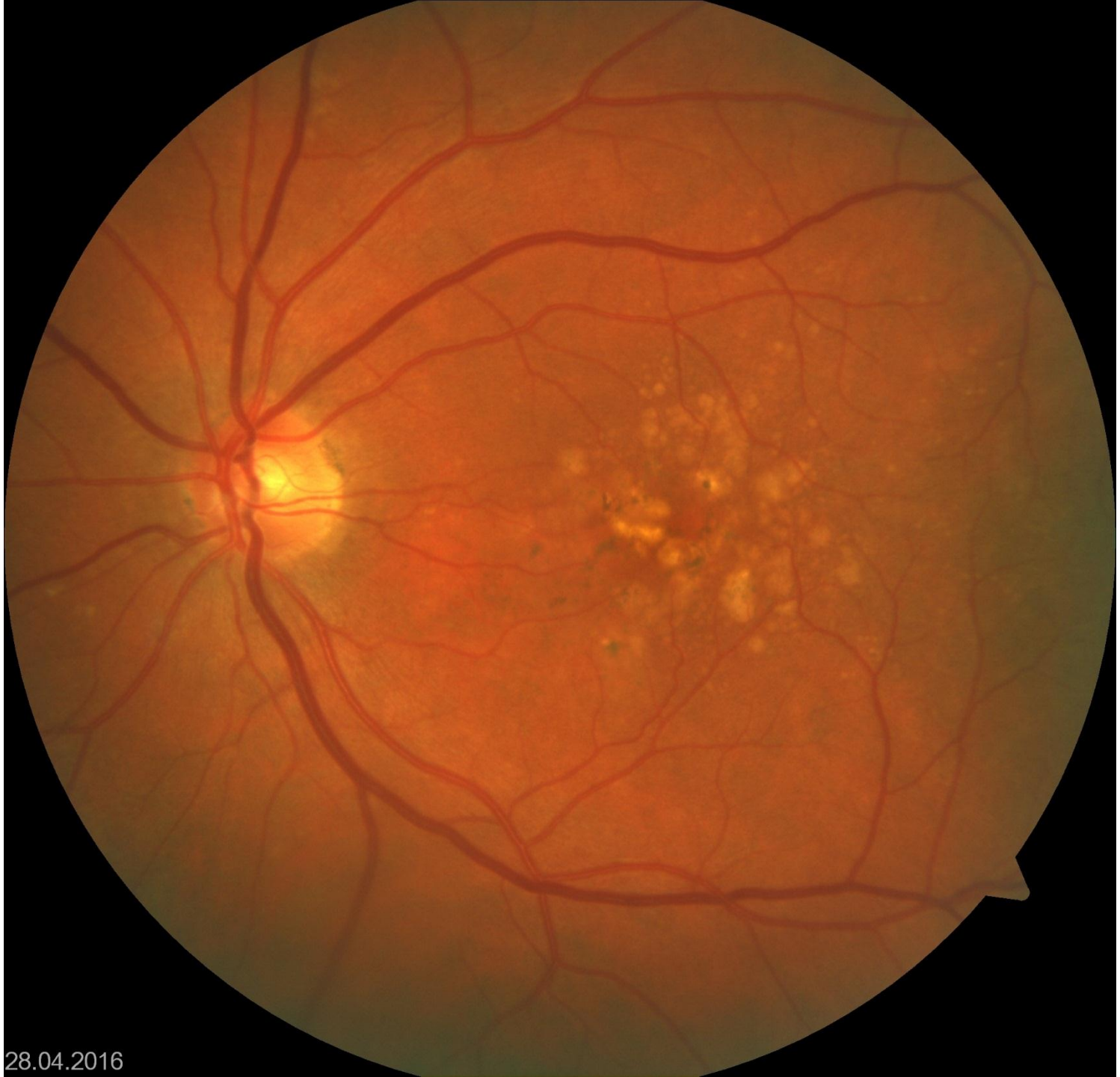
12/07/2017



AMD_67, 68 years at baseline

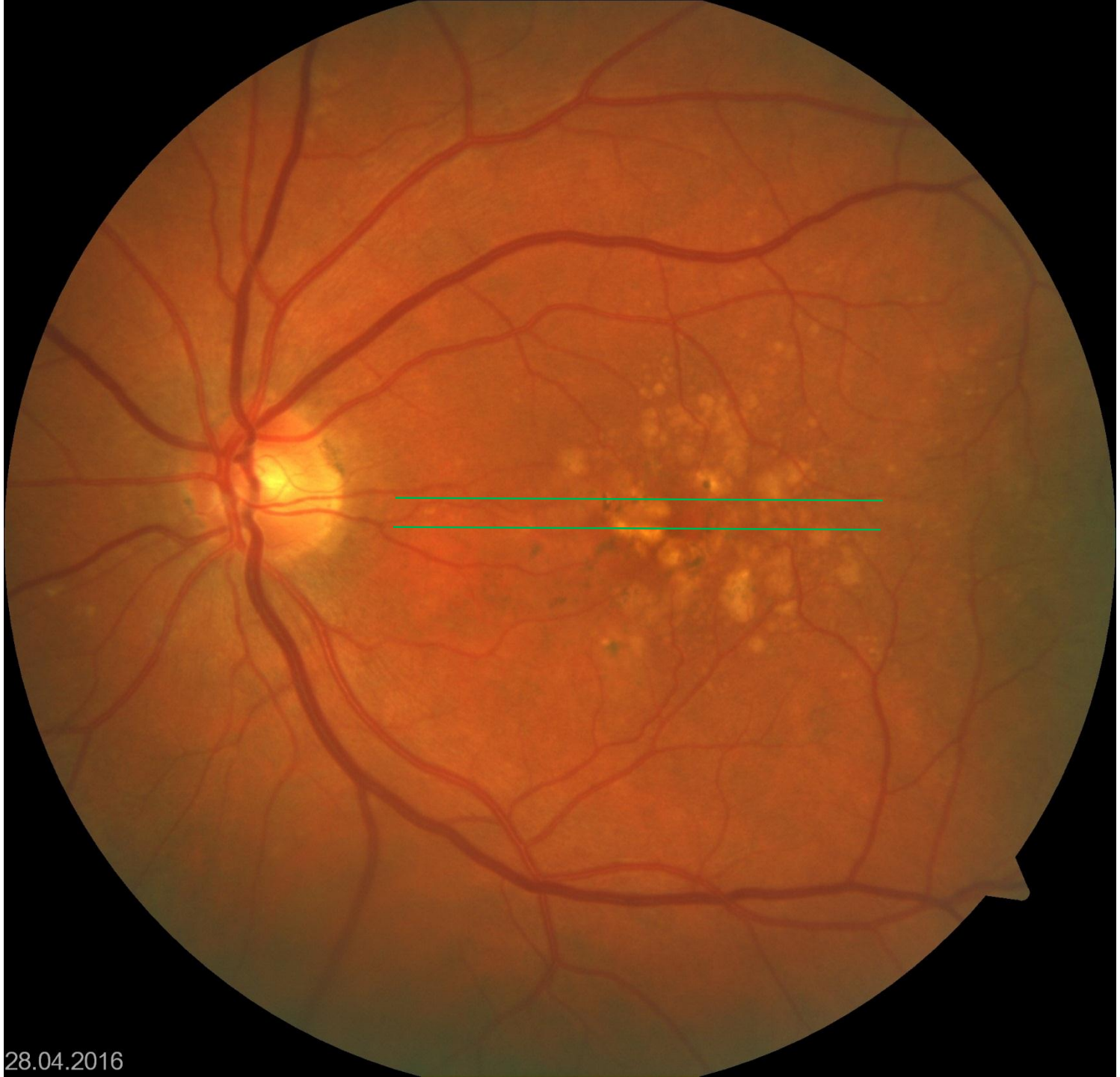
This Patient developed GA during follow up. The long FAF lifetime, seen at 8 o clock in LSC at baseline, might be an early indicator for the progression. The white arrowhead in the follow up images indicates a hyperpigmentation which shows the typical long lifetimes. In OCT, this lesion shows hyperreflectivity near the ILM. **Is it possible that the RPE migrates thus far?** The black arrow heads indicate areas of ORA evident in OCT. As in patient AMD_54, these areas have short lifetimes. **The question remains, if the long-living fluorophores might have gone with the outer retina.** However in contrast to AMD_54 (and other published cases), in this case, the area of ORA is hypofluorescent. Hyper-FAF in the absence of the outer retina is explained by the lack of absorbing photopigments. Whether this holds for FLIO too is unclear because photopigments are greatly bleached during the about 2 minutes scanning needed for a good FLIO image. **But how could hypofluorescence be explained?**

04/28/2016

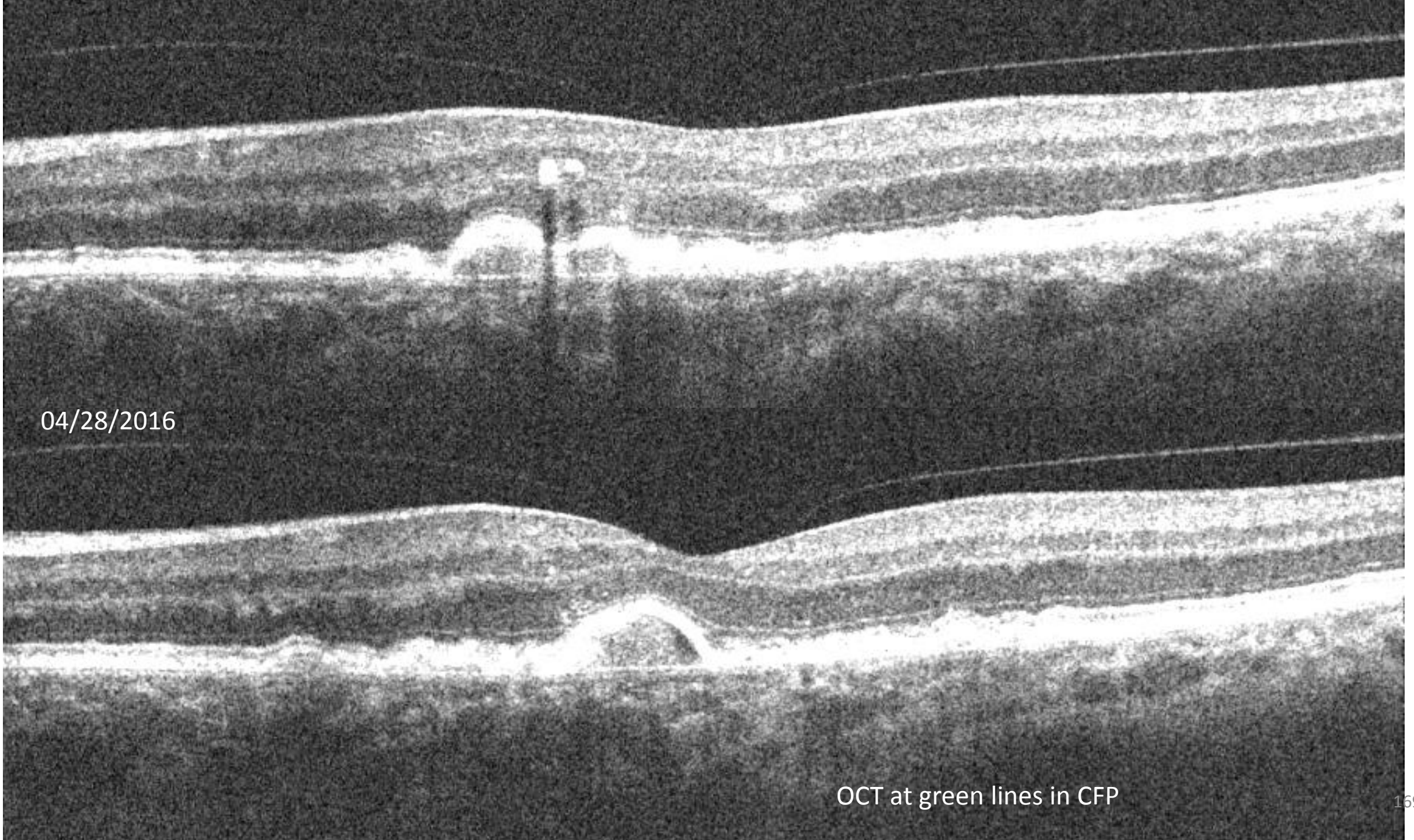


CFP

04/28/2016

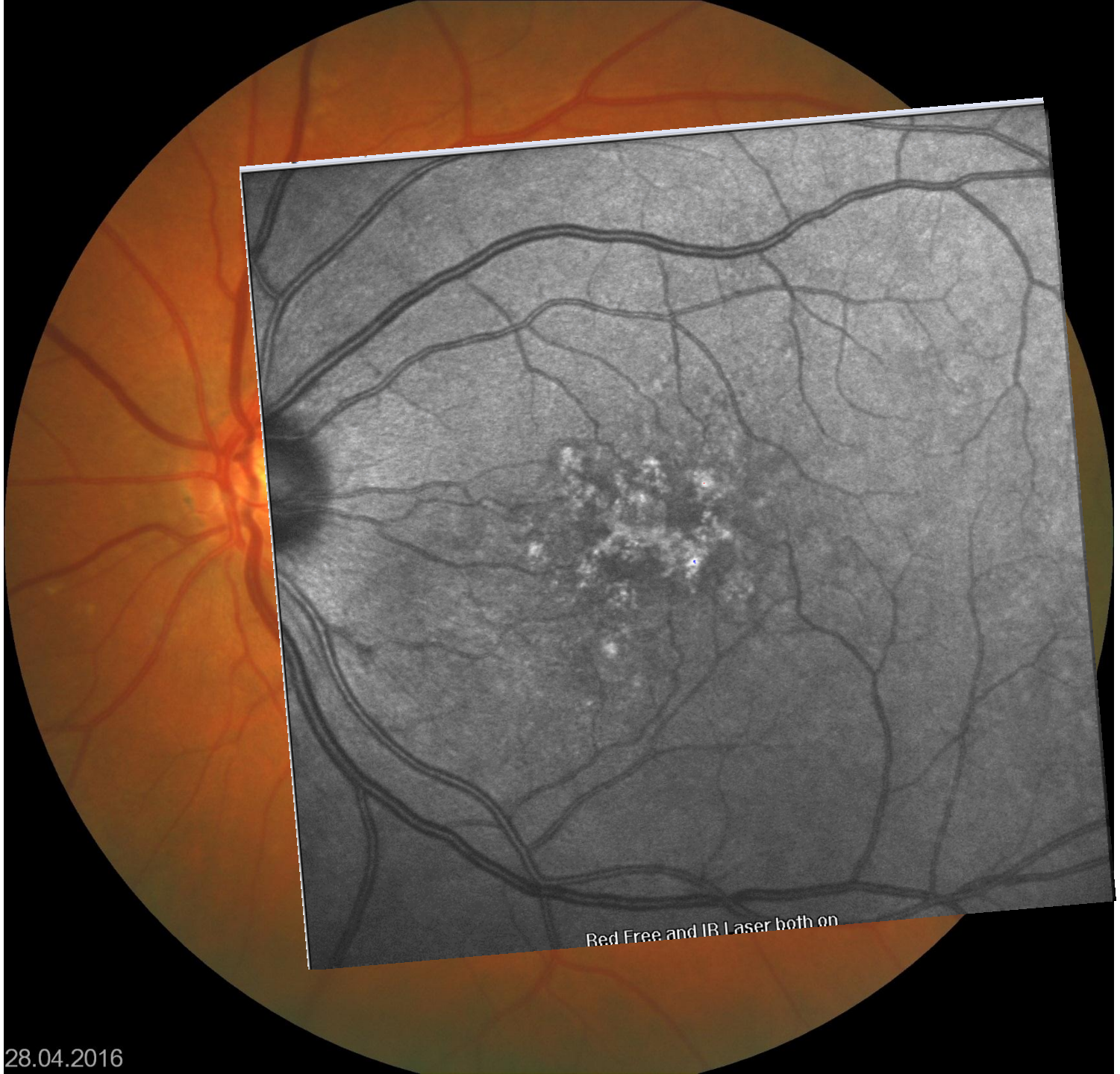


CFP



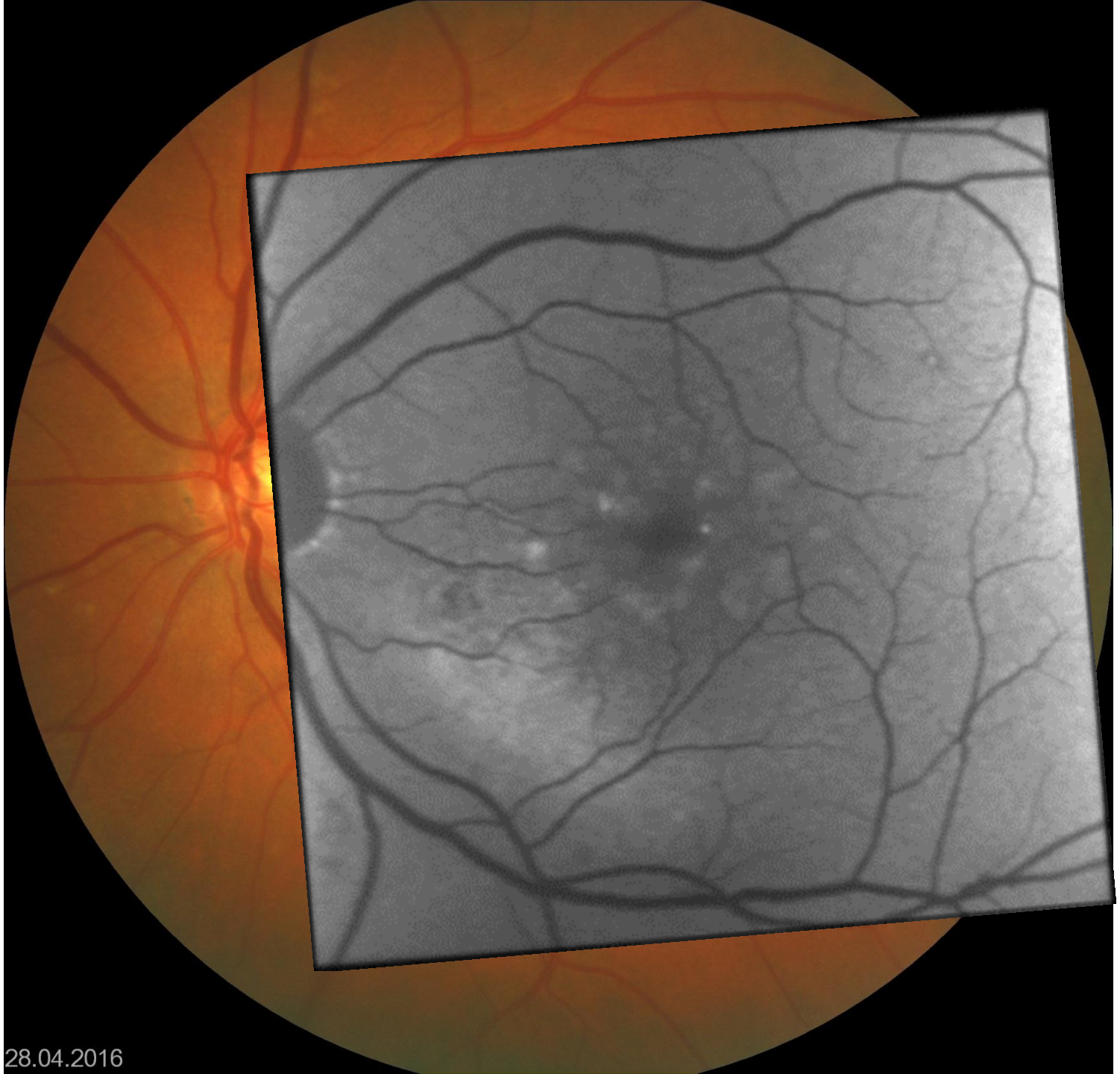
OCT at green lines in CFP

04/28/2016

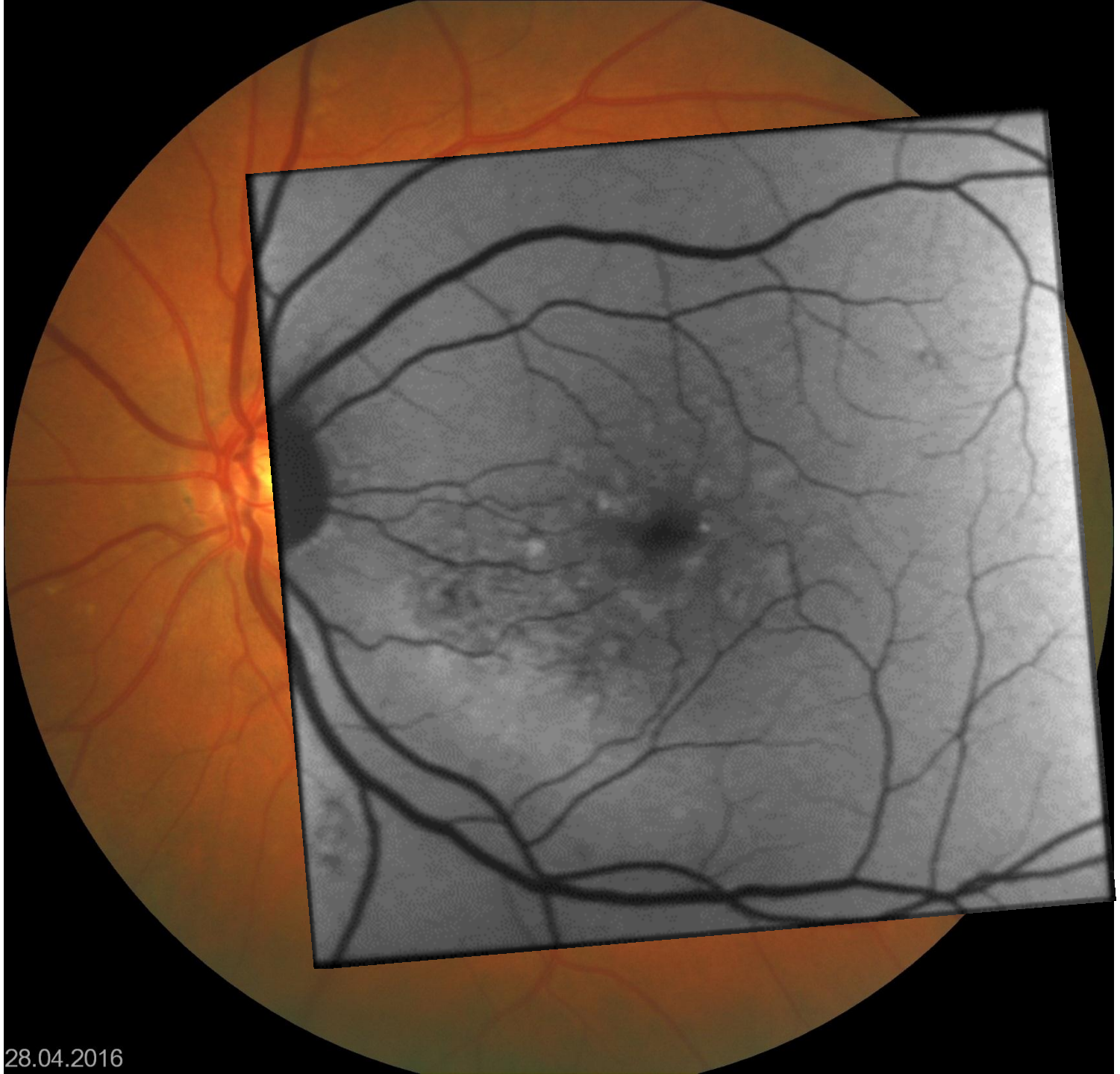


IR

04/28/2016

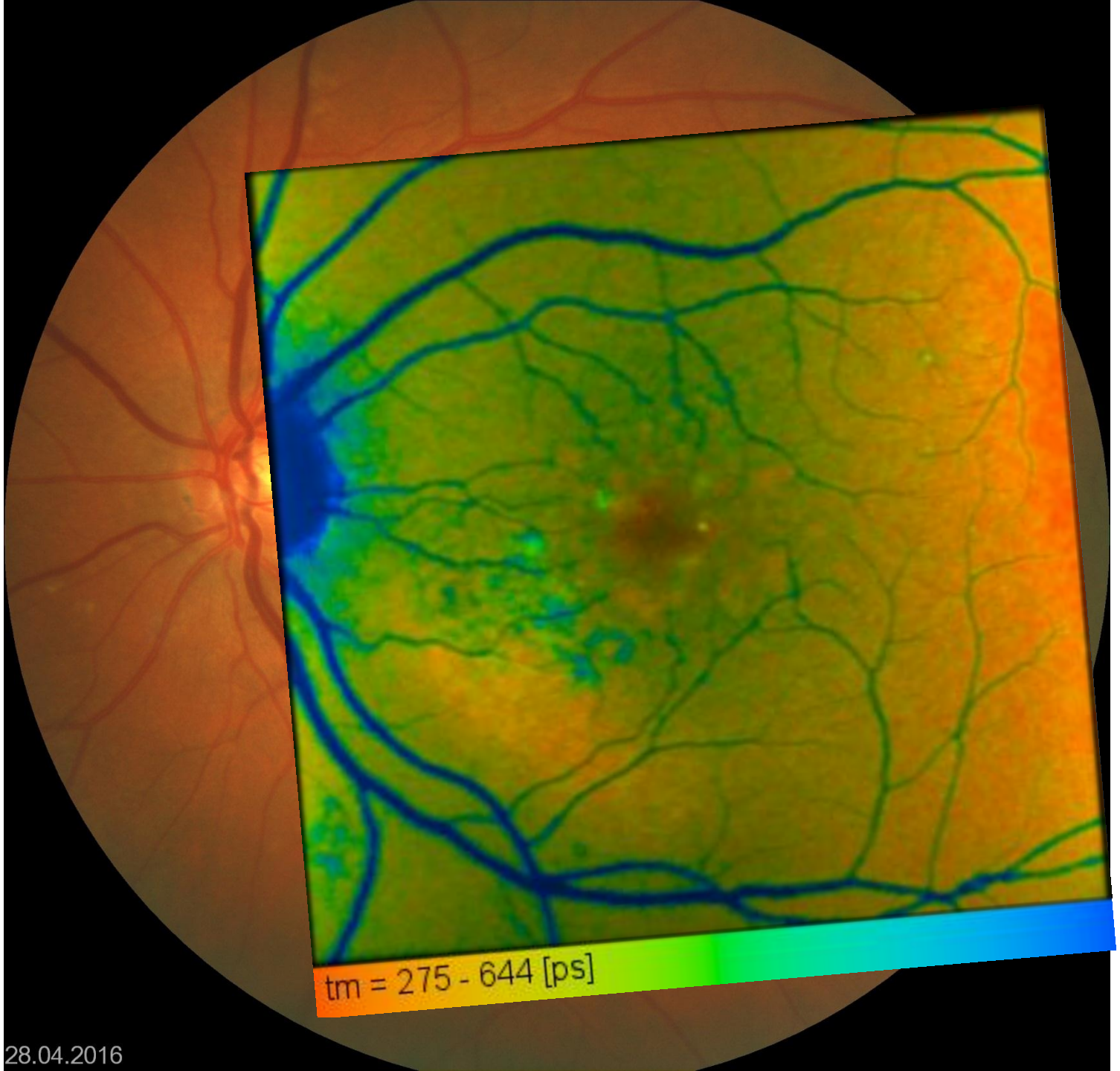


04/28/2016



FAF LSC

04/28/2016



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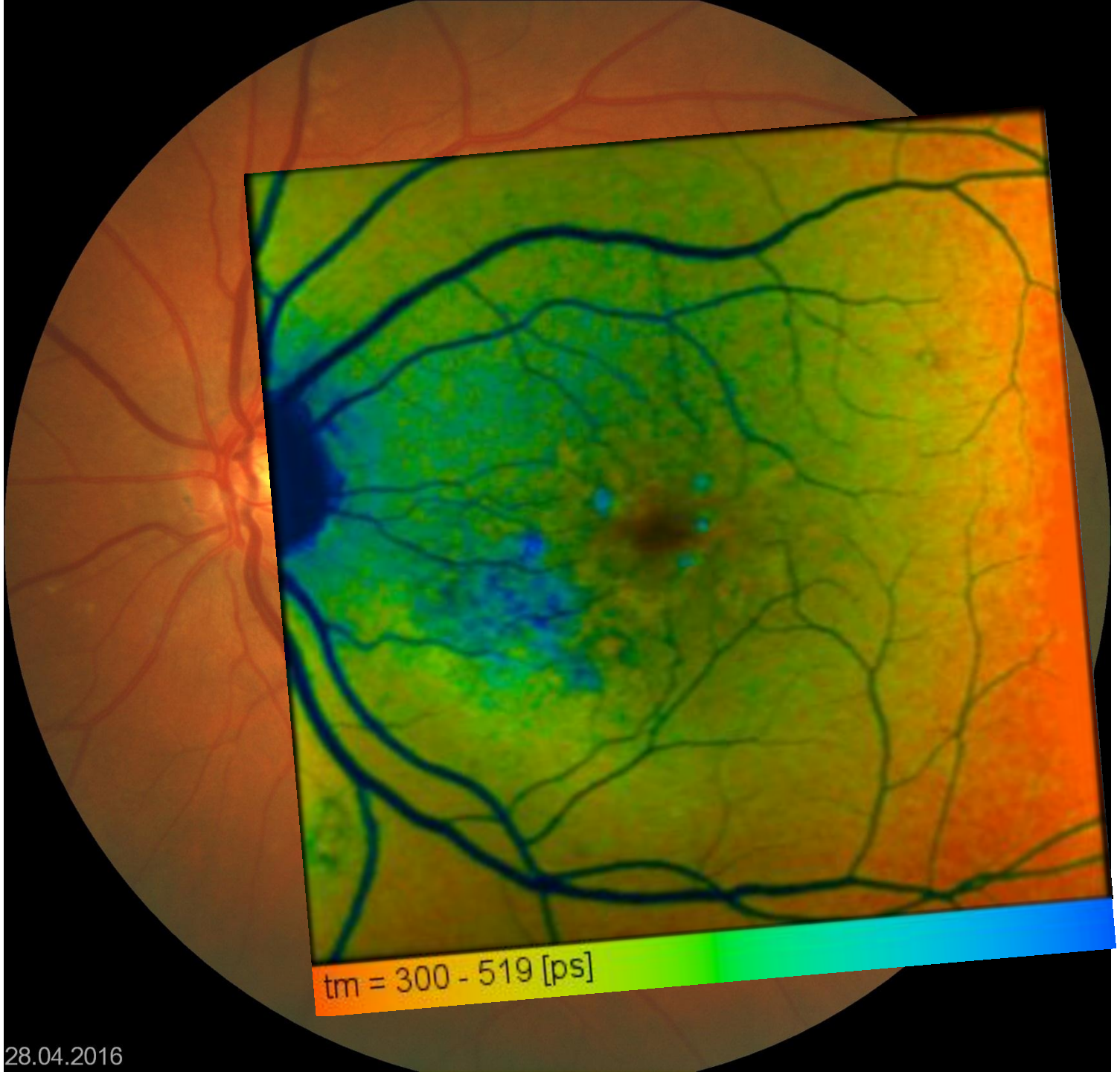
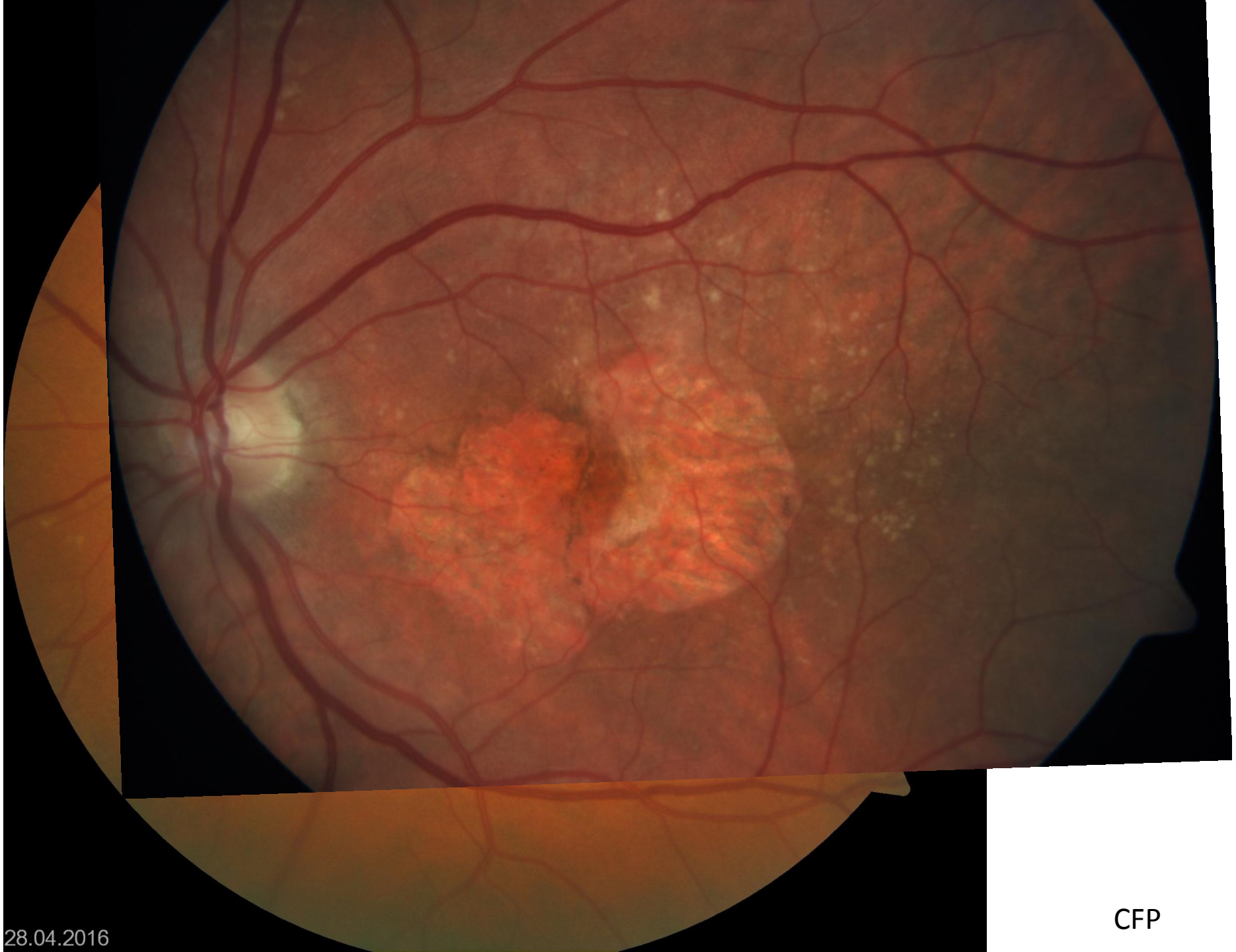
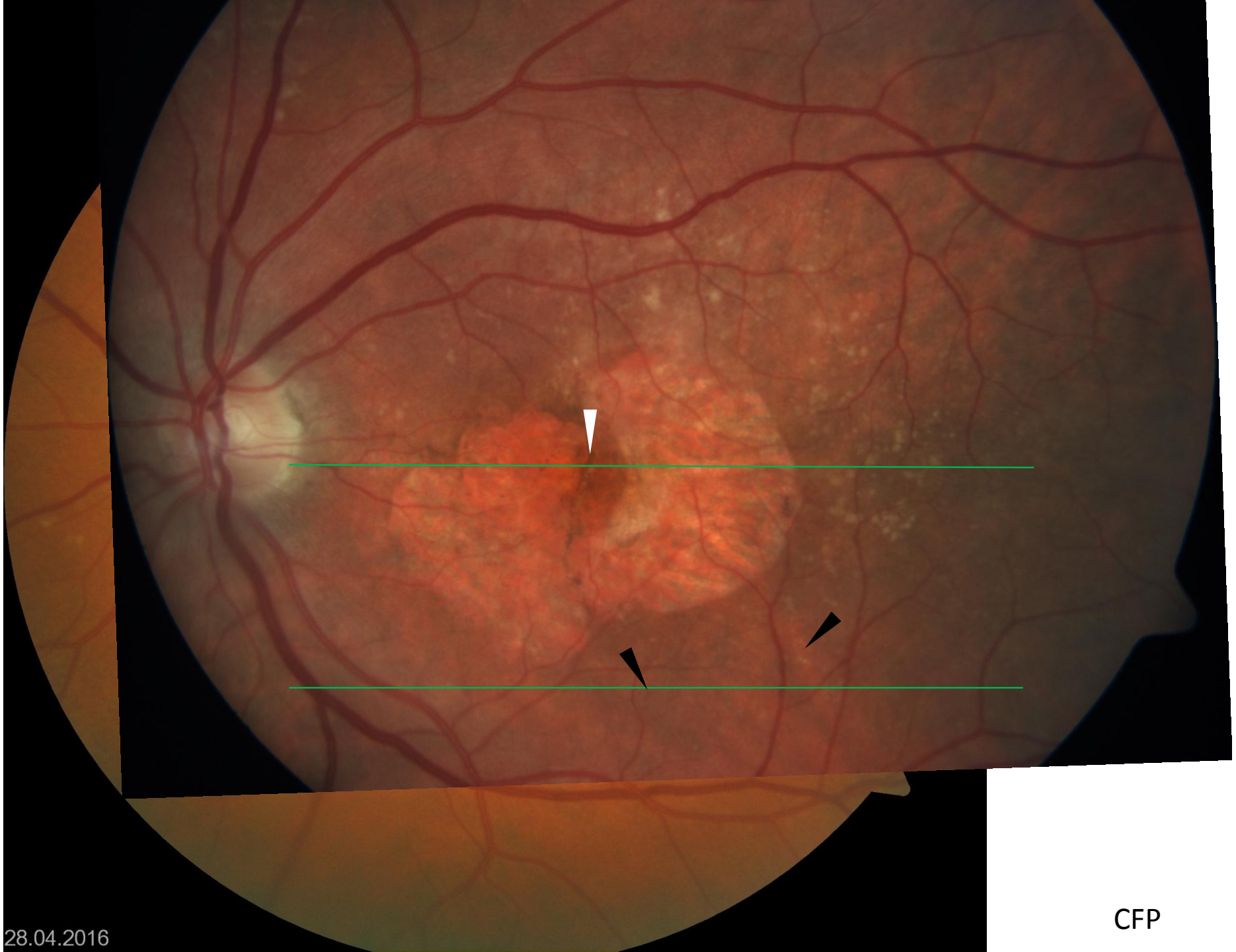


09/09/2020

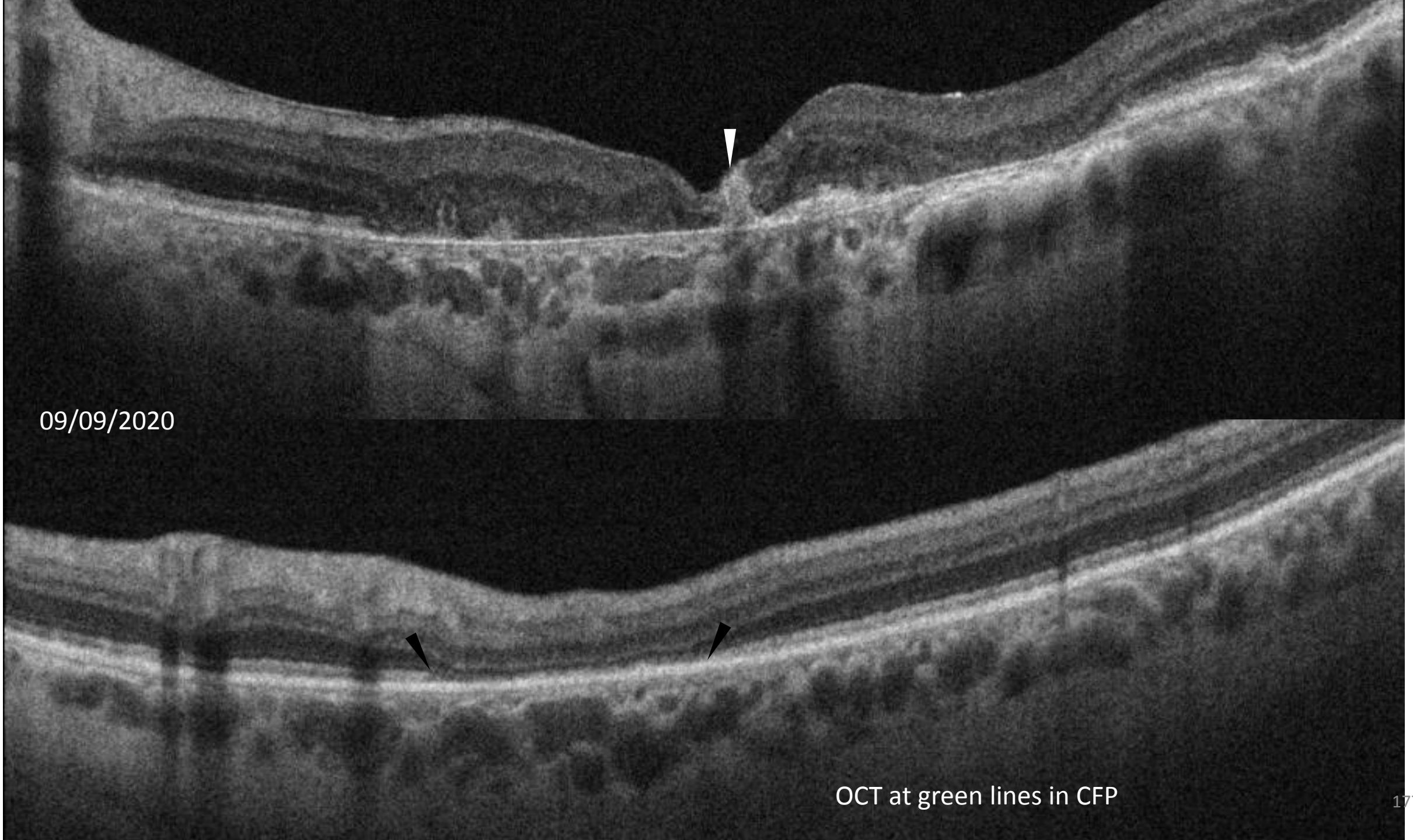


CFP

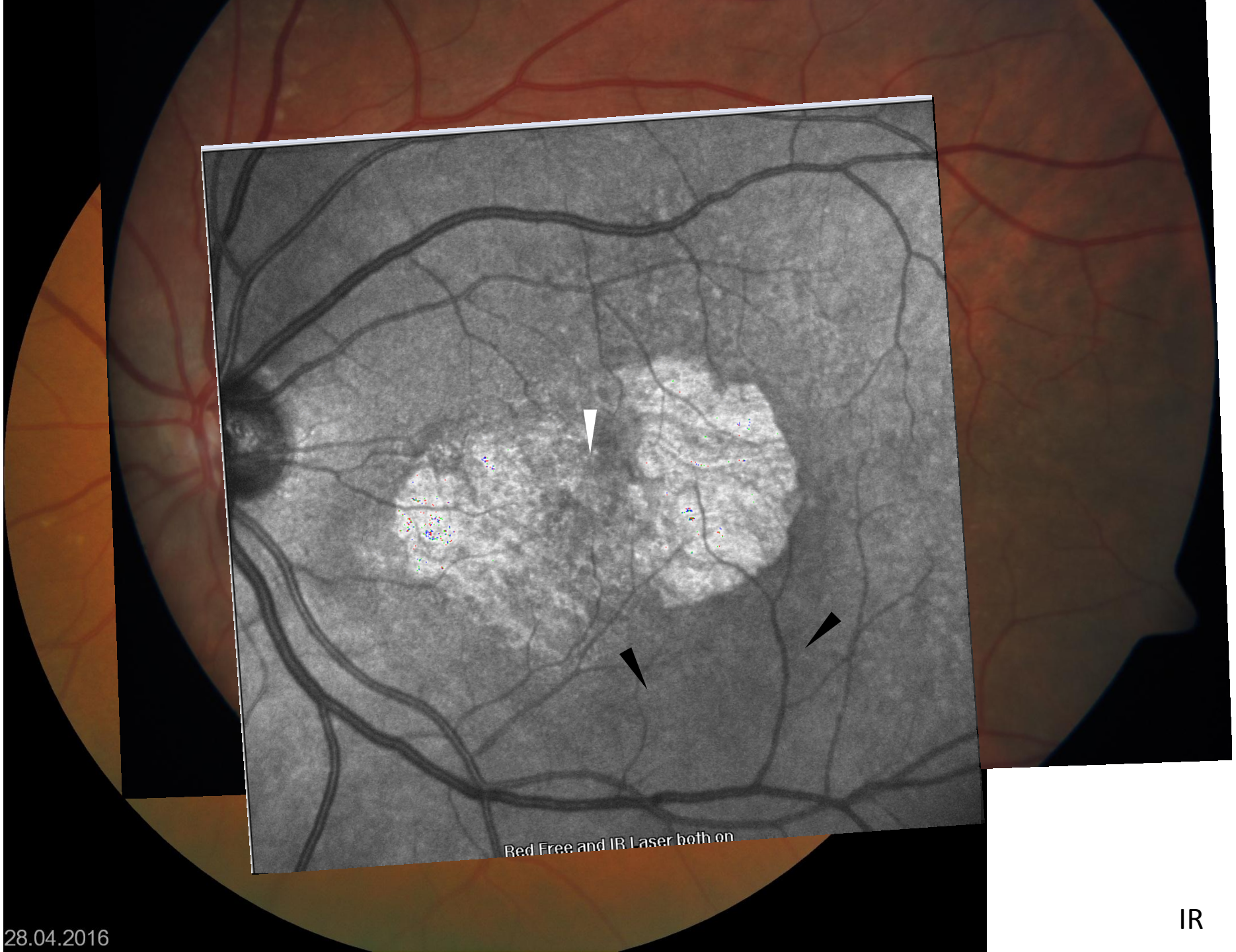
09/09/2020



CFP


09/09/2020
OCT at green lines in CFP

09/09/2020



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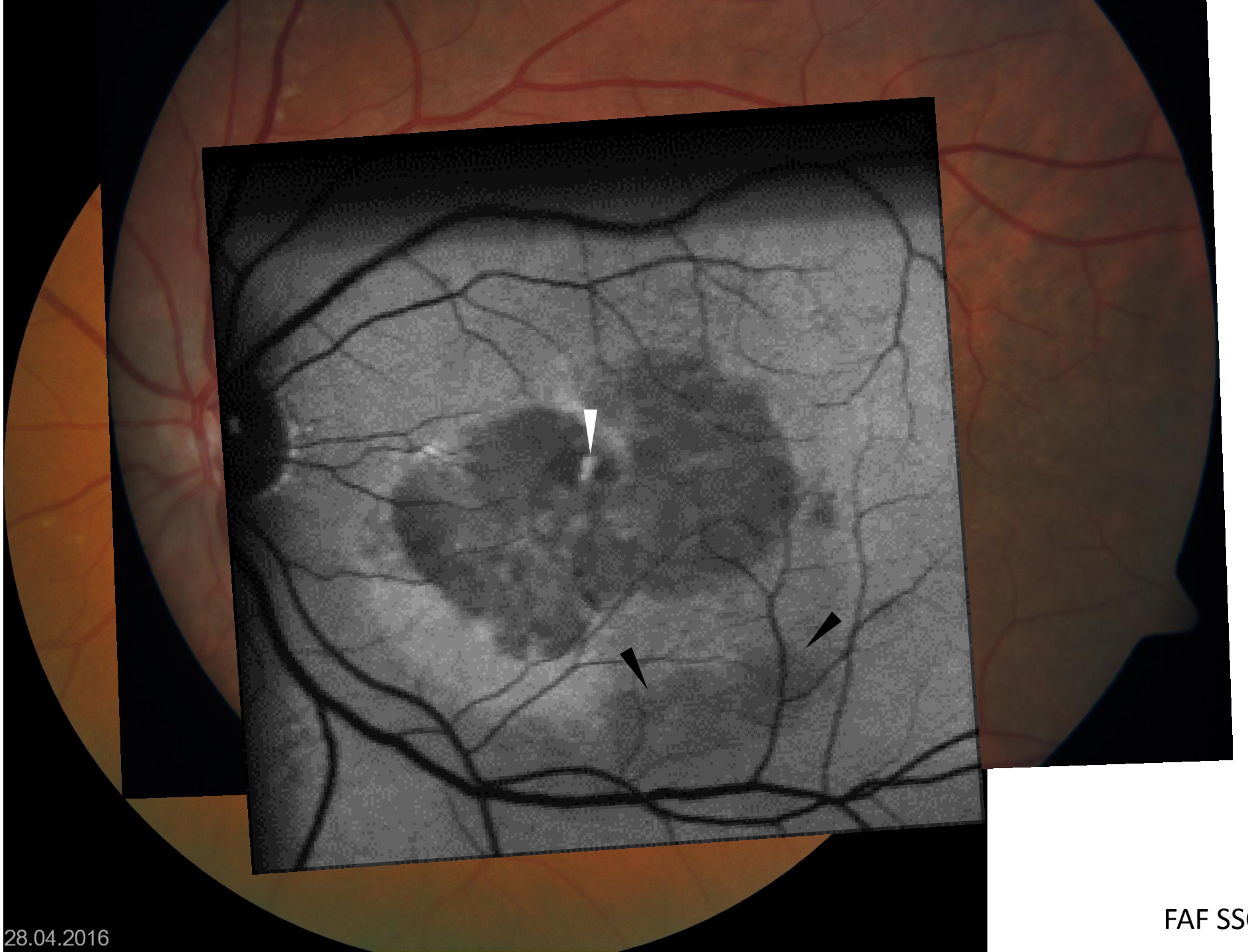
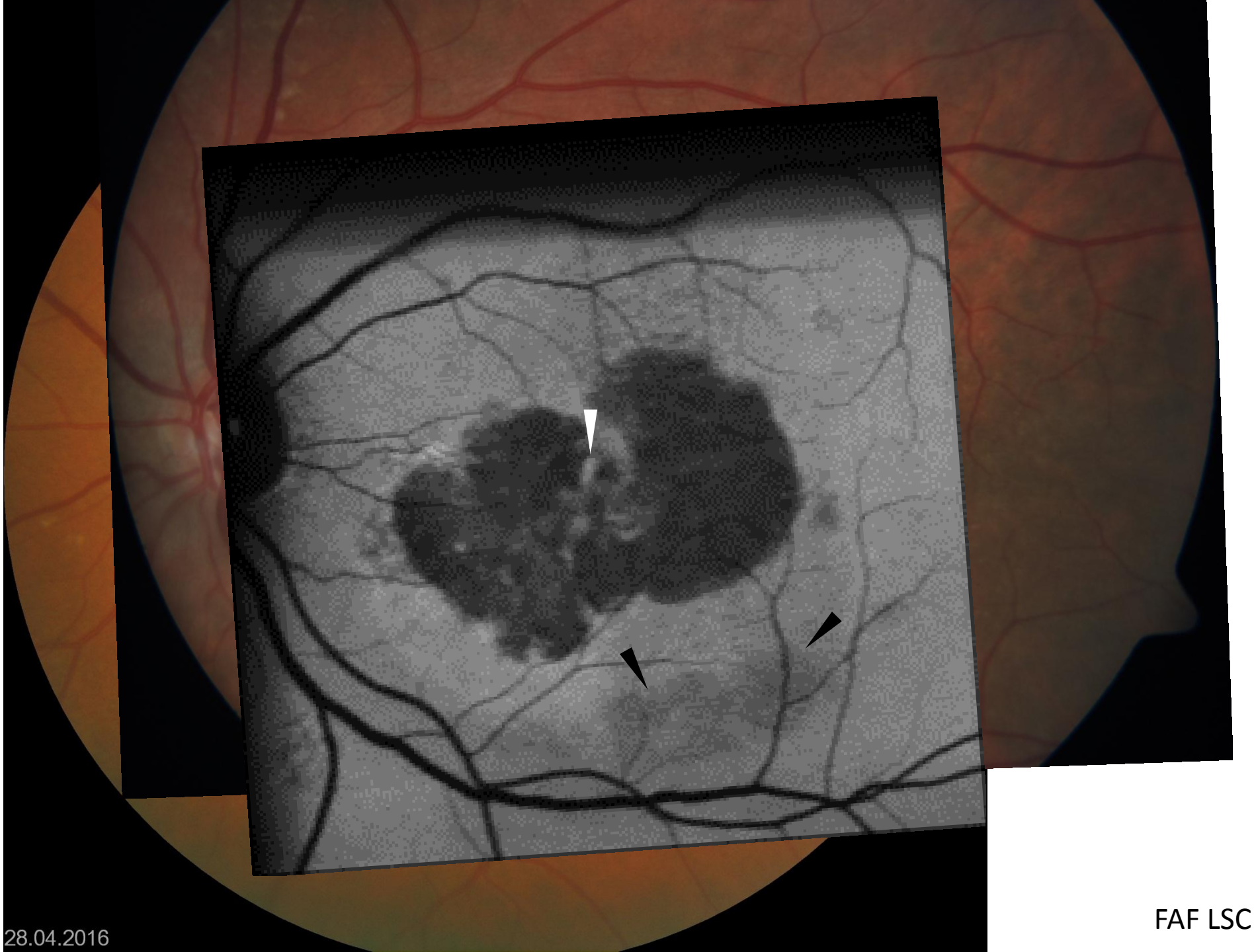
09/09/2020



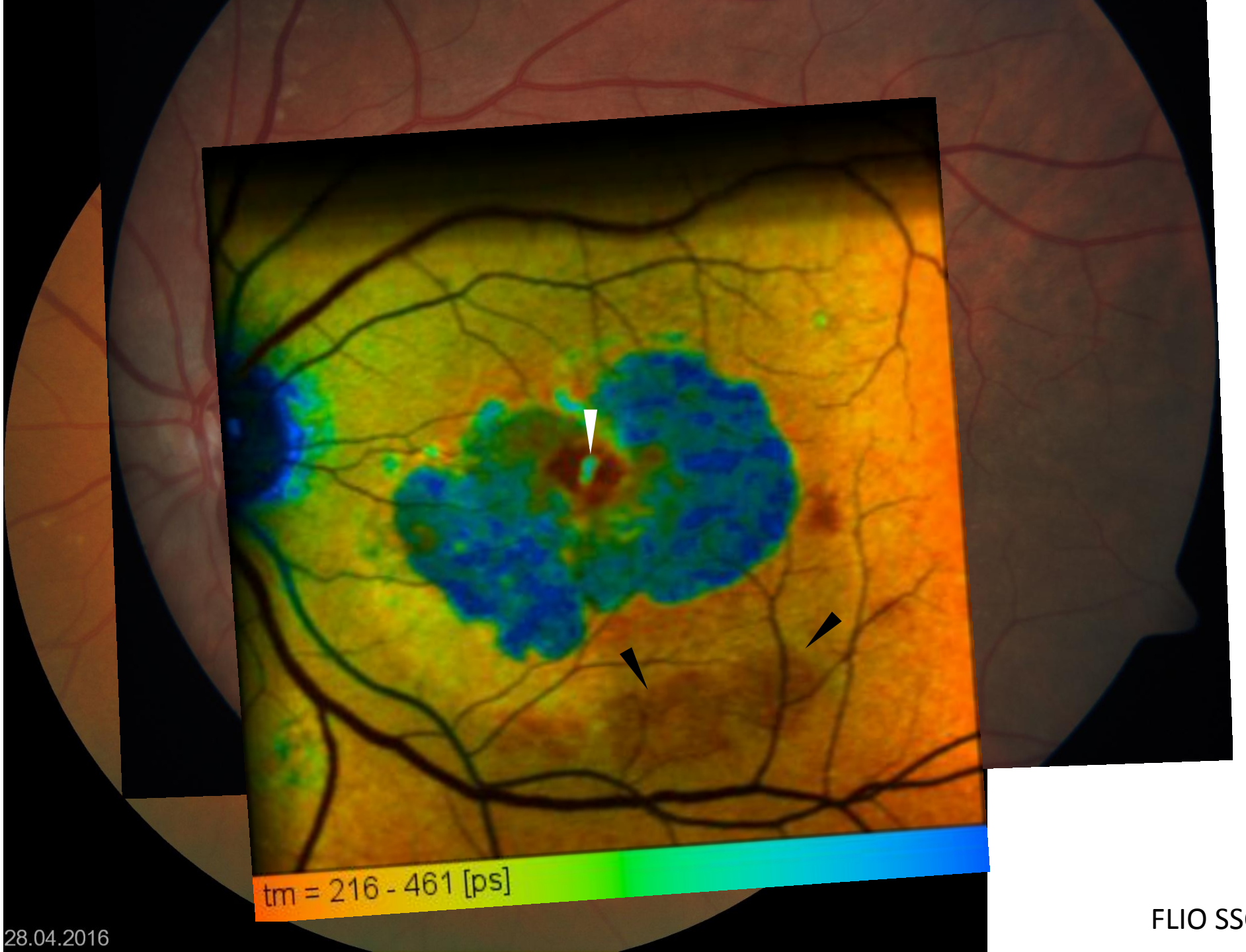
FAF SSC

09/09/2020



FAF LSC

09/09/2020



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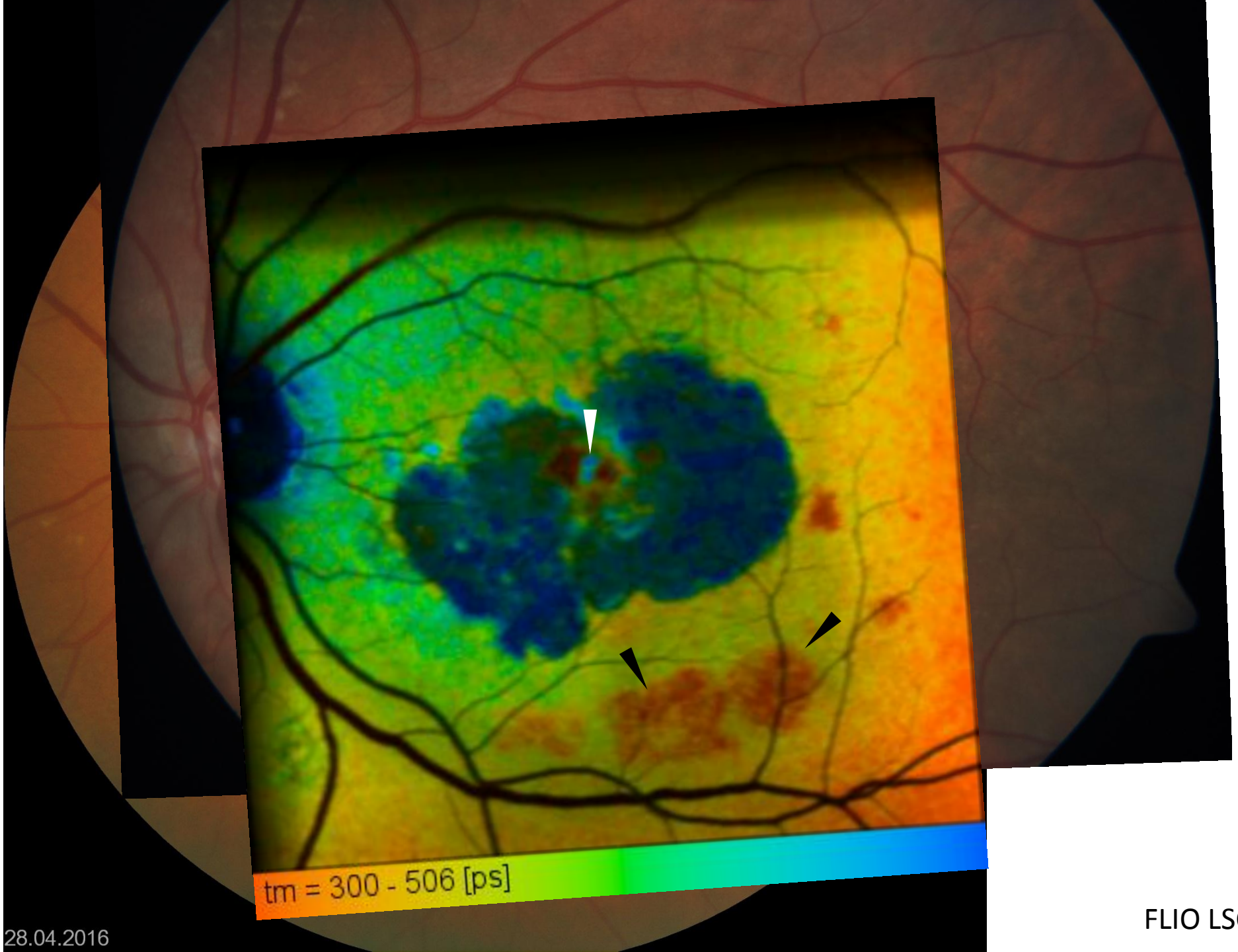
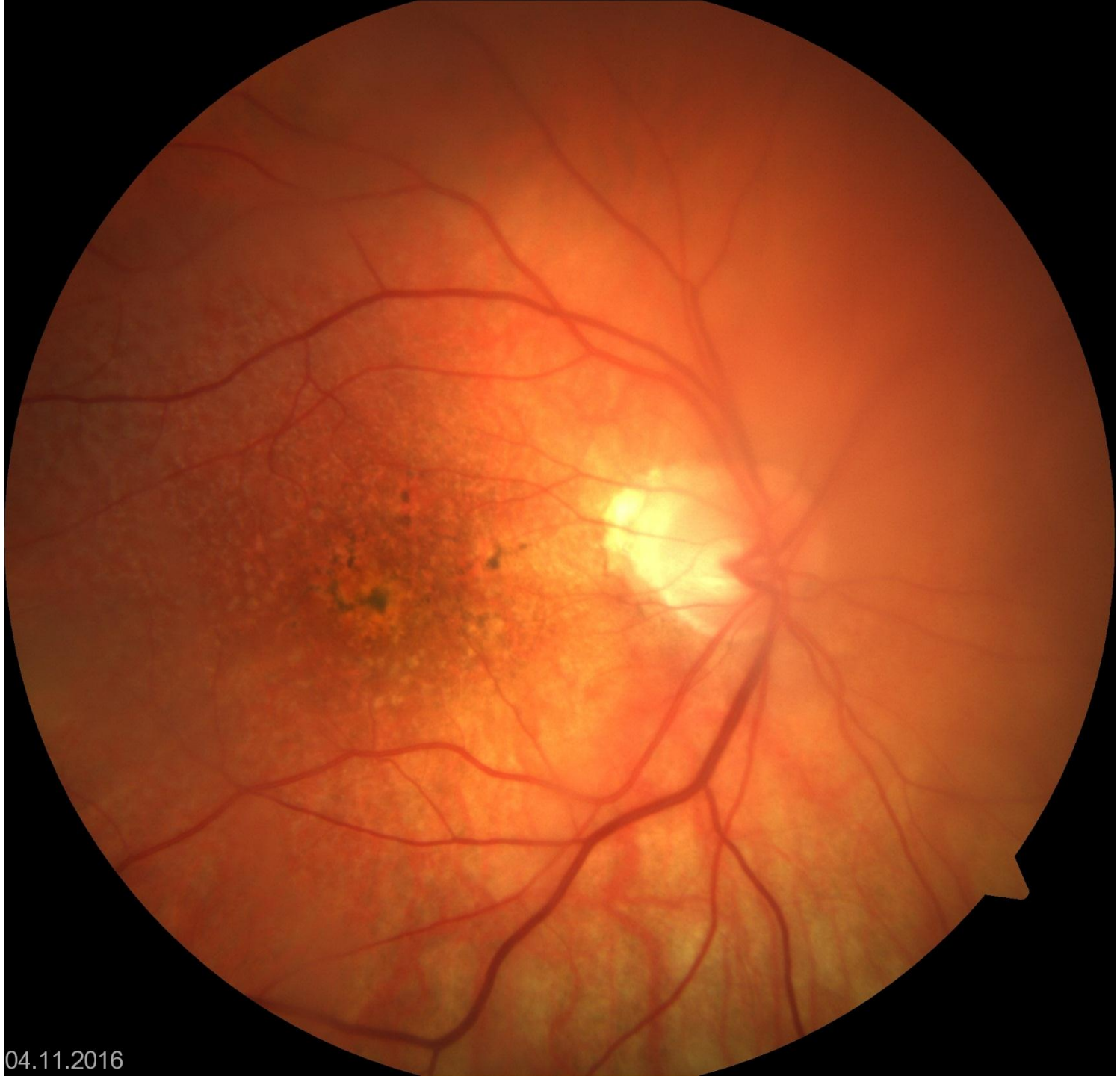


AMD_68, 74 years at baseline

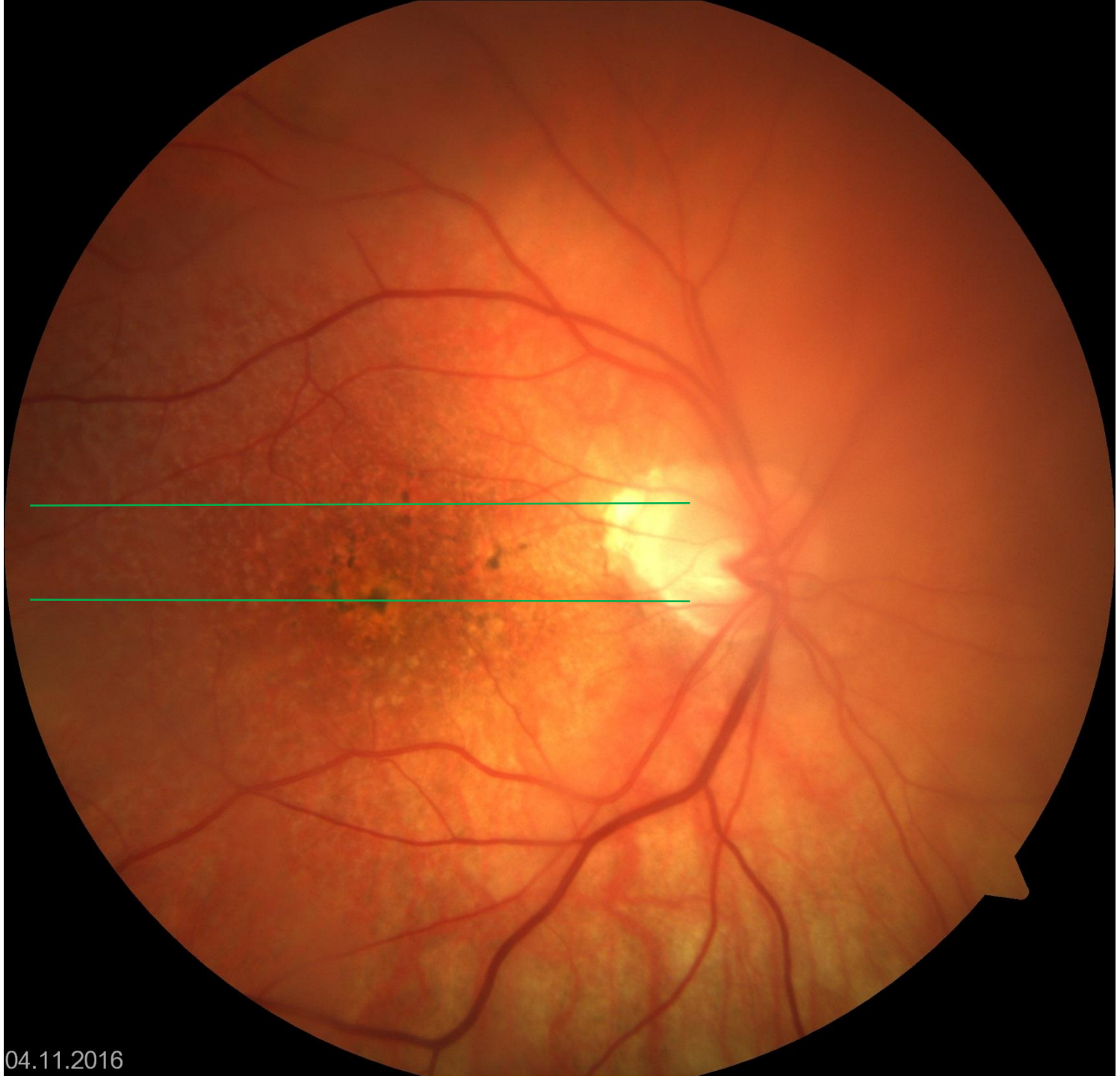
This Patient showed up with massive hyperpignetations and developed a GA in the area of these hyperpigmentations 4 years later (black arrowhead). However, it has some peculiarities: 1. the hyperpigmentations seem to be associated with SDD rather than soft drusen. 2. the GA is clearly seen in OCT and FLIO, but shows rather normal fluorescence at long wavelength (LSC) and is hyperfluorescent at short wavelength (LSC). **What might cause this hyperfluorescence?**

11/04/2016

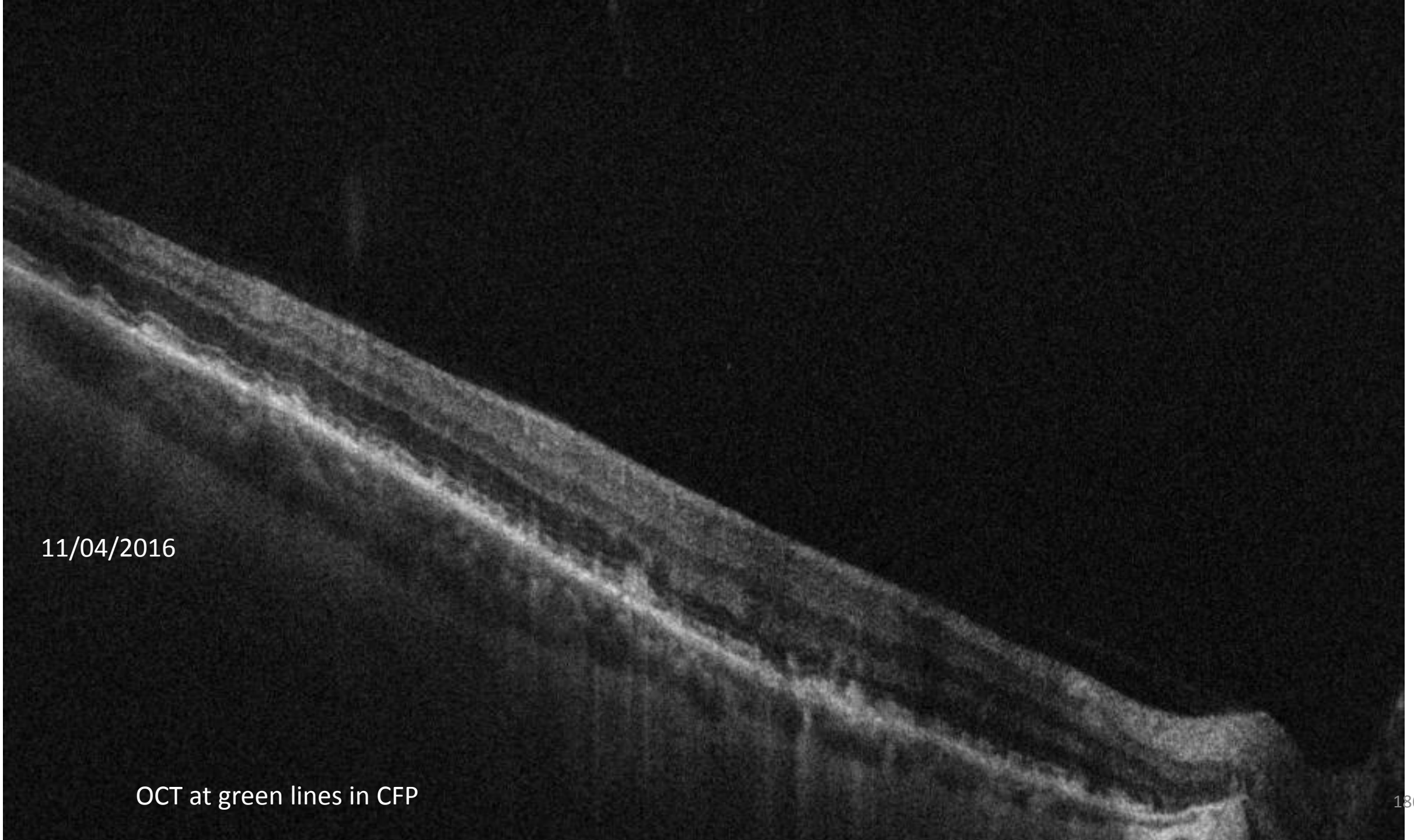


CFP

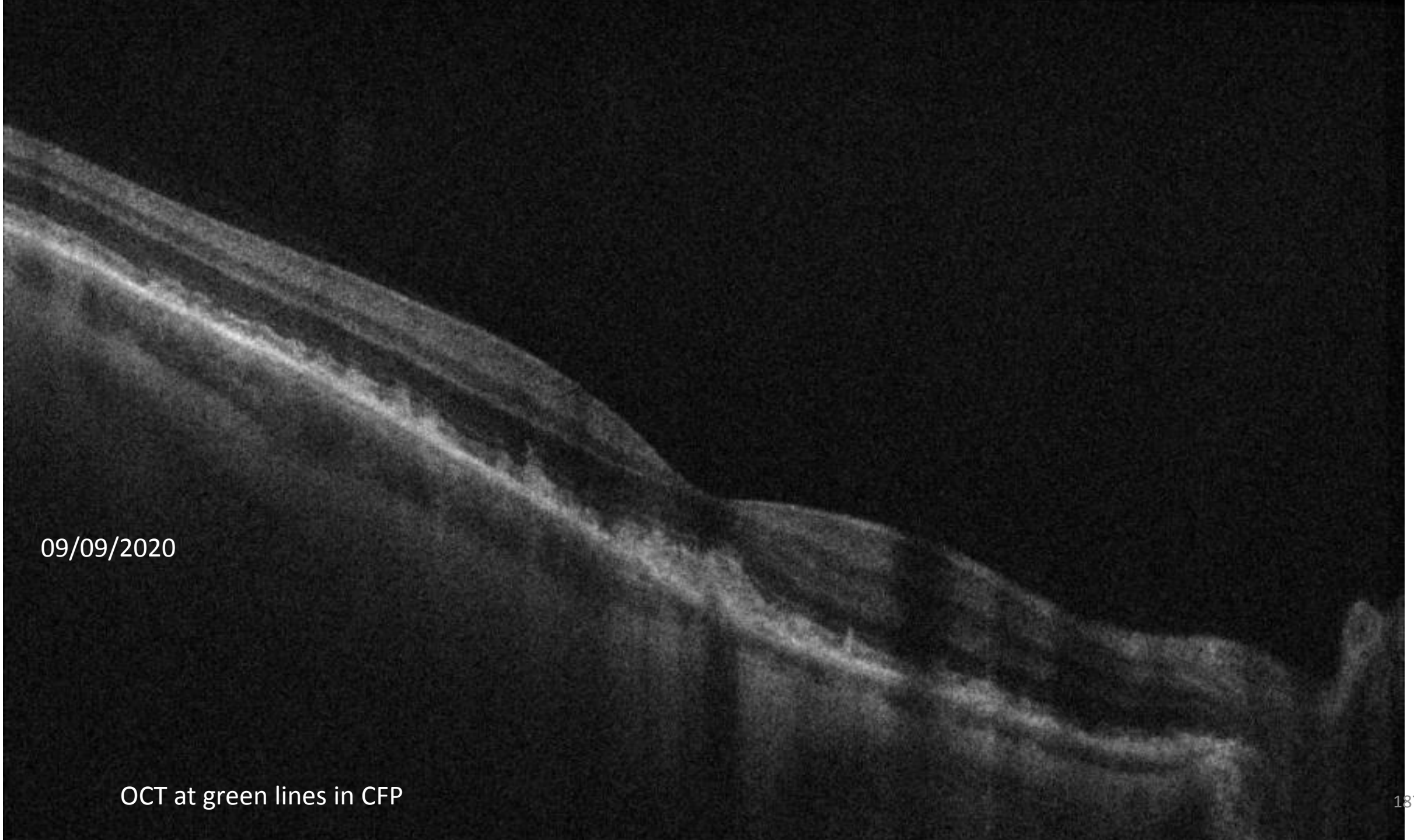
11/04/2016



CFP

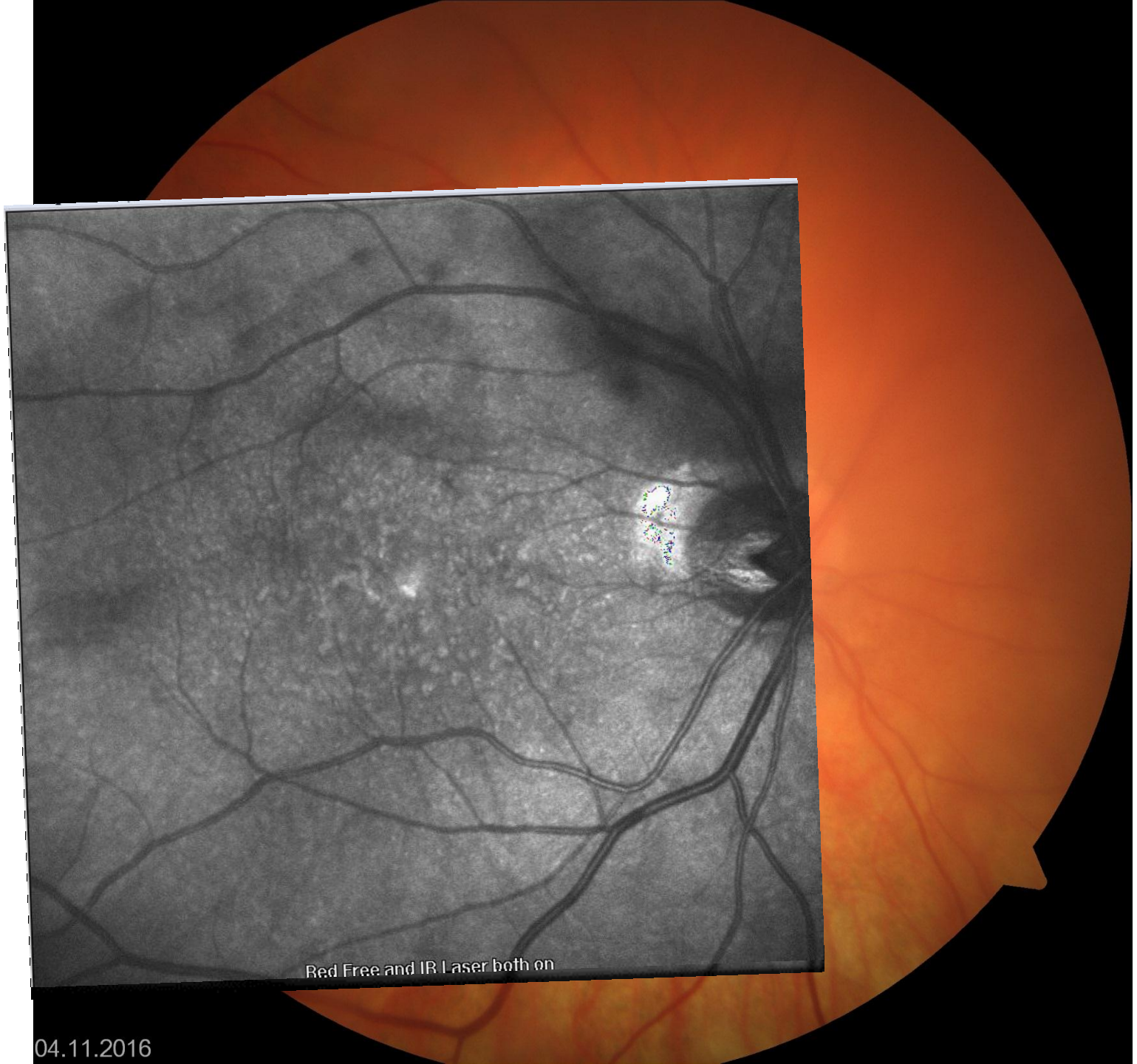


OCT at green lines in CFP



OCT at green lines in CFP

11/04/2016



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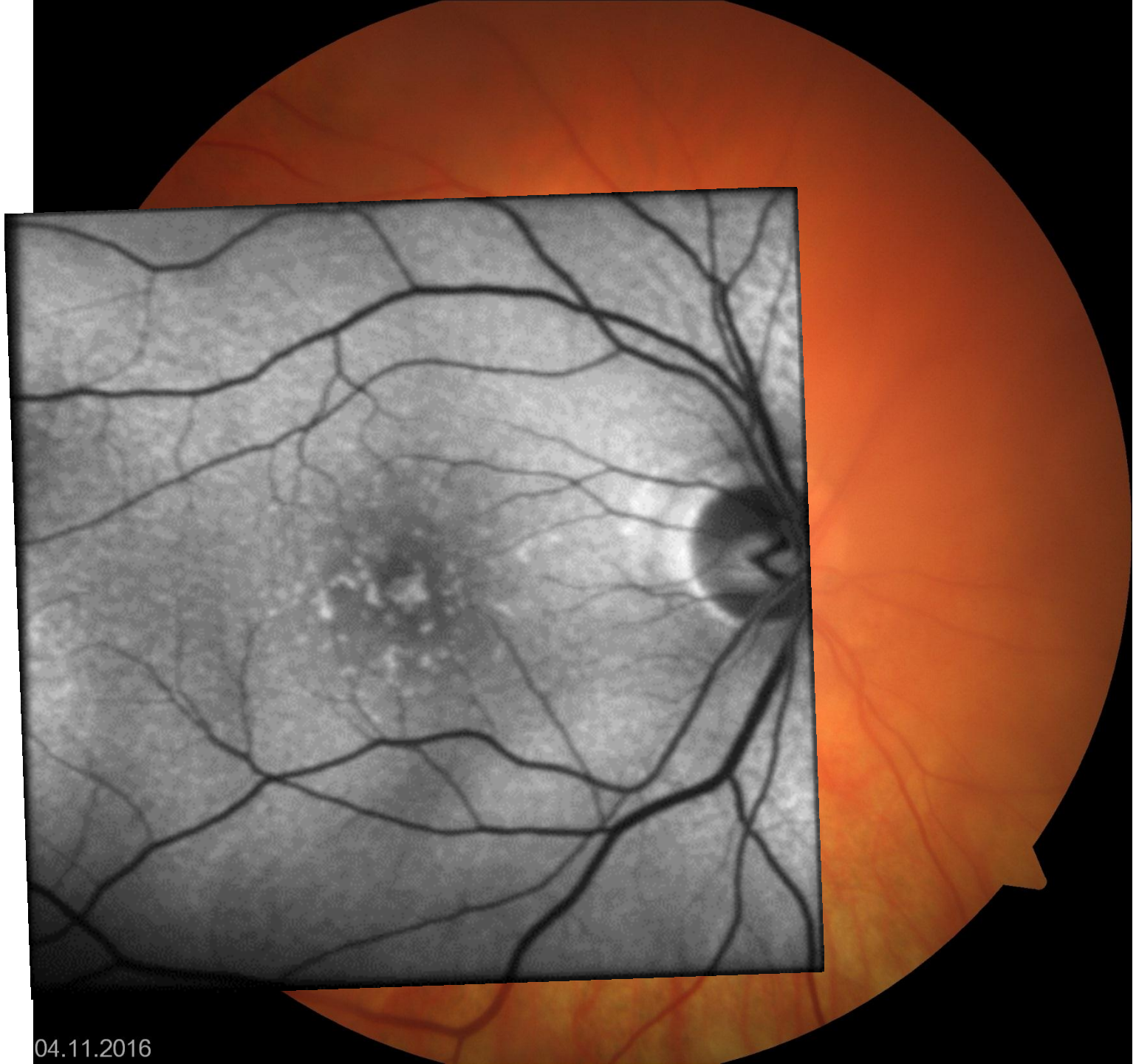
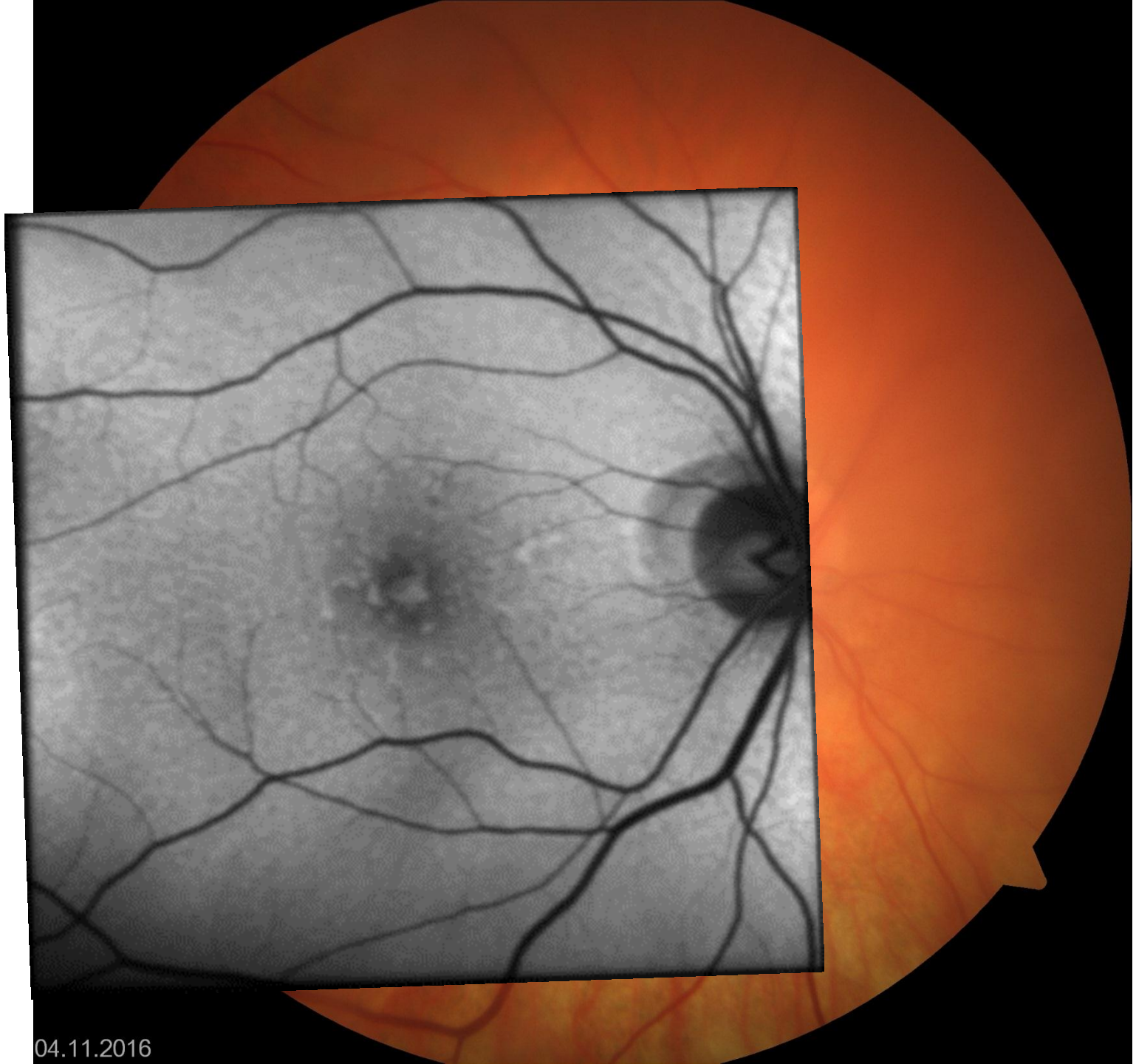
11/04/2016



FAF SSC

11/04/2016



FAF LSC

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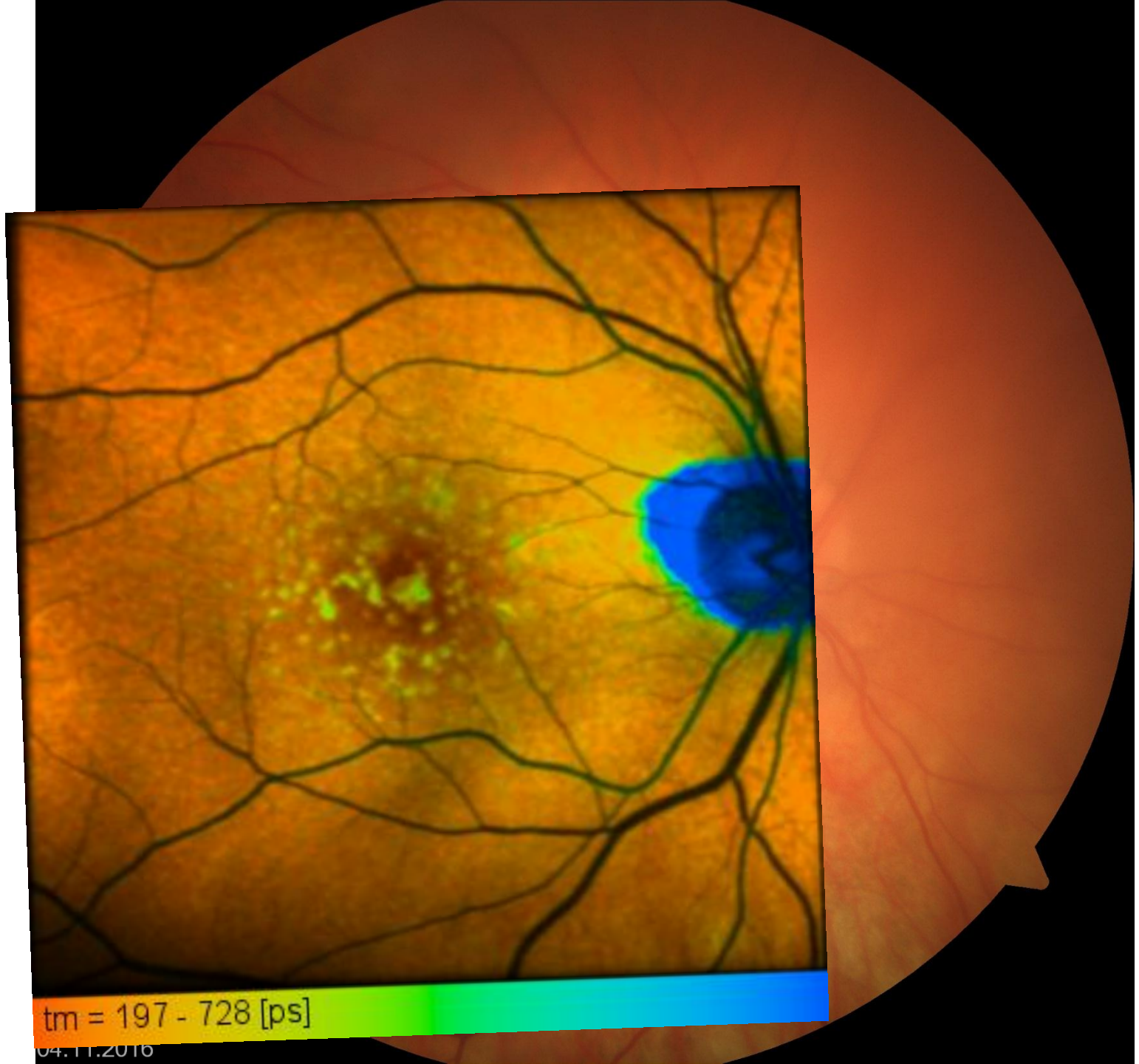

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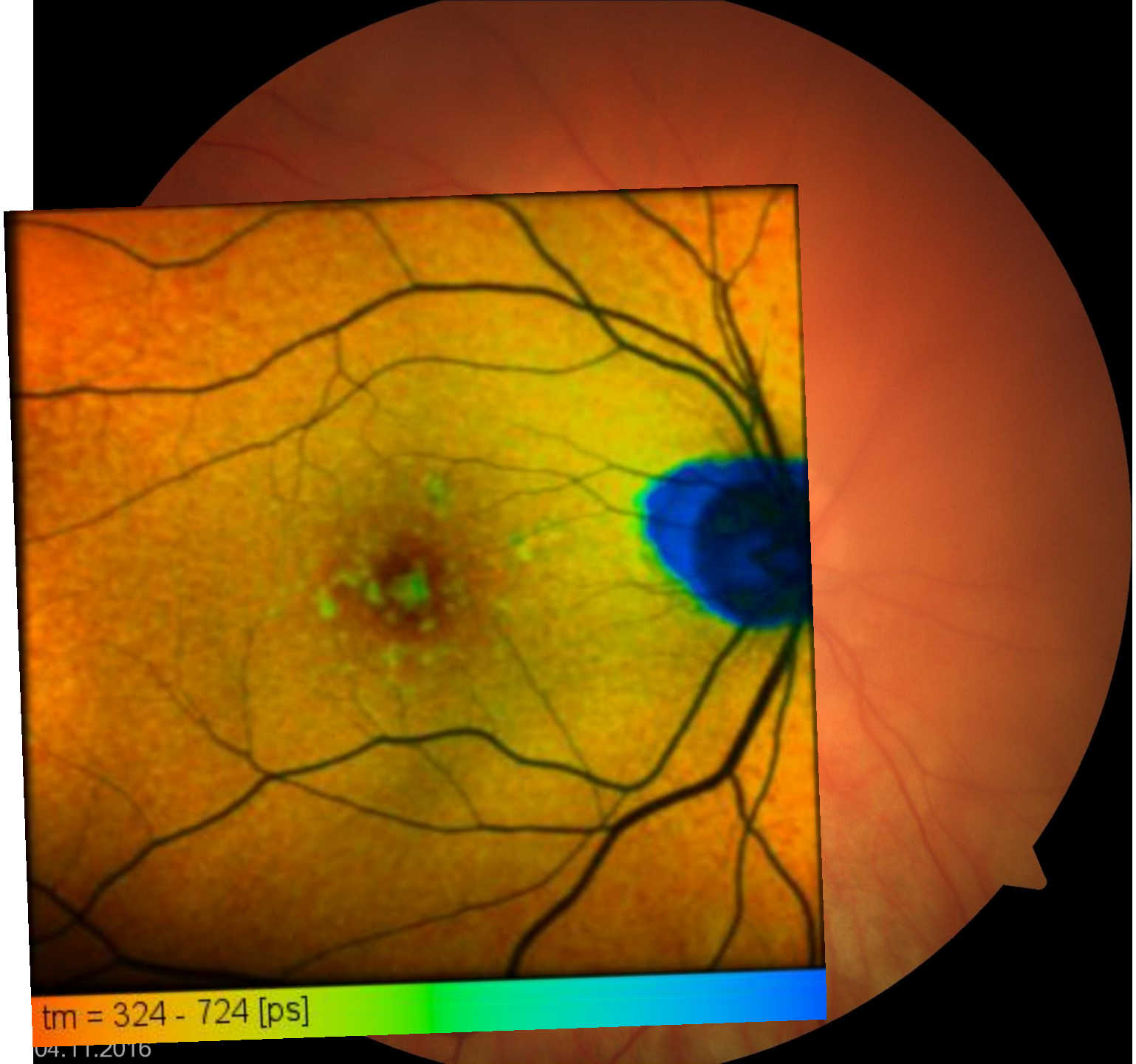
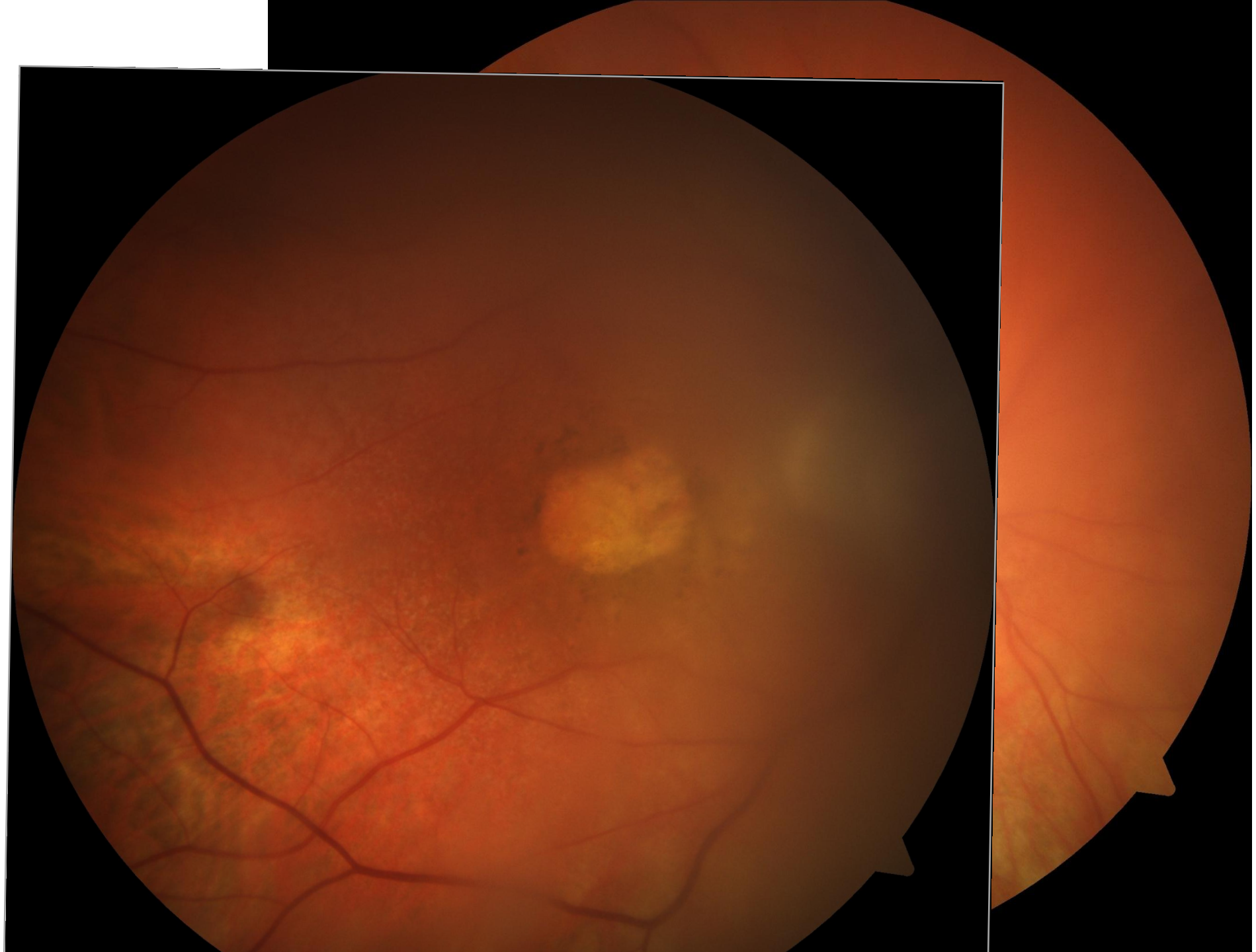
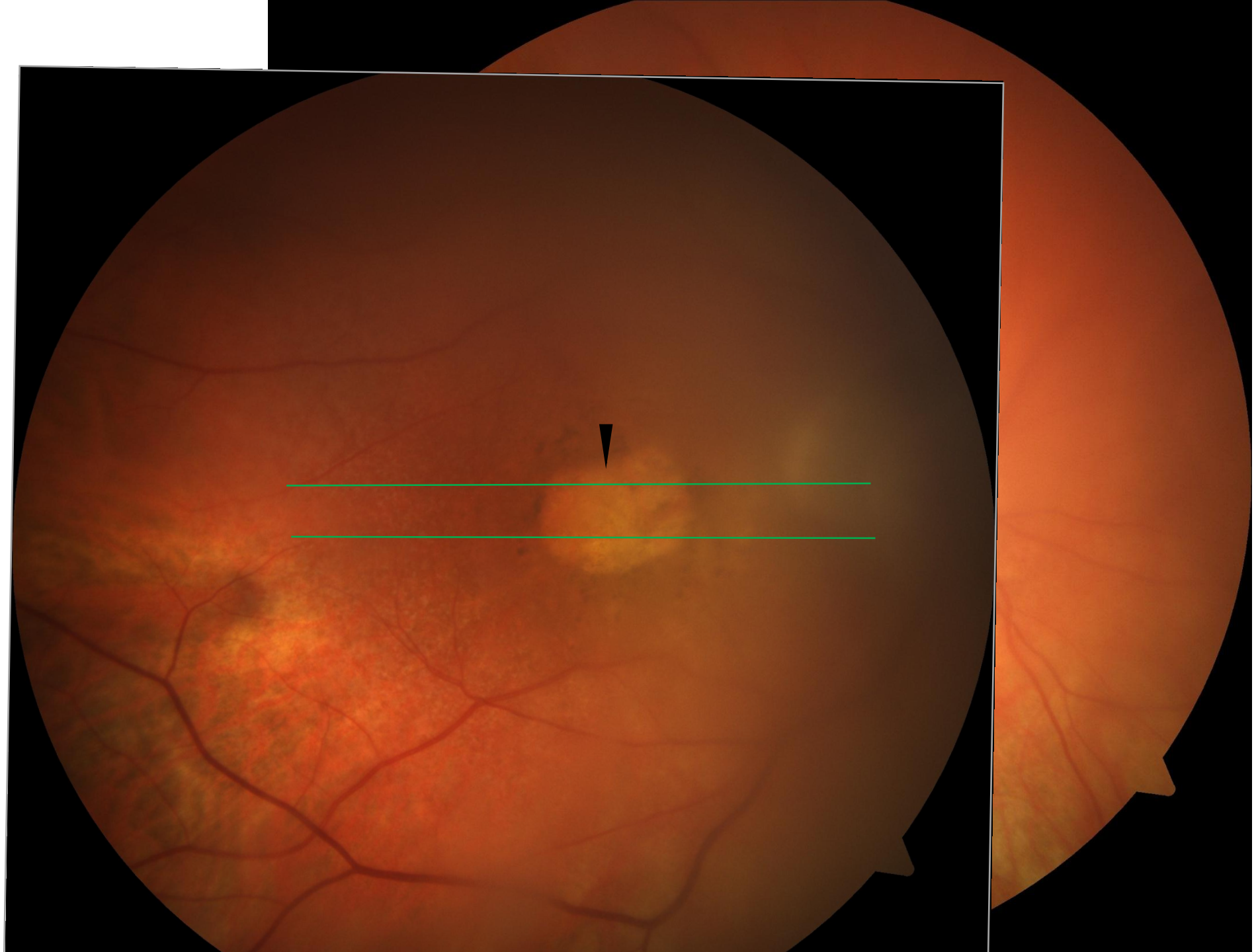




10/12/2020

CFP



10/12/2020

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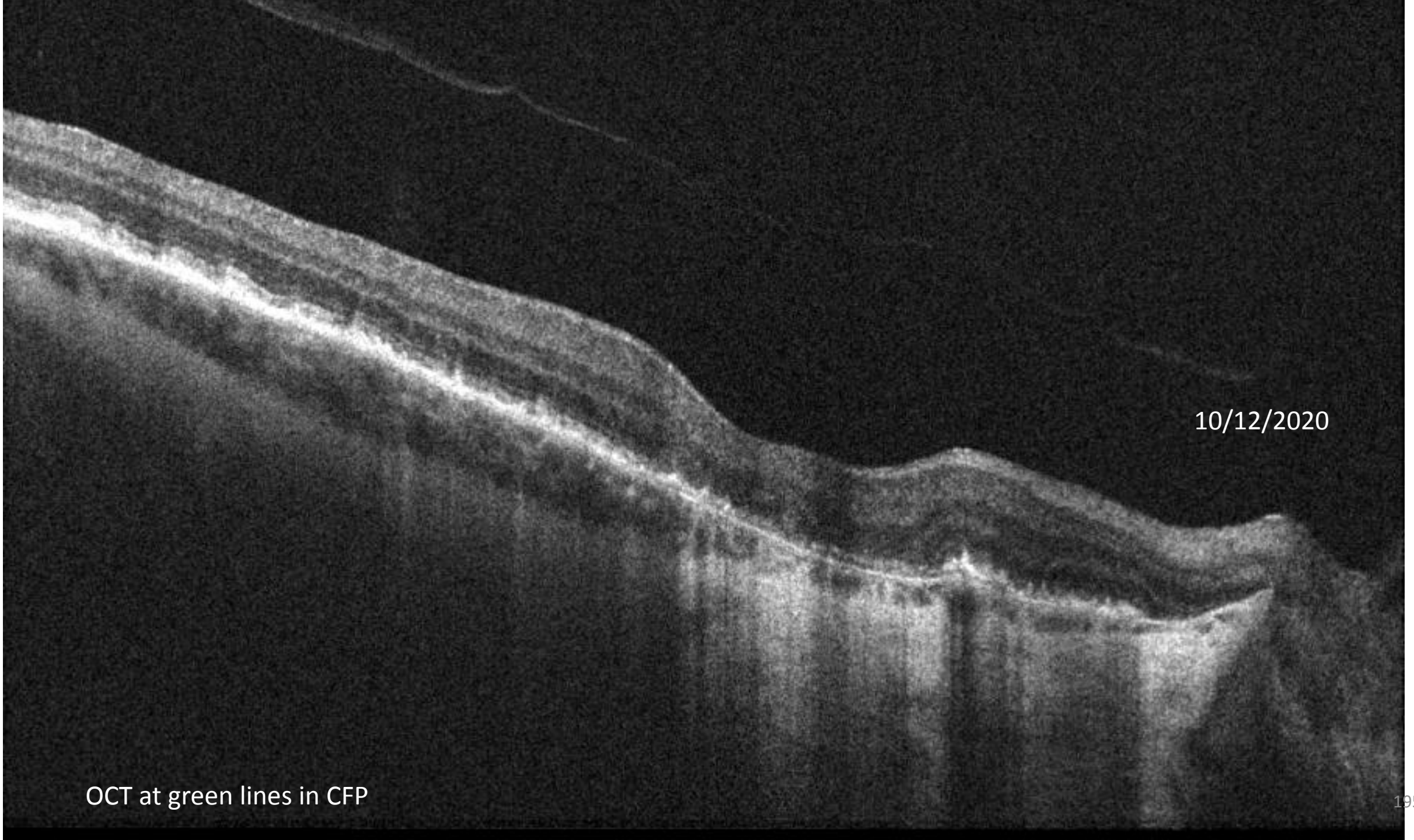
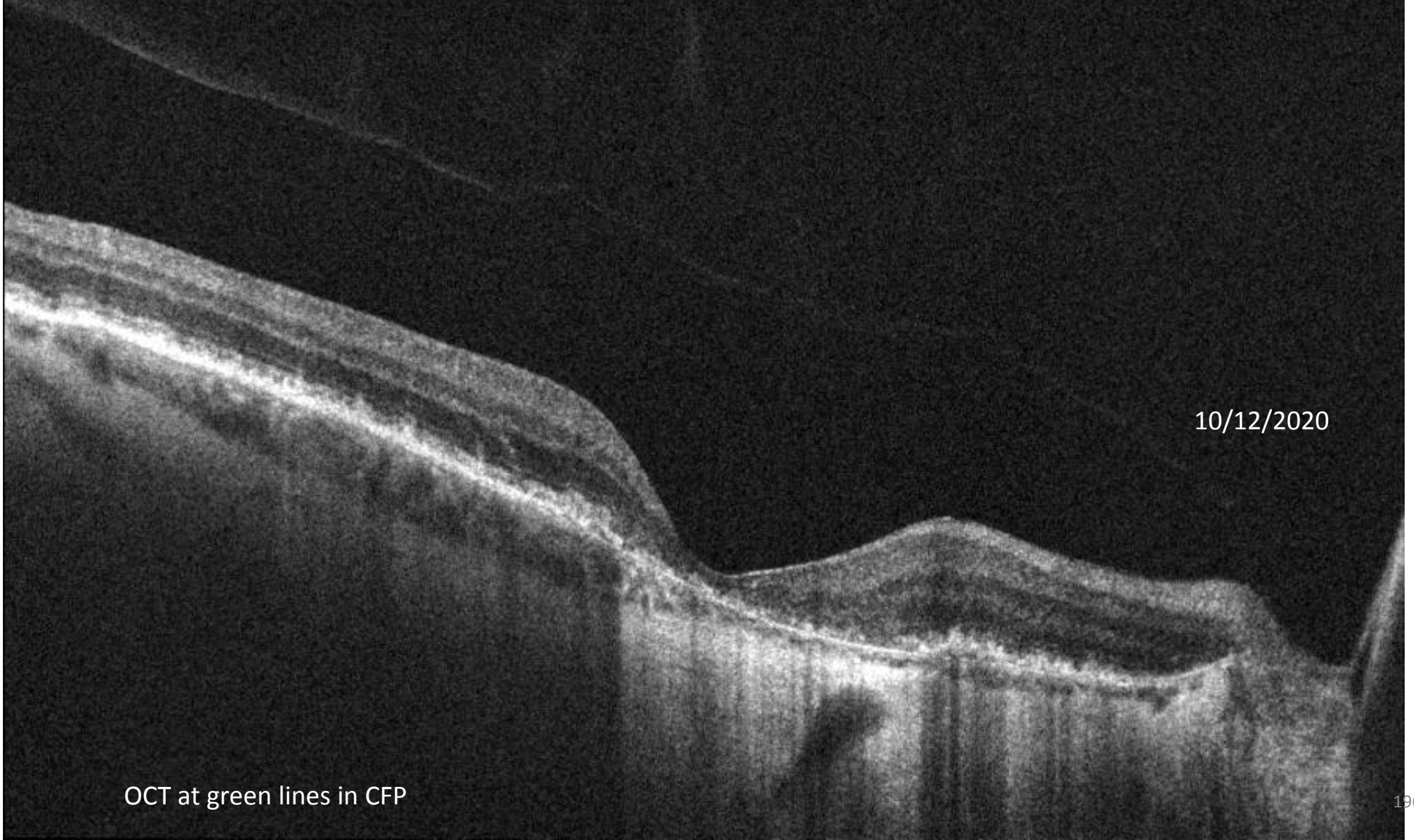
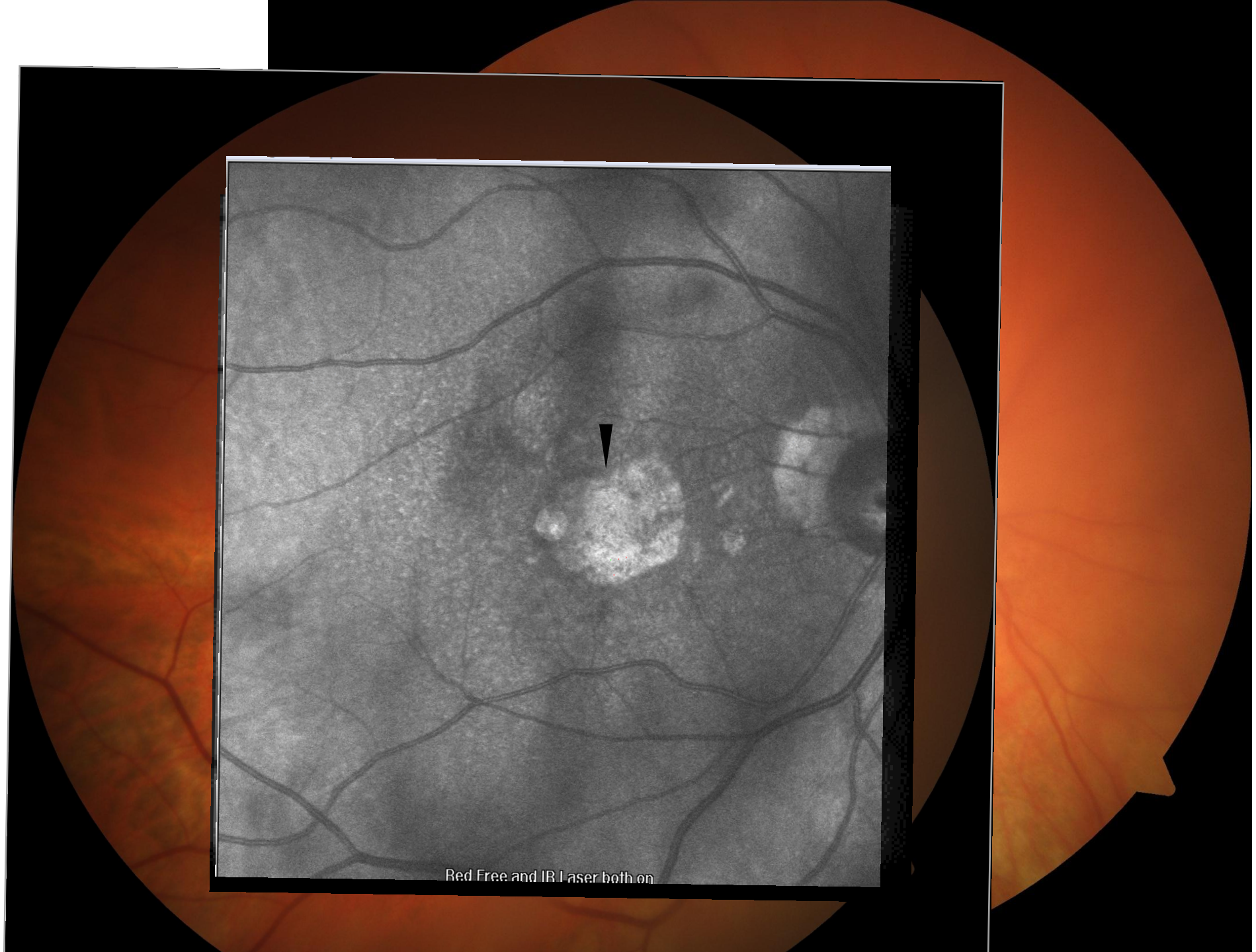


OCT at green lines in CFP

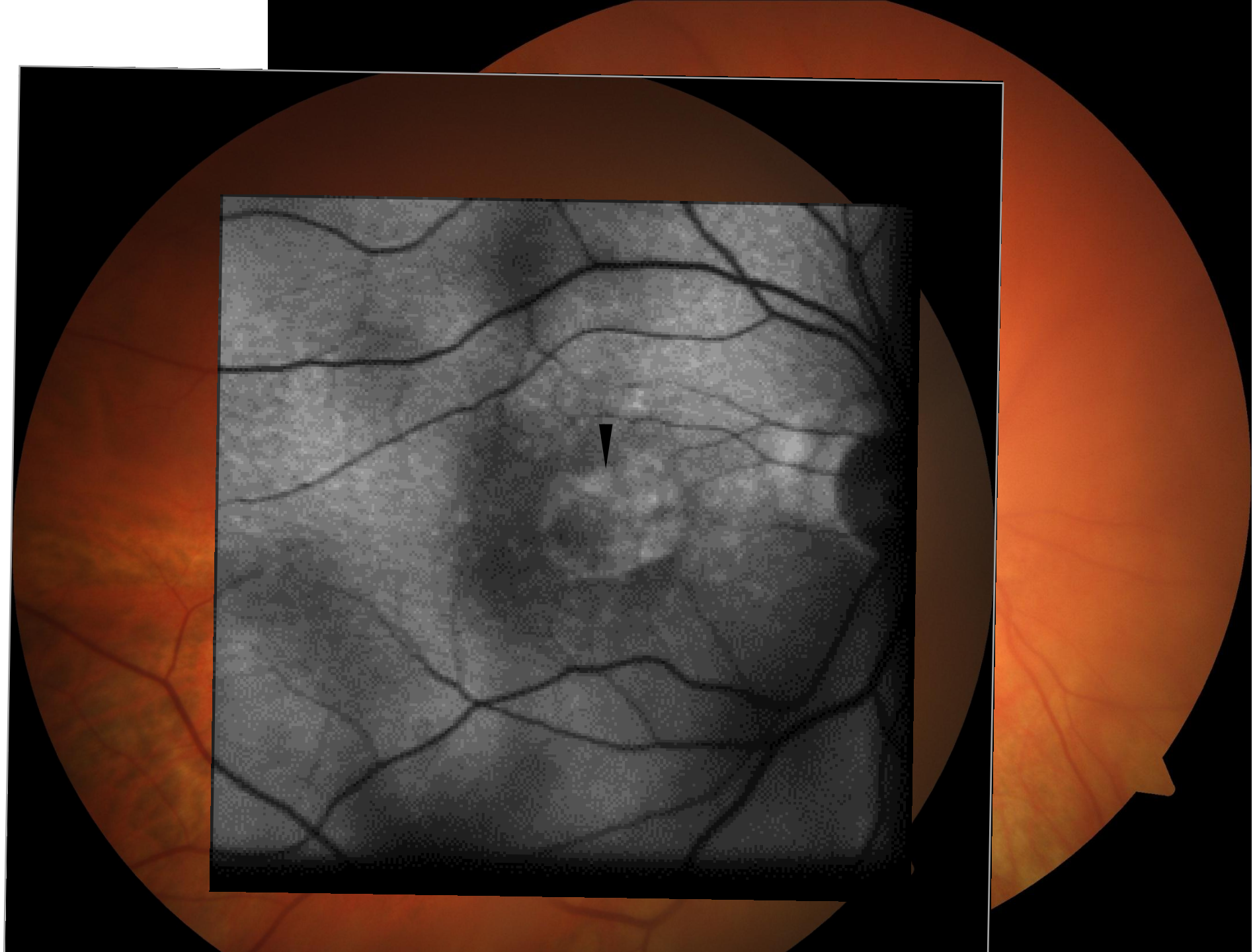


OCT at green lines in CFP

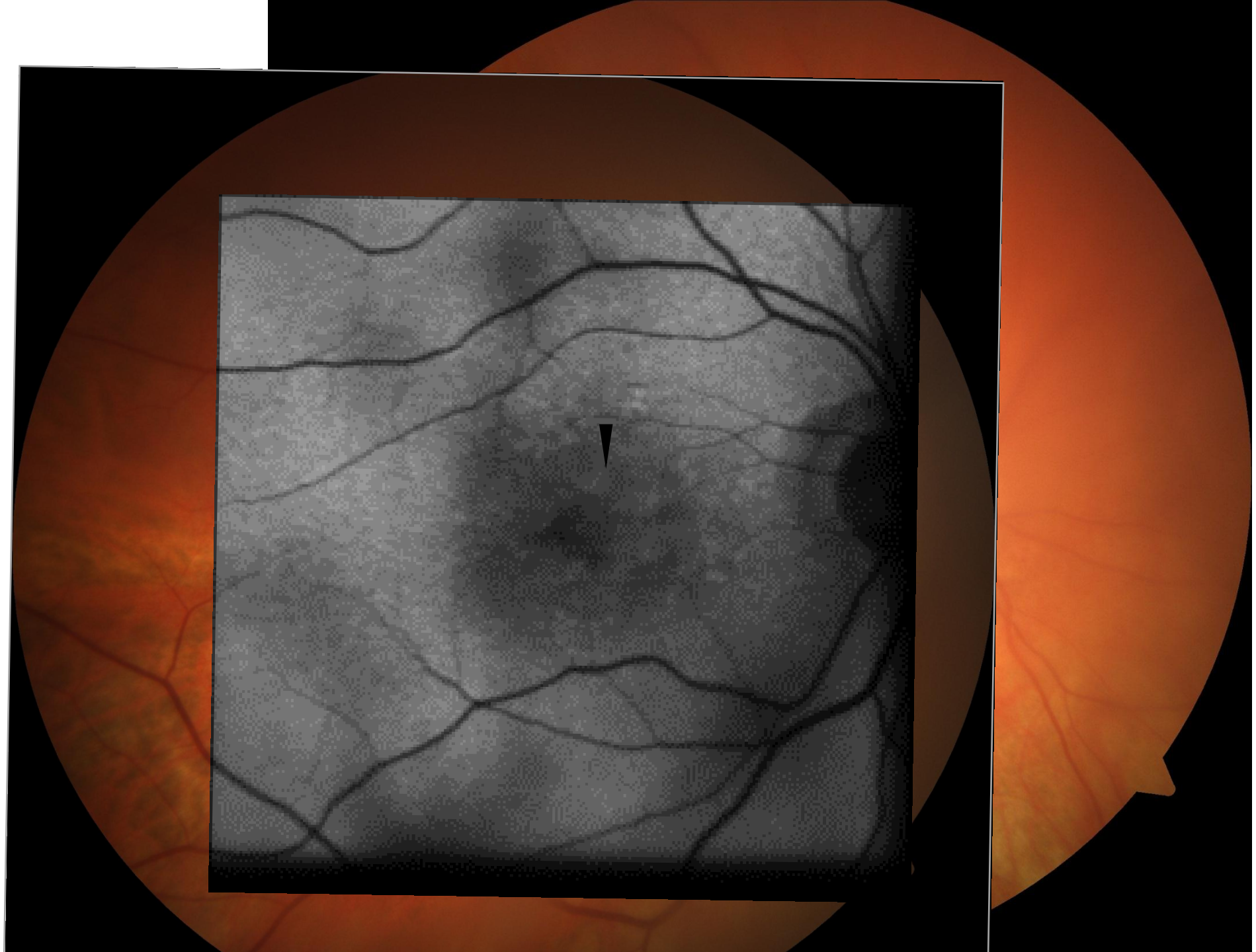


10/12/2020

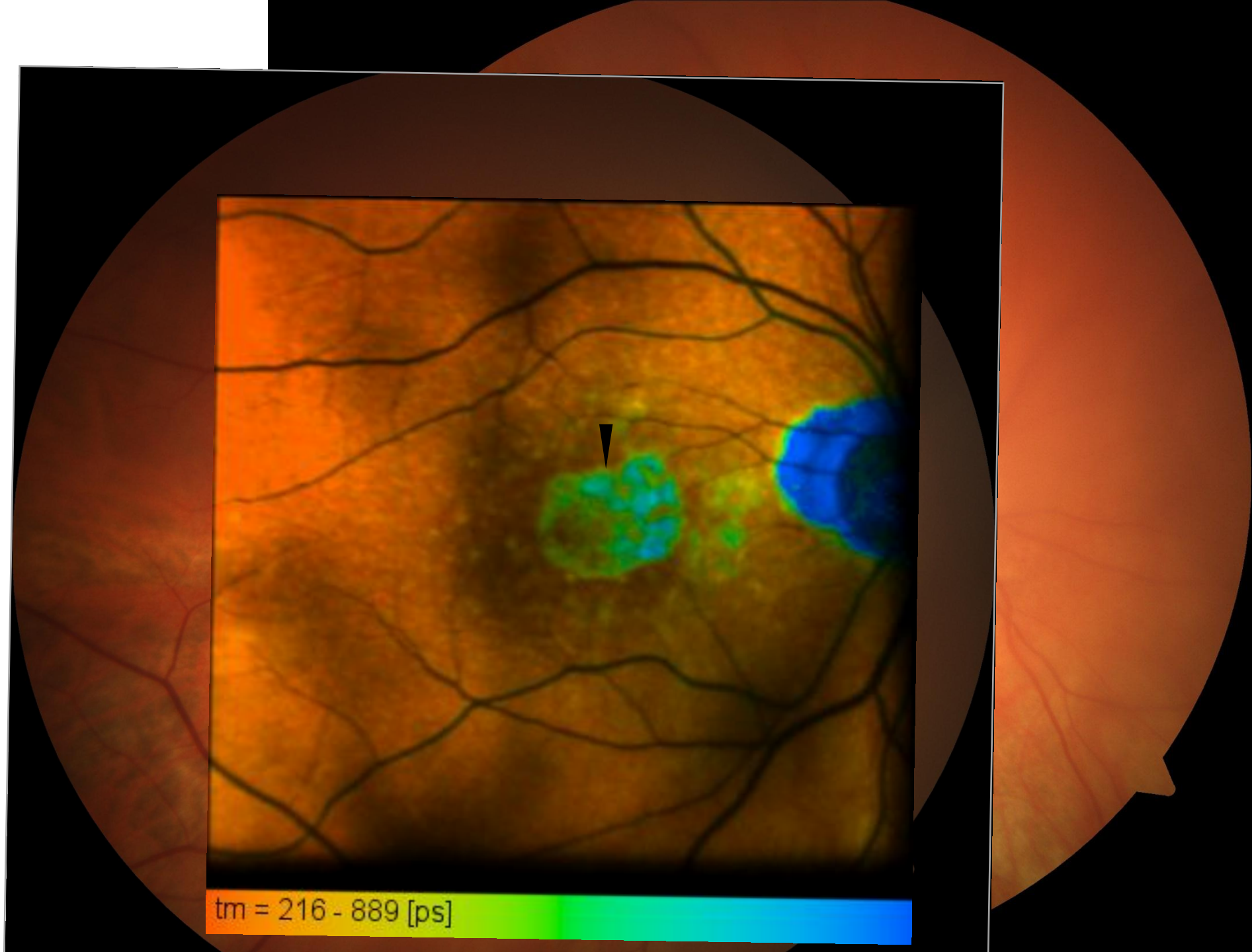
IR



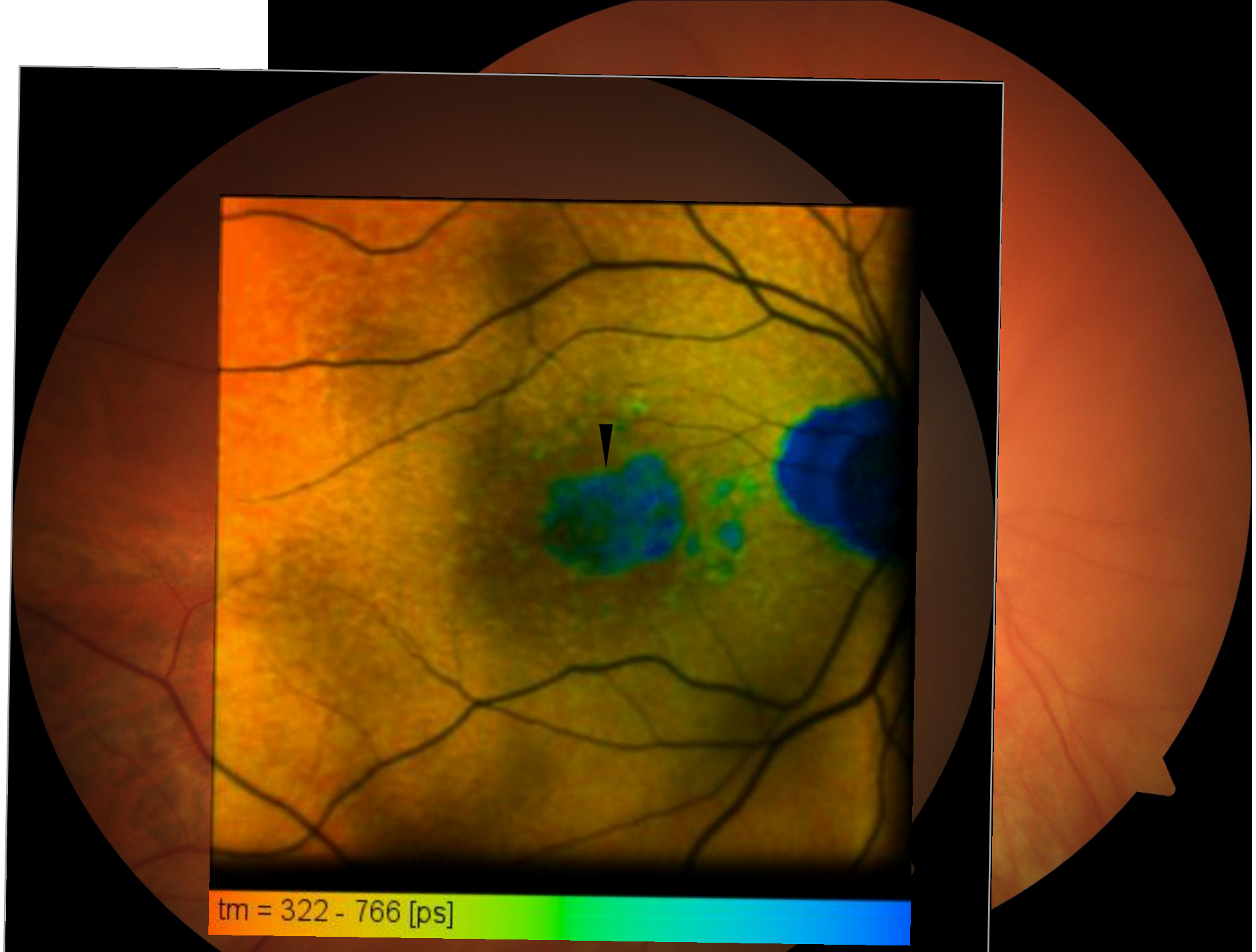
10/12/2020



10/12/2020



10/12/2020

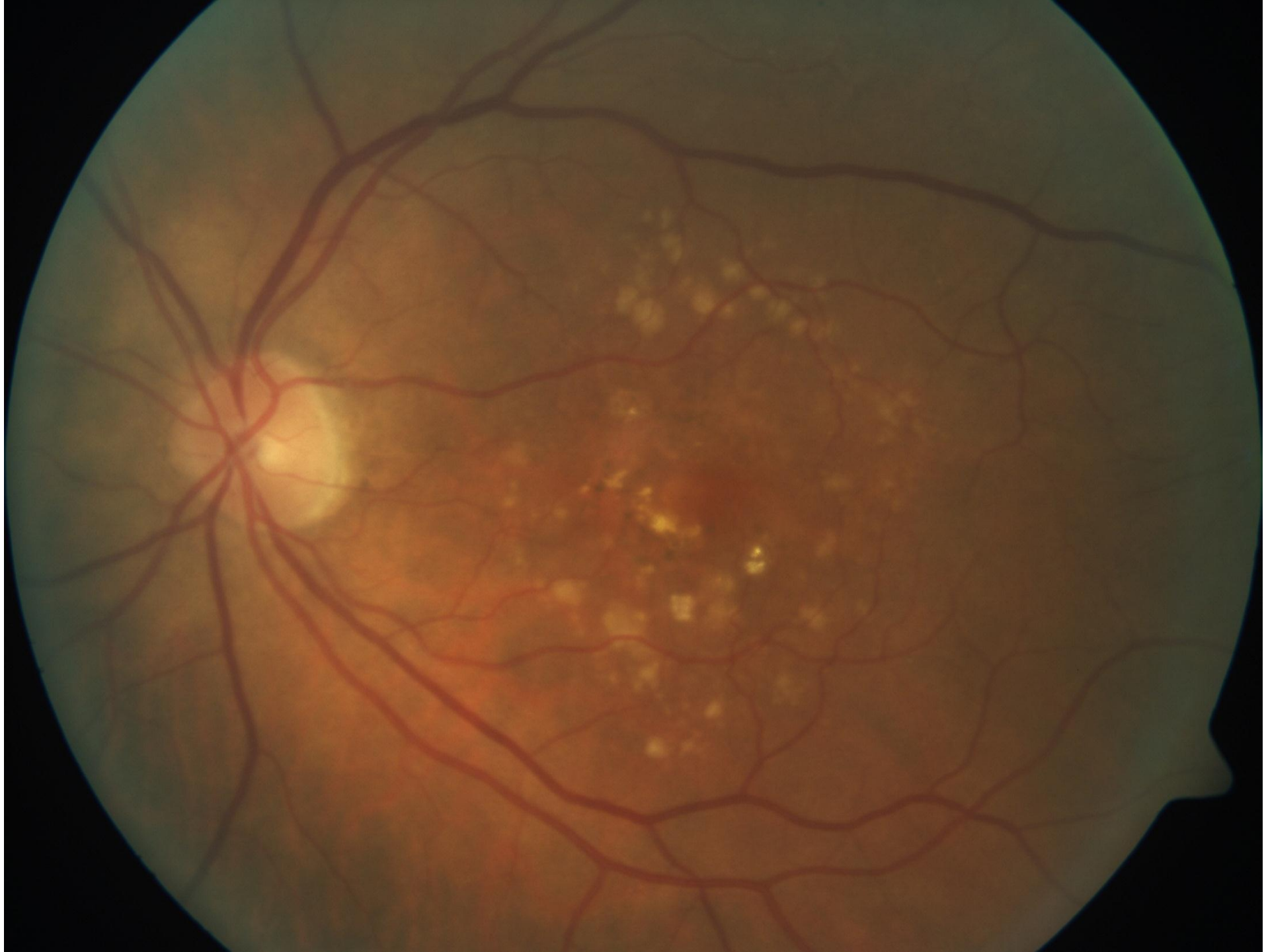


10/12/2020

FLIO-33, 71 years

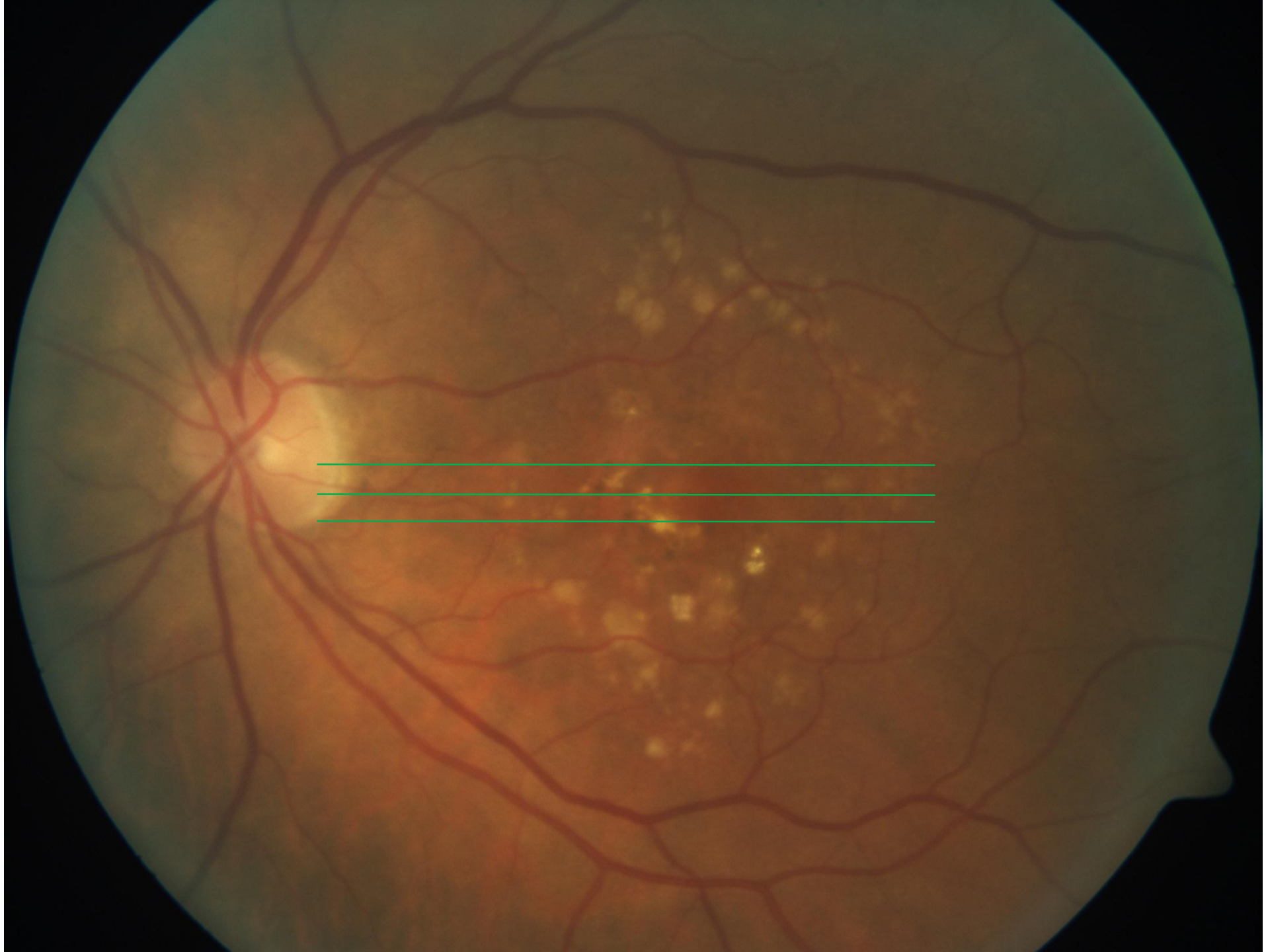
In this Patient, I am wondering about the long lifetimes in the macula. As in all other subjects, FAF intensity is lowest in the macula because macular pigment (MP) absorbs the excitation light. However, MP also has an intrinsic fluorescence with short lifetime (Sauer et al. IOVS 56(2015) 4668-79 and IOVS 59(2018) 3094-3103). Here we see no lack of MP (low macular FAF intensity) but long lifetimes in the macula. One explanation would be the incipient cataract of the subject. In this case, the weak short FAF of the macula would be overlaid by a strong, long living lens fluorescence (which is suppressed but not completely eliminated by the confocal scanning principle of FLIO). This explanation, however, is not completely convincing as the lens emits much stronger in SSC than in LSC, but here we see the long lifetimes in the macula predominantly in LSC. **What might be the reason?**

04/09/2016



CFP

04/09/2016



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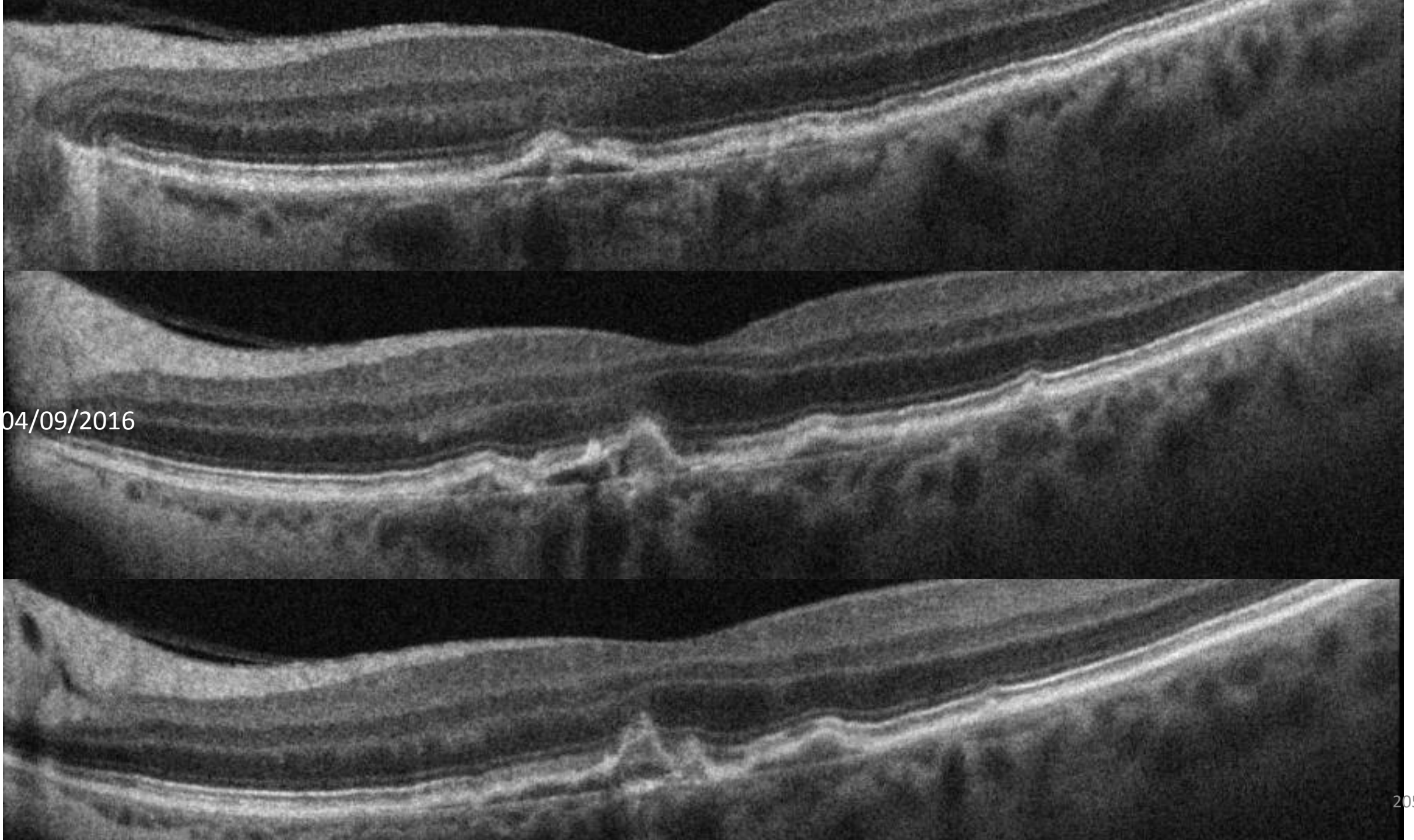
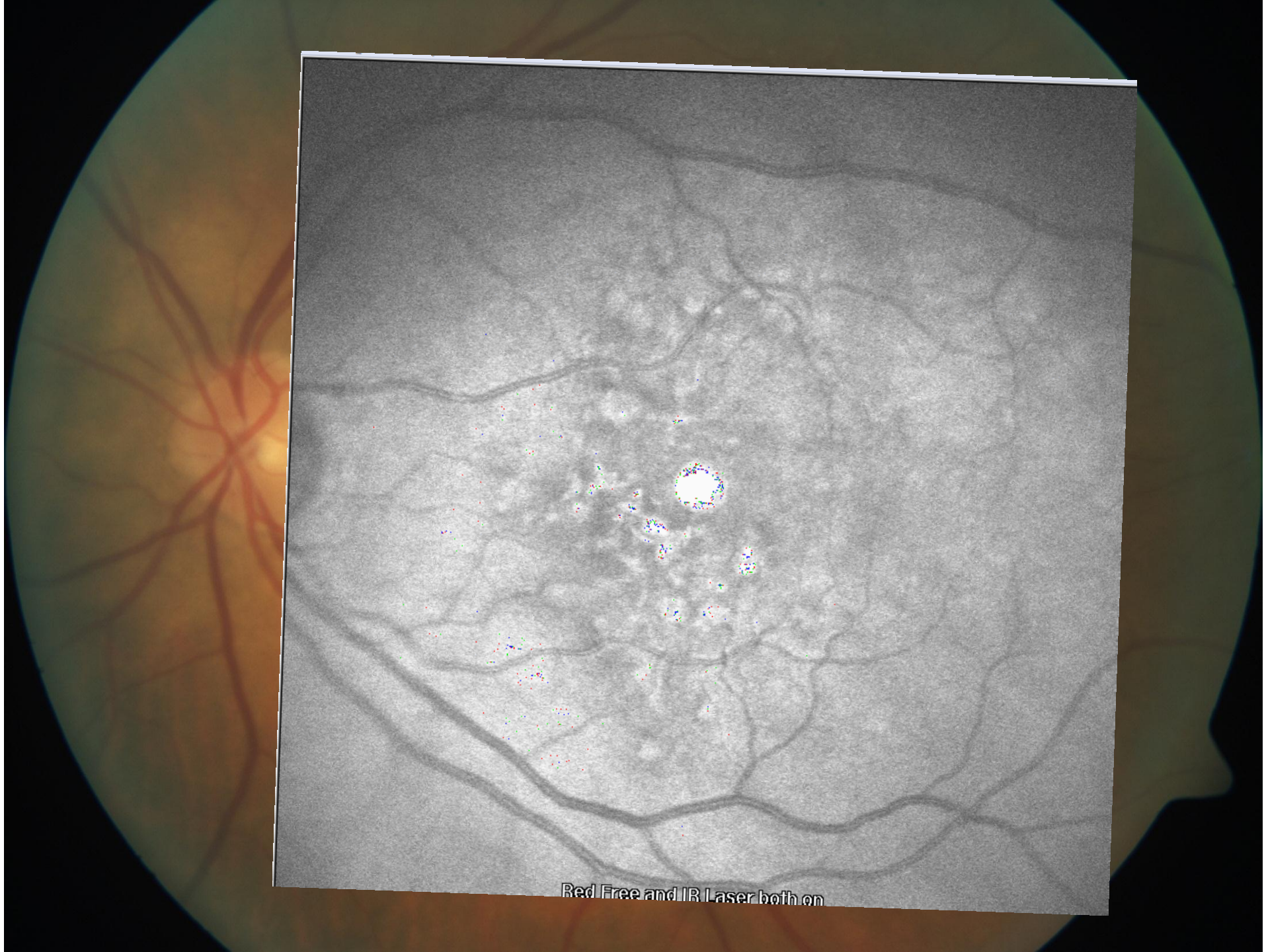
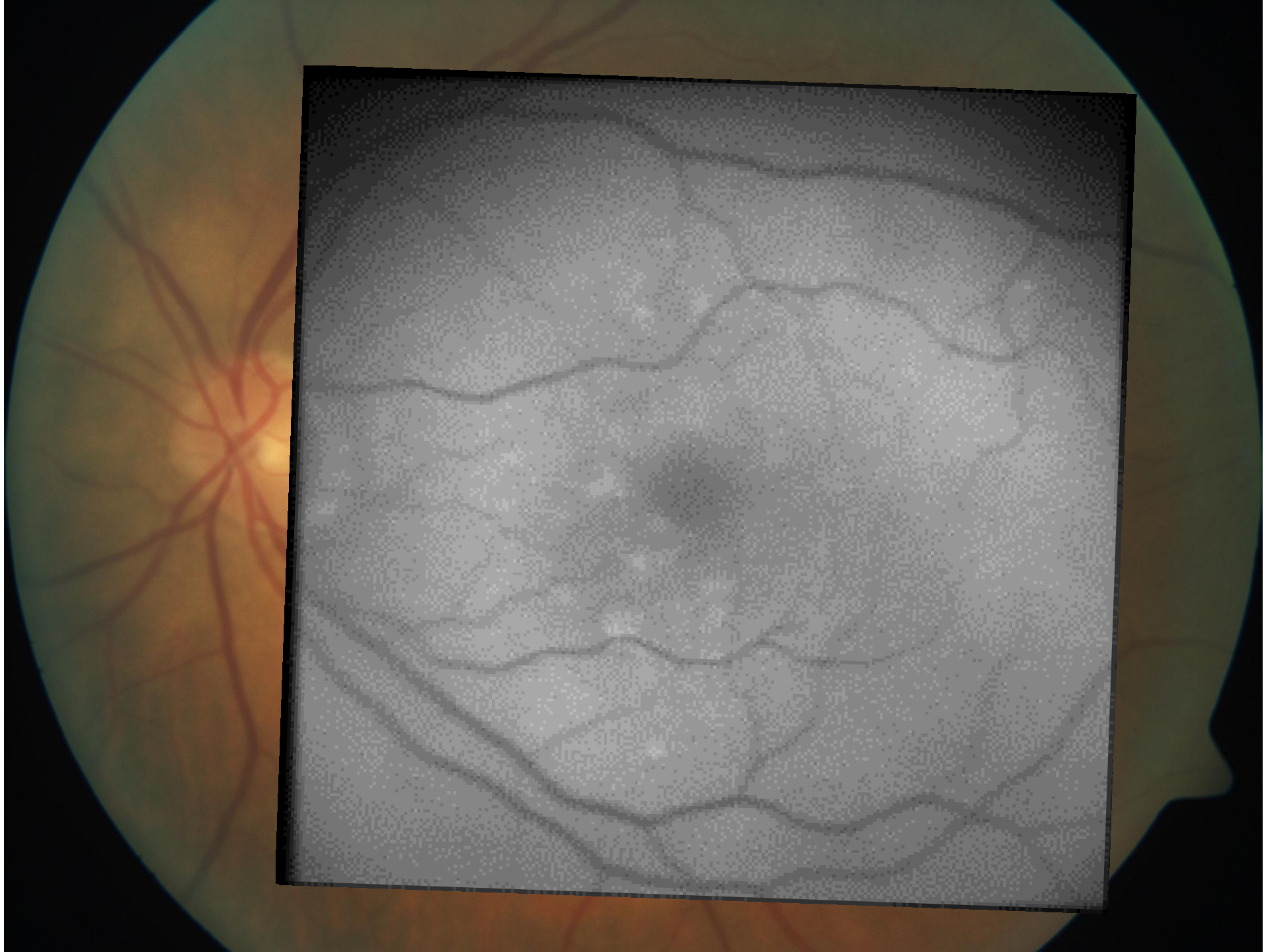

04/09/2016

04/09/2016

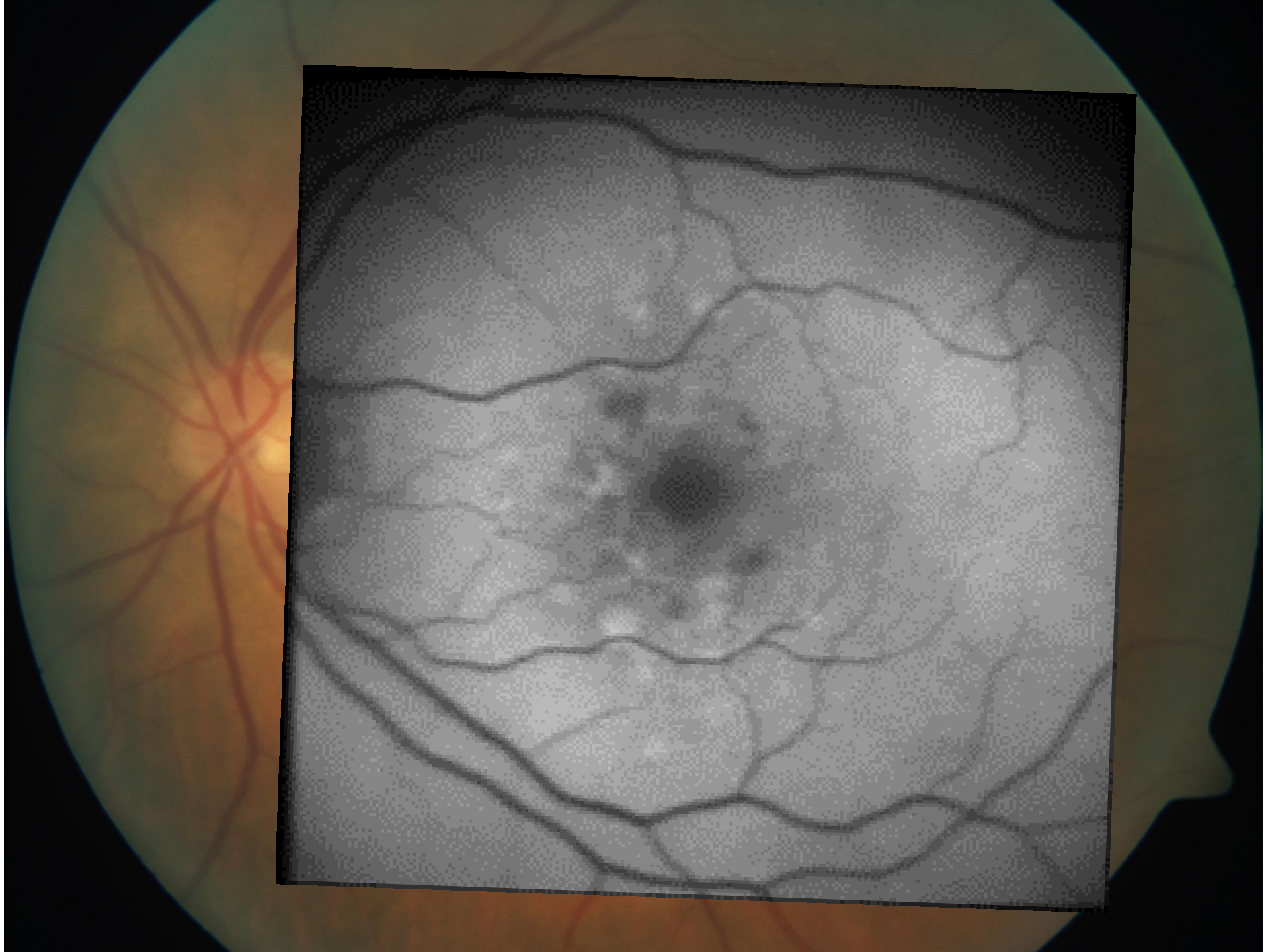


IR

04/09/2016

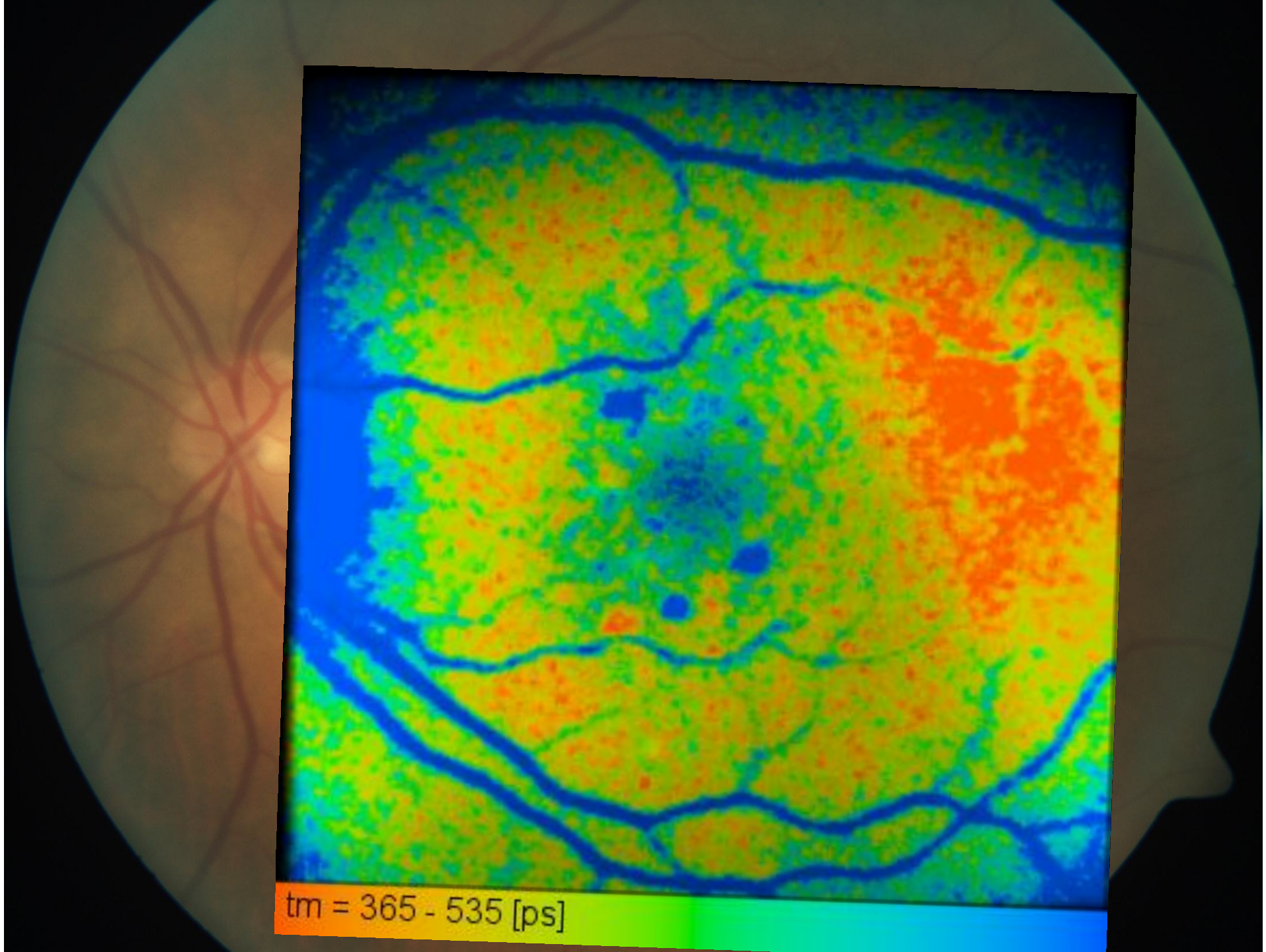


04/09/2016



FAF LSC

04/09/2016



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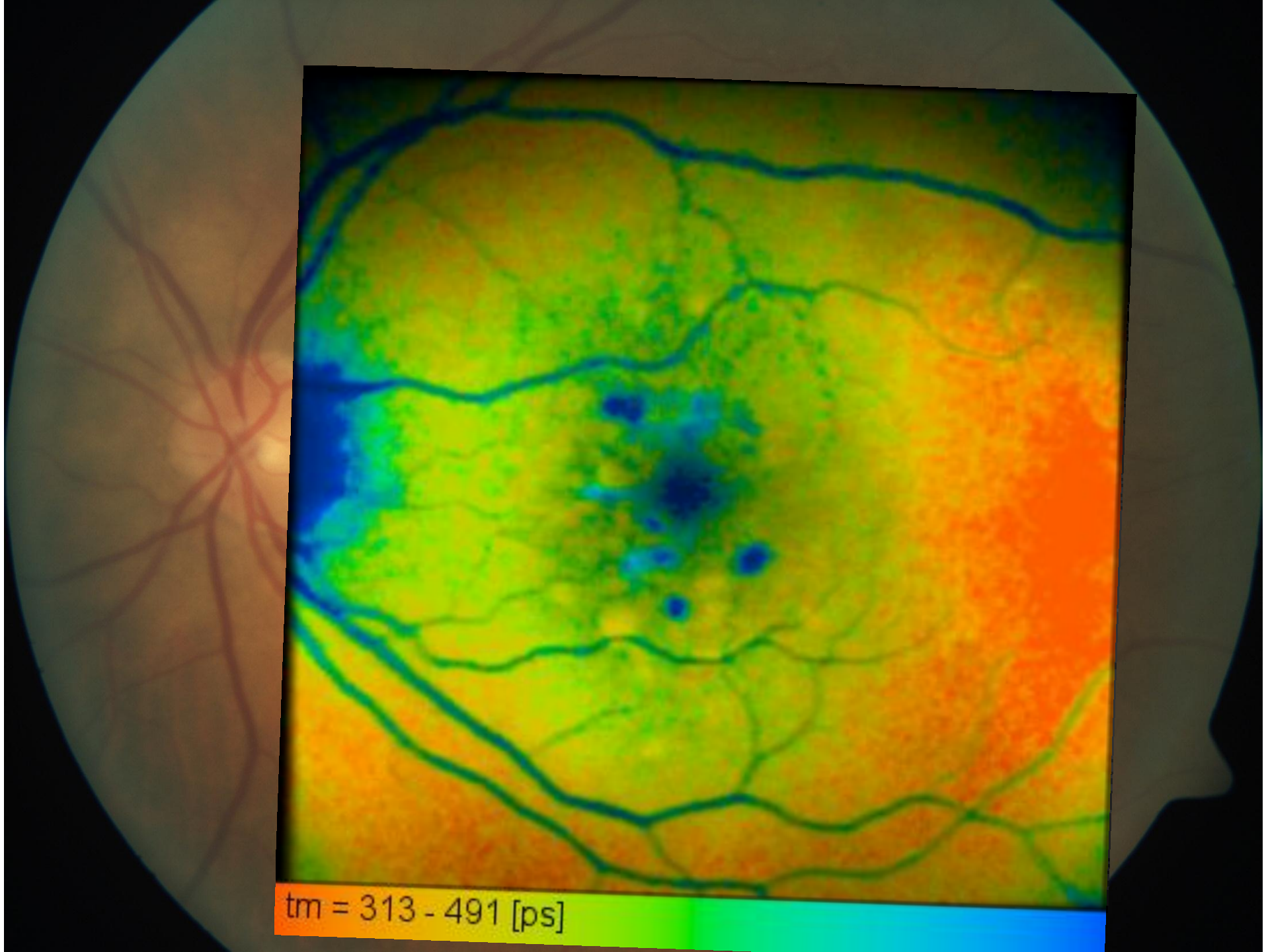
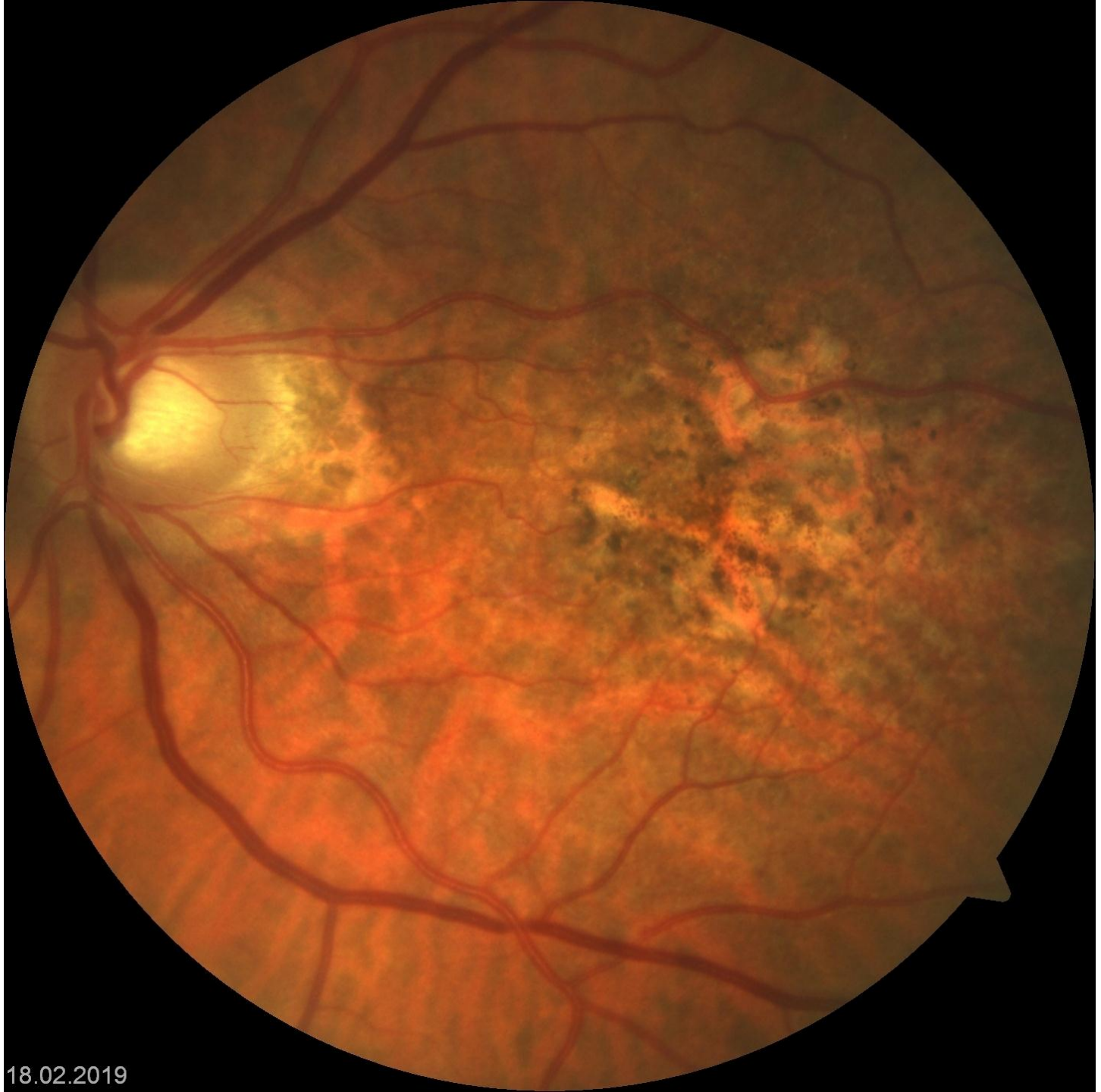


FLIO-37, 73 years

Patient with an atrophic scar showing long FAF lifetimes, However, FAF intensity is increased in SSC, in LSC there is also stronger FAF as we normally see it in GA. OCT (sorry, no HD scans available) shows increased macular thickness (central subfield thickness: 317 µm, no macular pit was found) and massive accumulation of subretinal material (arrowhead). **Is that BlamD or rather connective tissue (fibrosis)?**

02/18/2016



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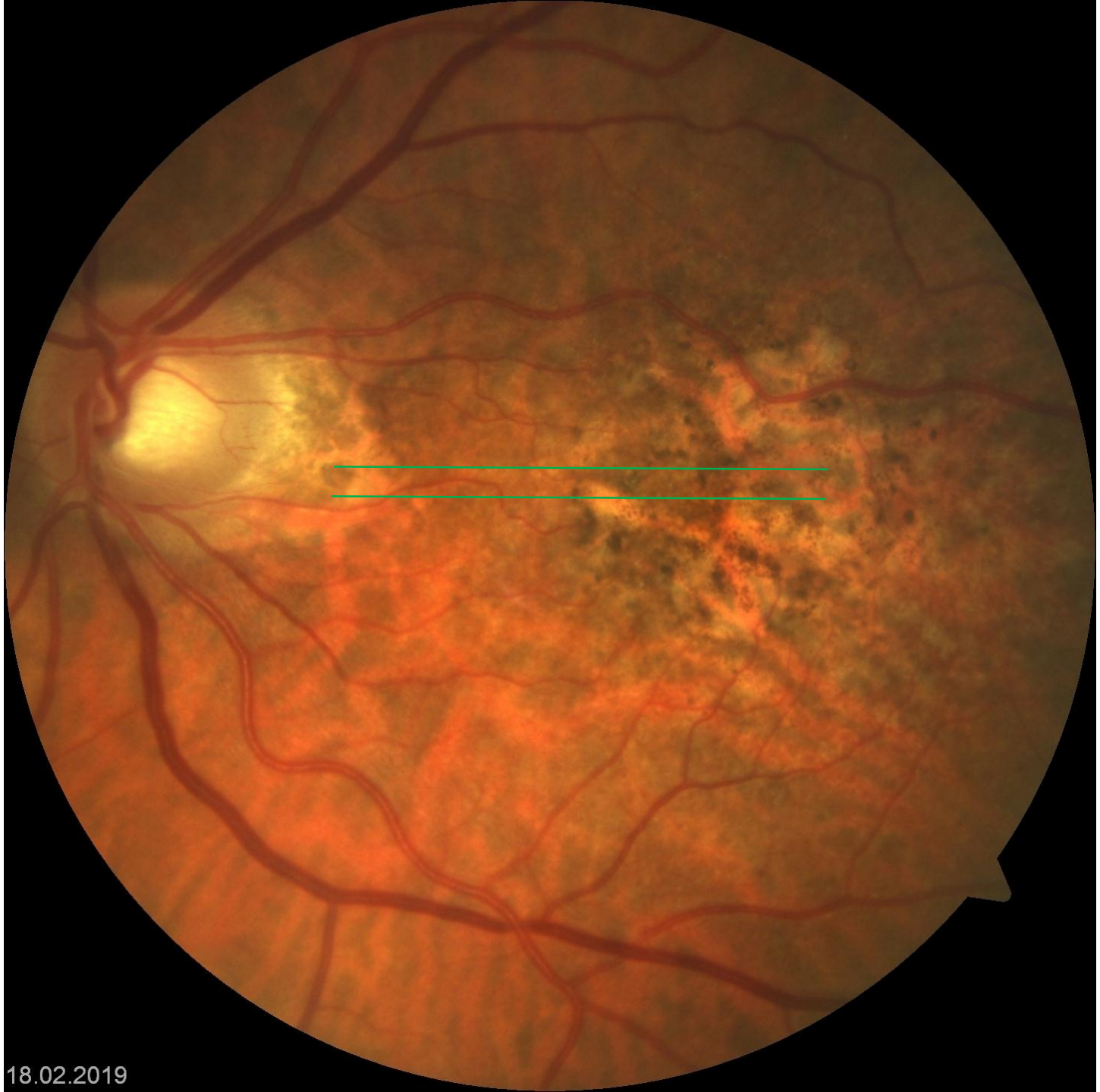
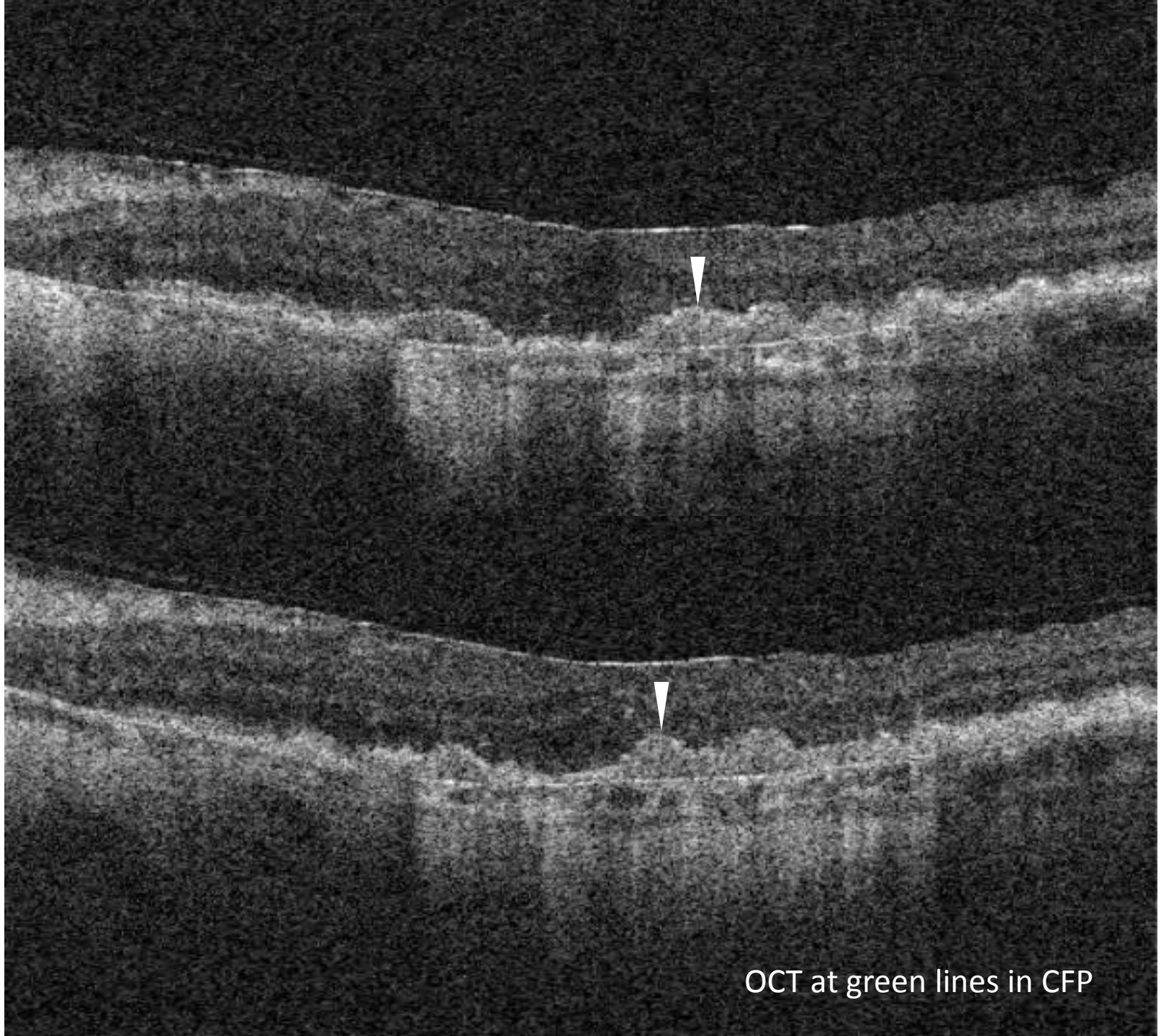
02/18/2016



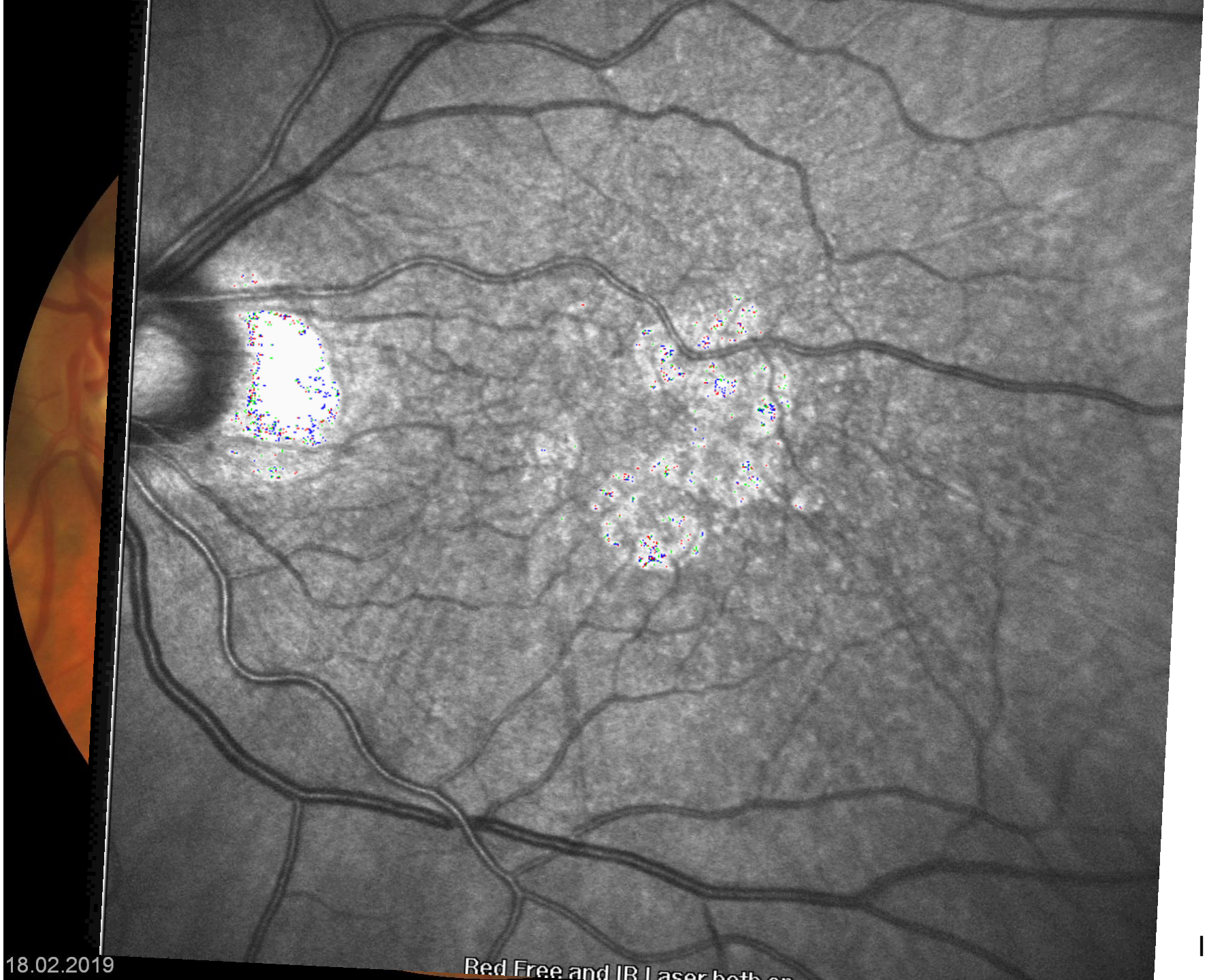
CFP

02/18/2016



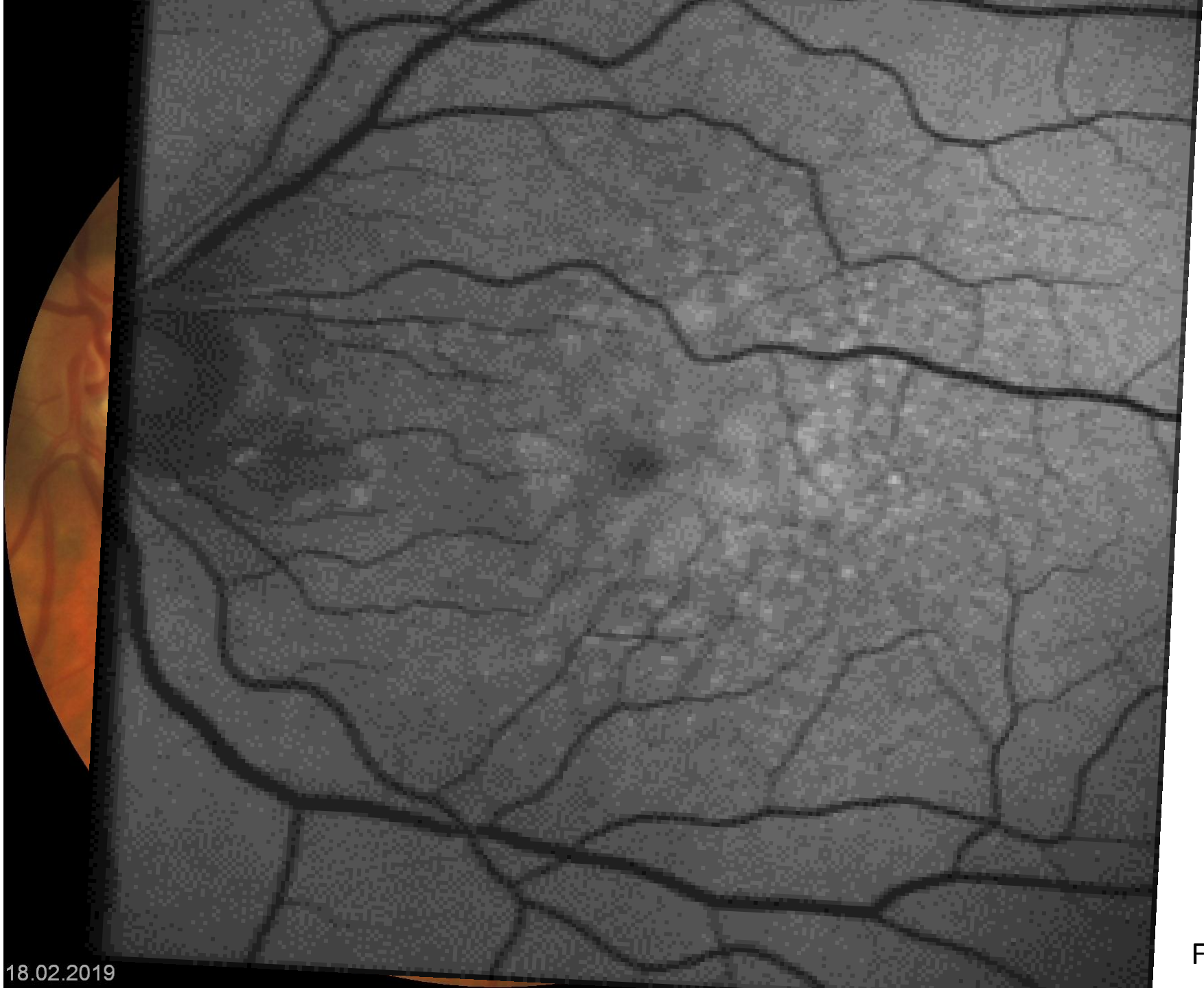
OCT at green lines in CFP

02/18/2016



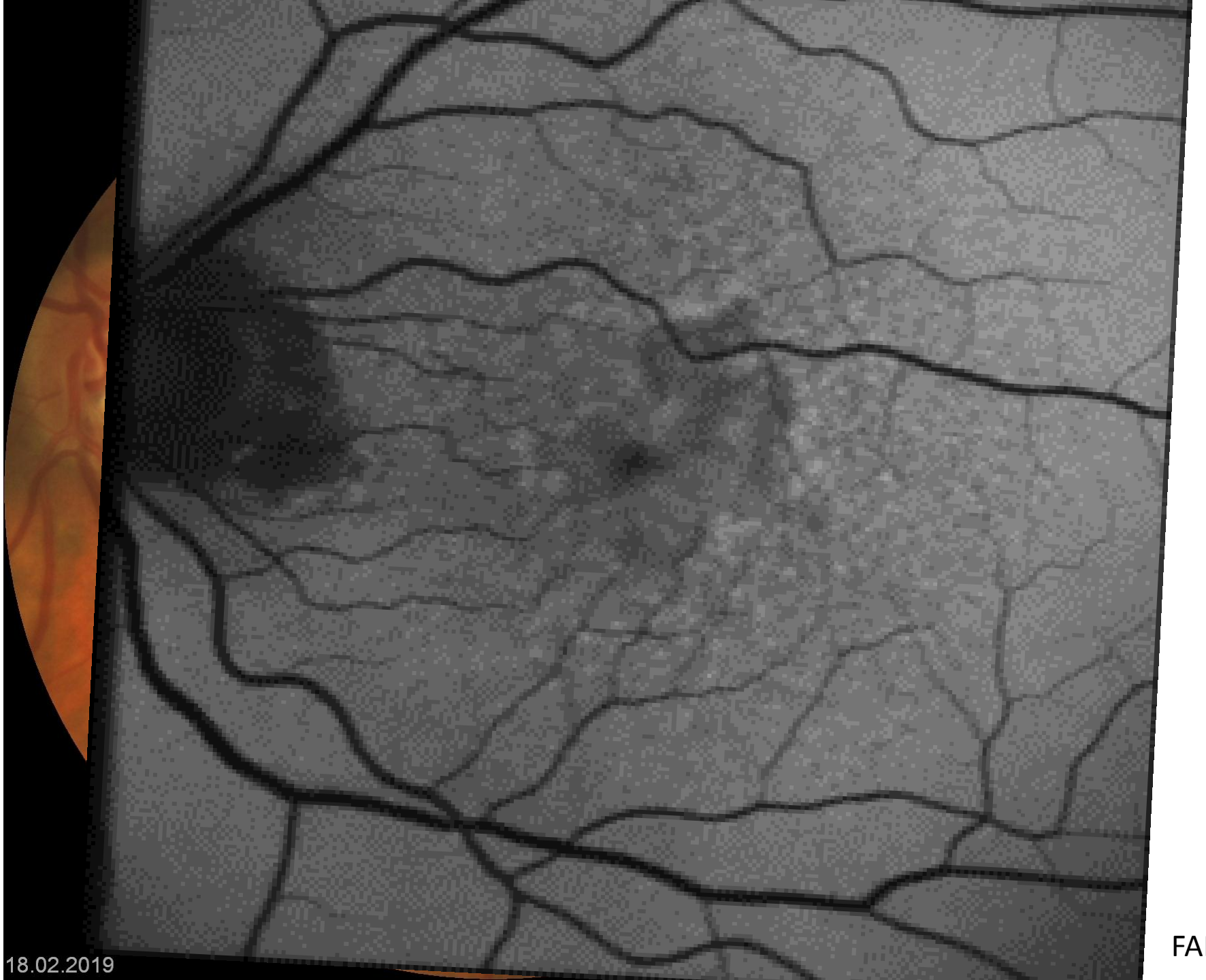
IR

02/18/2016



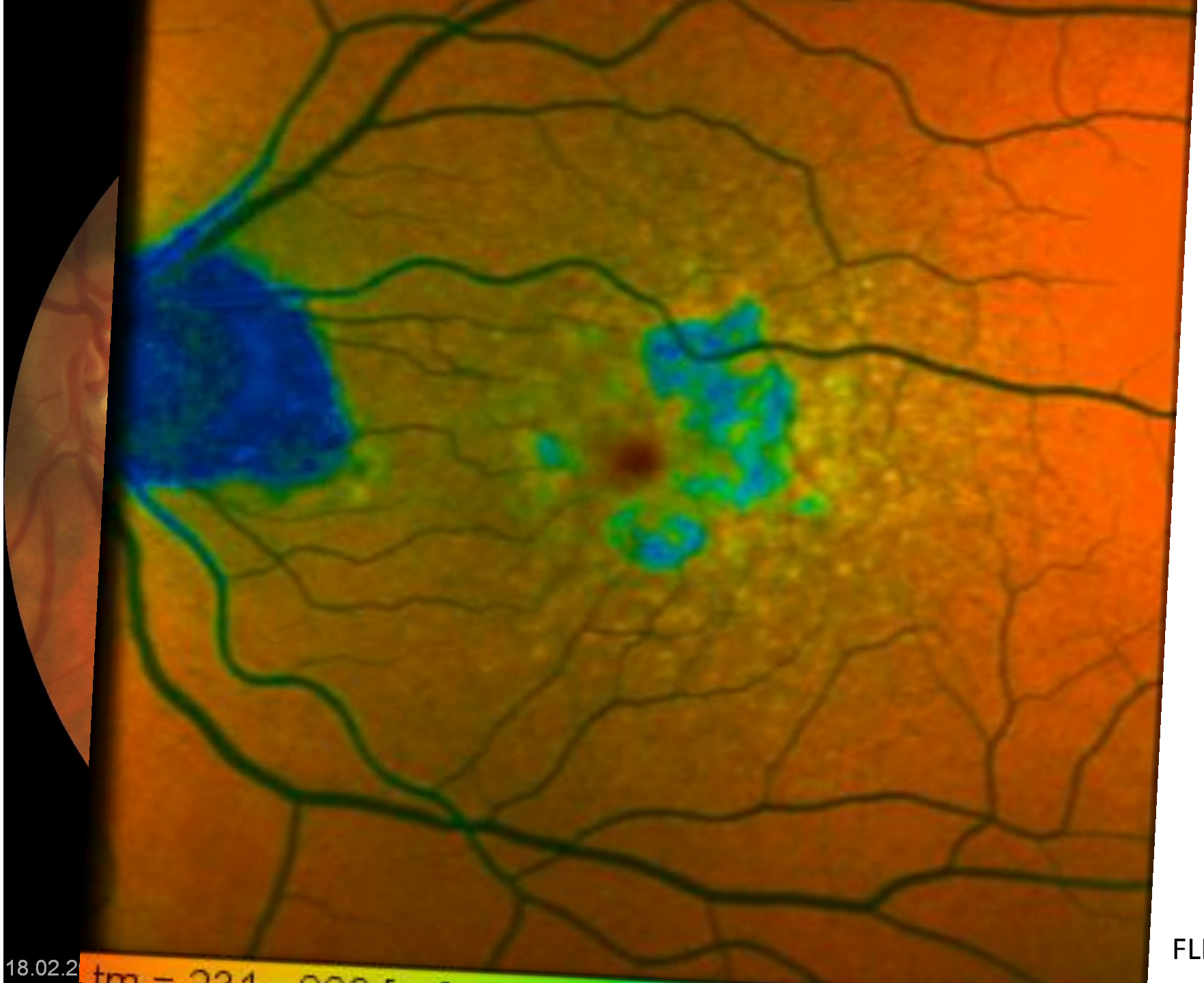
FAF SSC

02/18/2016

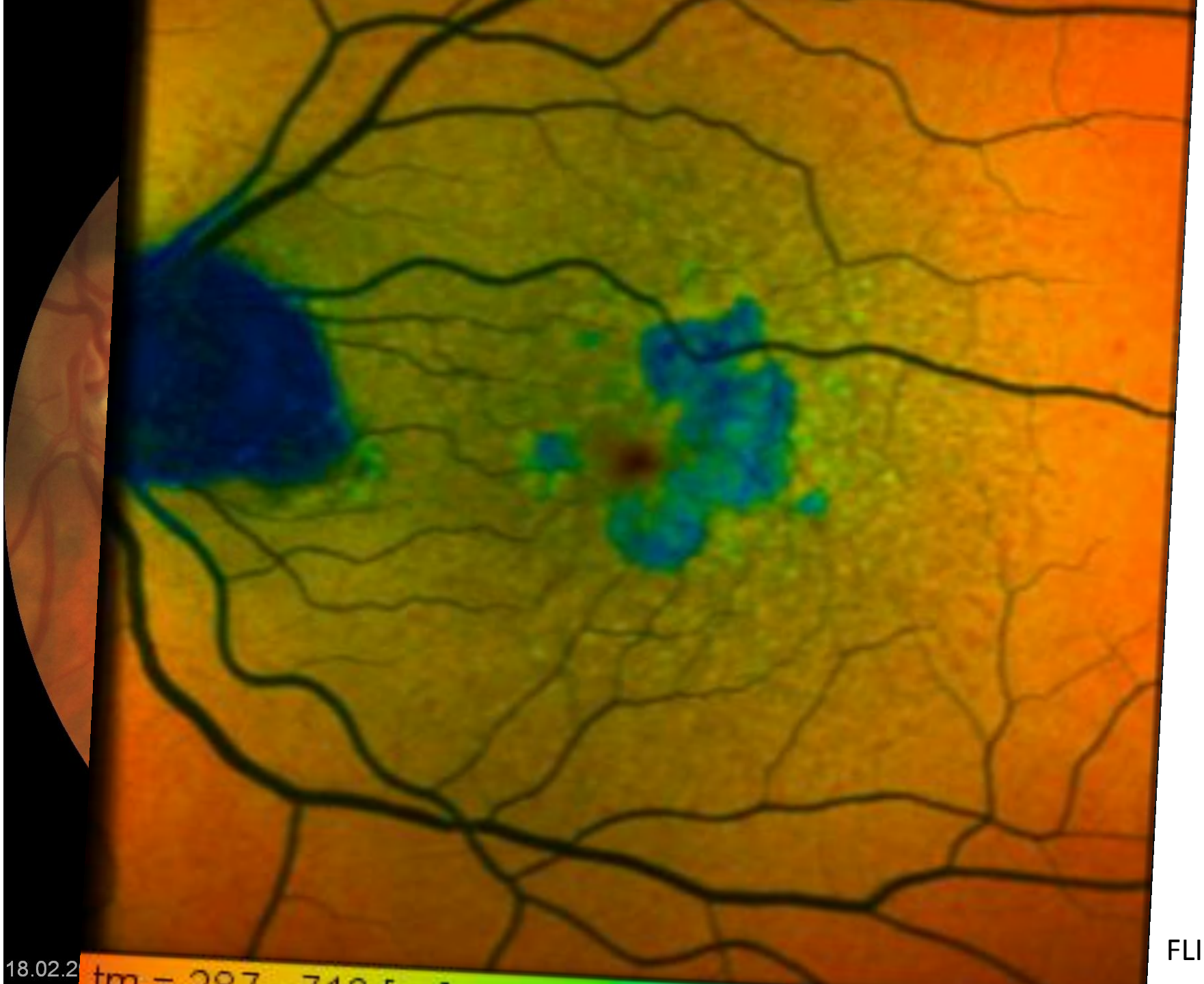


FAF LSC

02/18/2016



02/18/2016

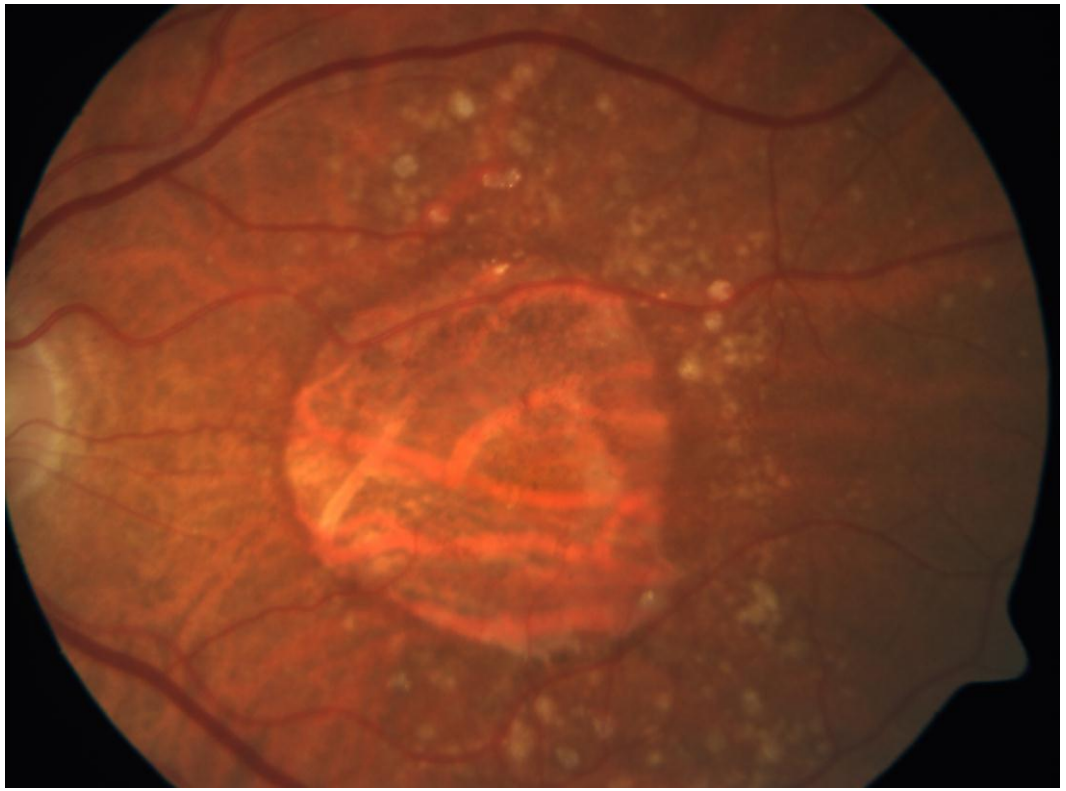


FLIO LSC

GA_19, 76 years at baseline

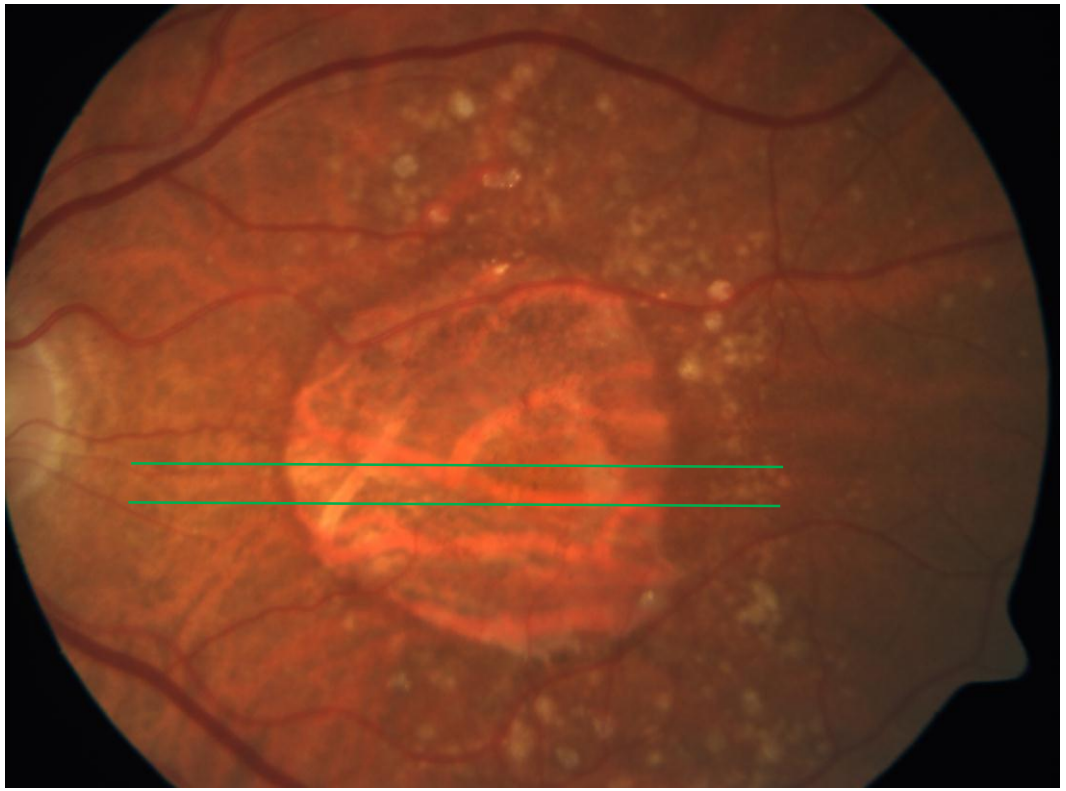
In this patient, we see a slightly hyperfluoerscent structure inside the GA which corresponds to hyperreflective material in OCT (arrowheads, clearly seen in the follow up, baseline OCT is poor). This material persists over 4 ½ years. In contrast to all other deposits or fibrotic tissue, it has short FAF lifetimes. But it is unlikely lipofuscin as the RPE is gone definitely. **What do we see here? BlamD?**

04/14/2014

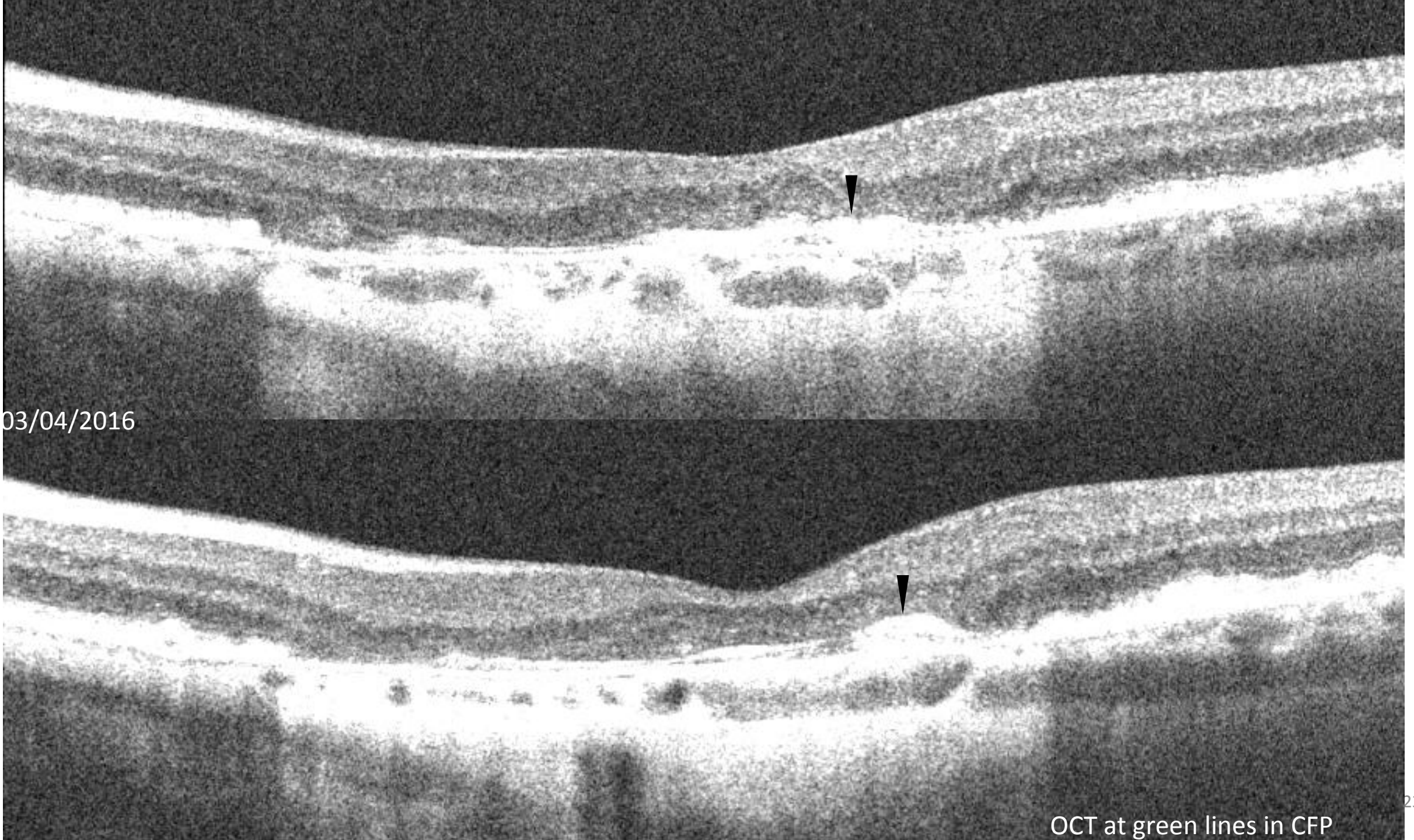


CFP

04/14/2014

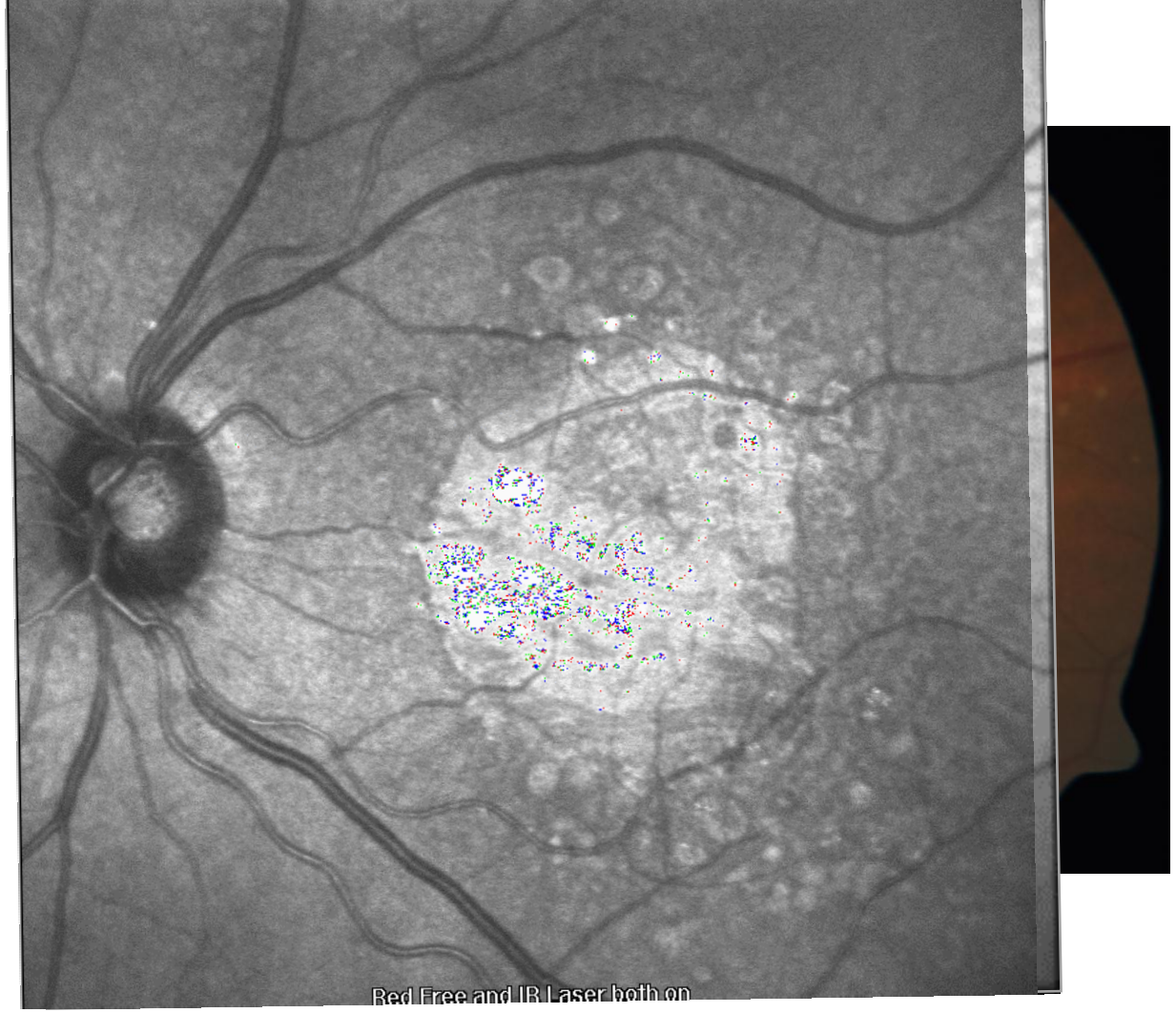


CFP



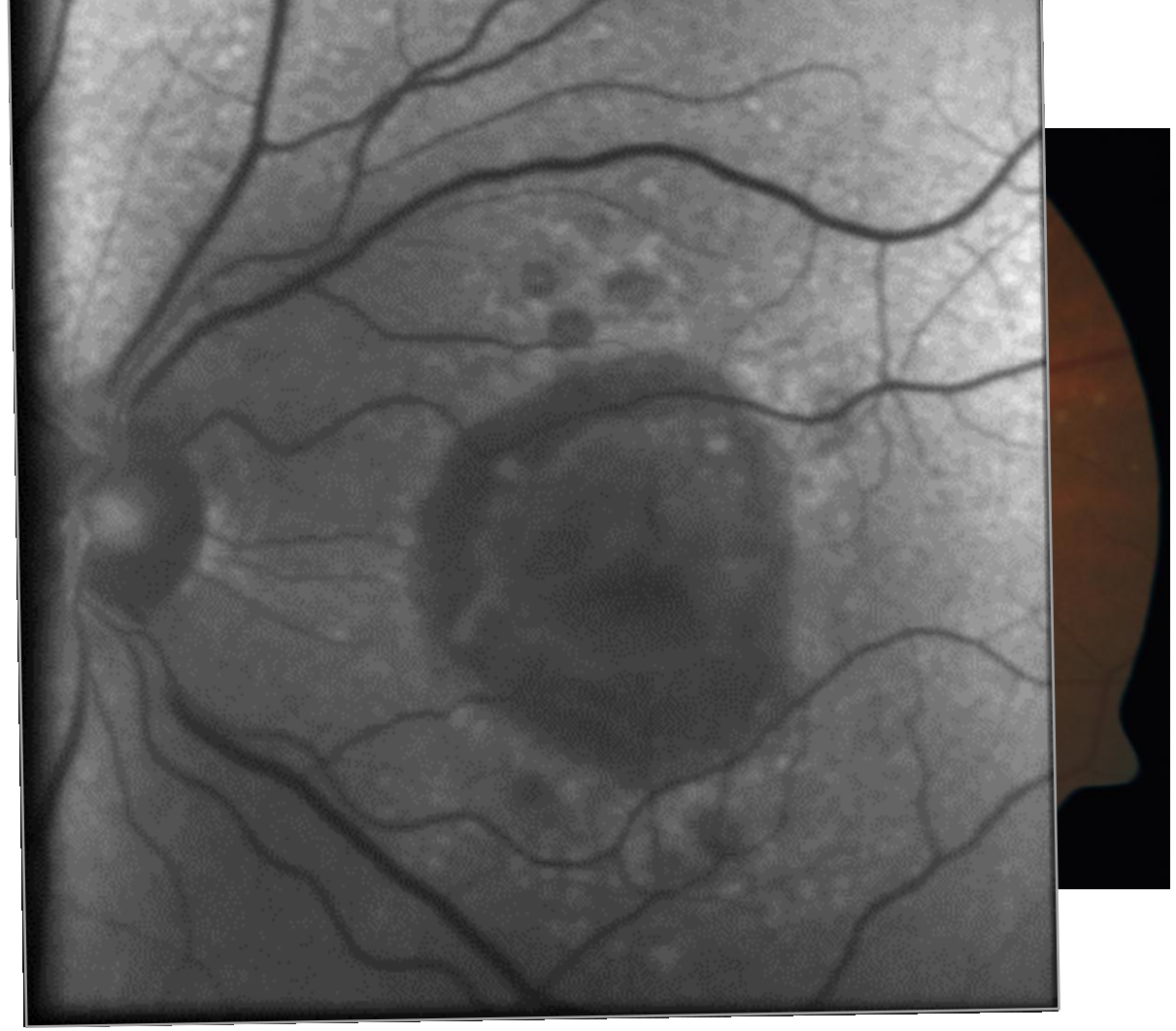
OCT at green lines in CFP

03/04/2016

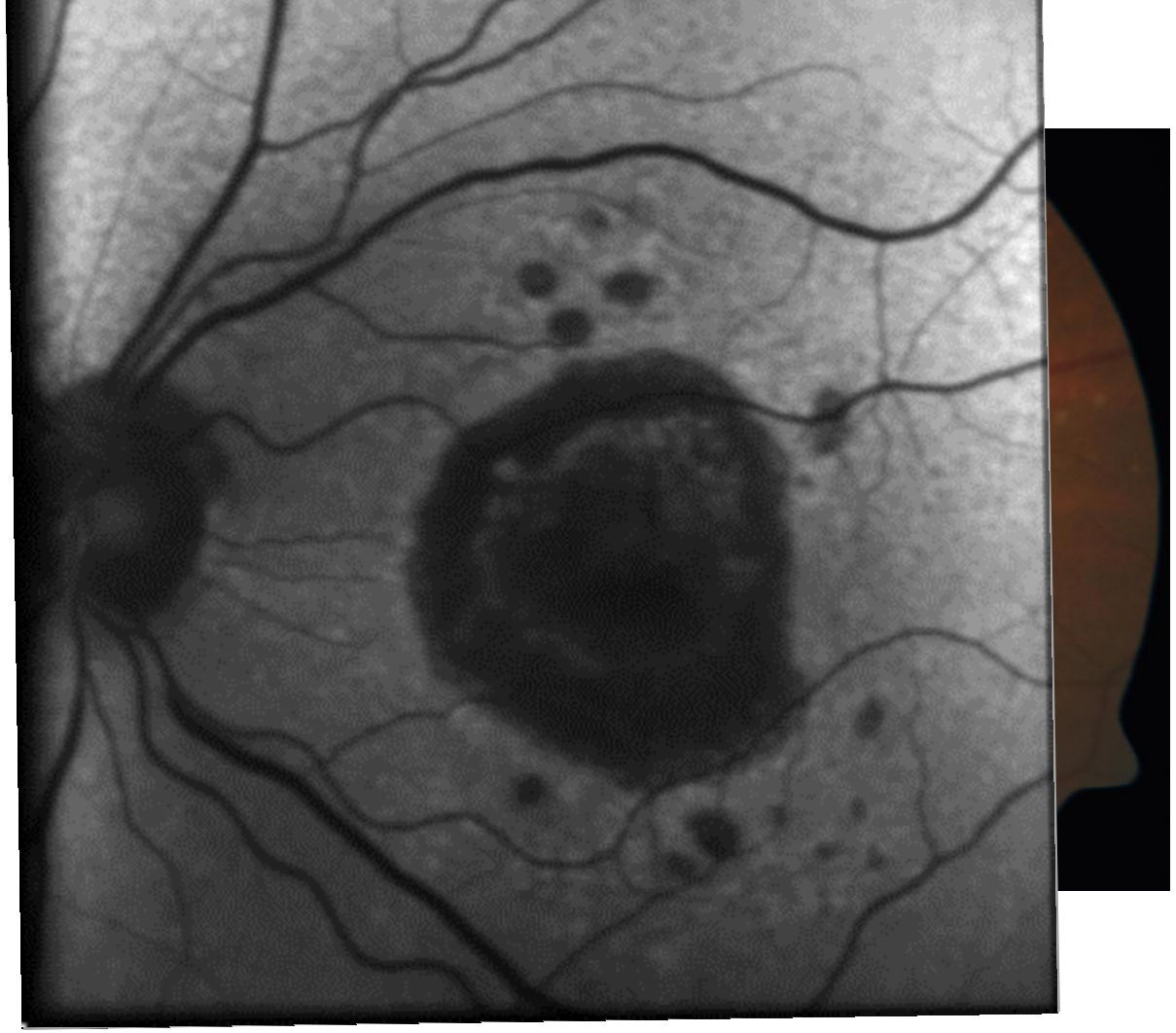


IR

03/04/2016



03/04/2016



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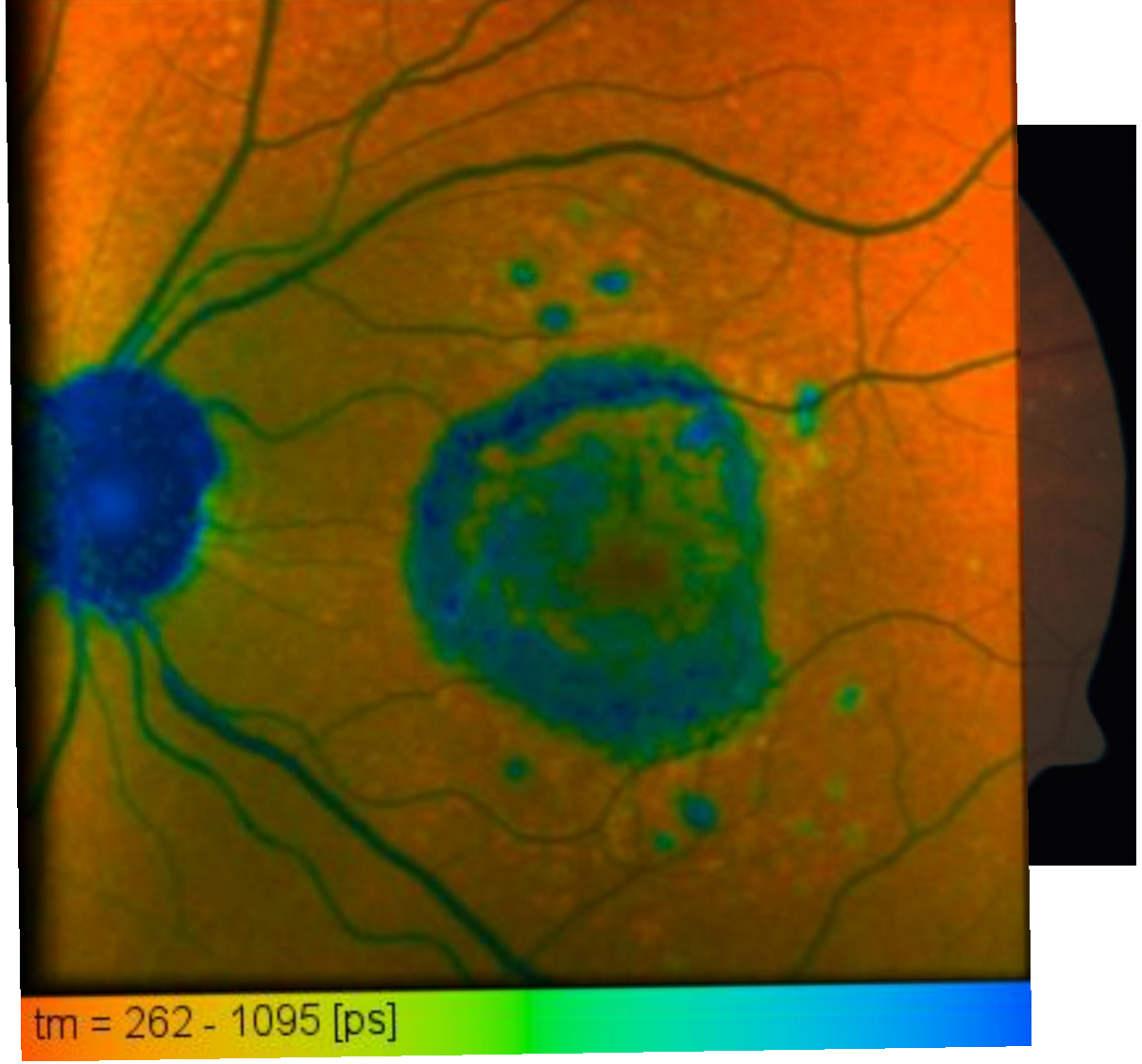
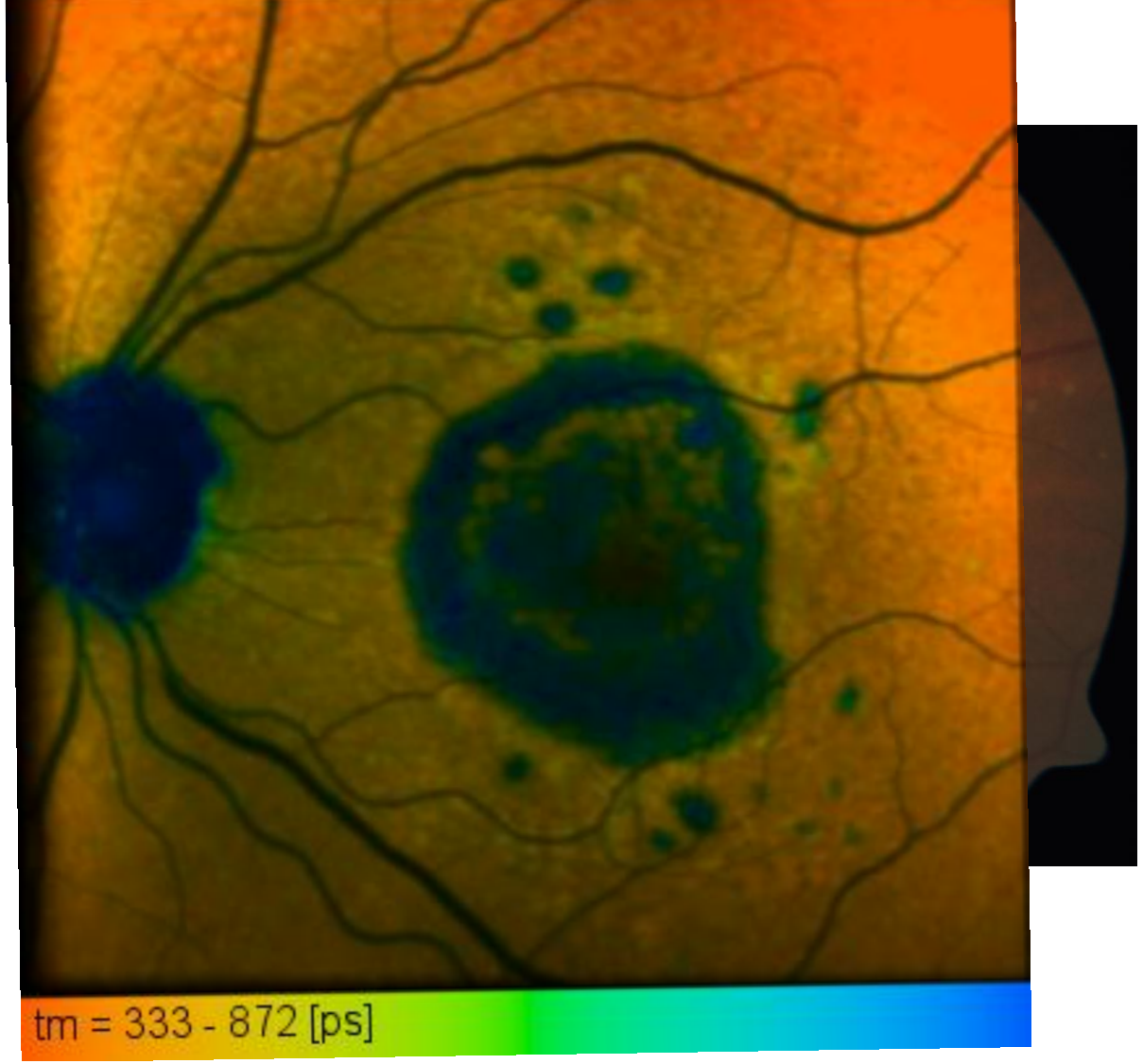
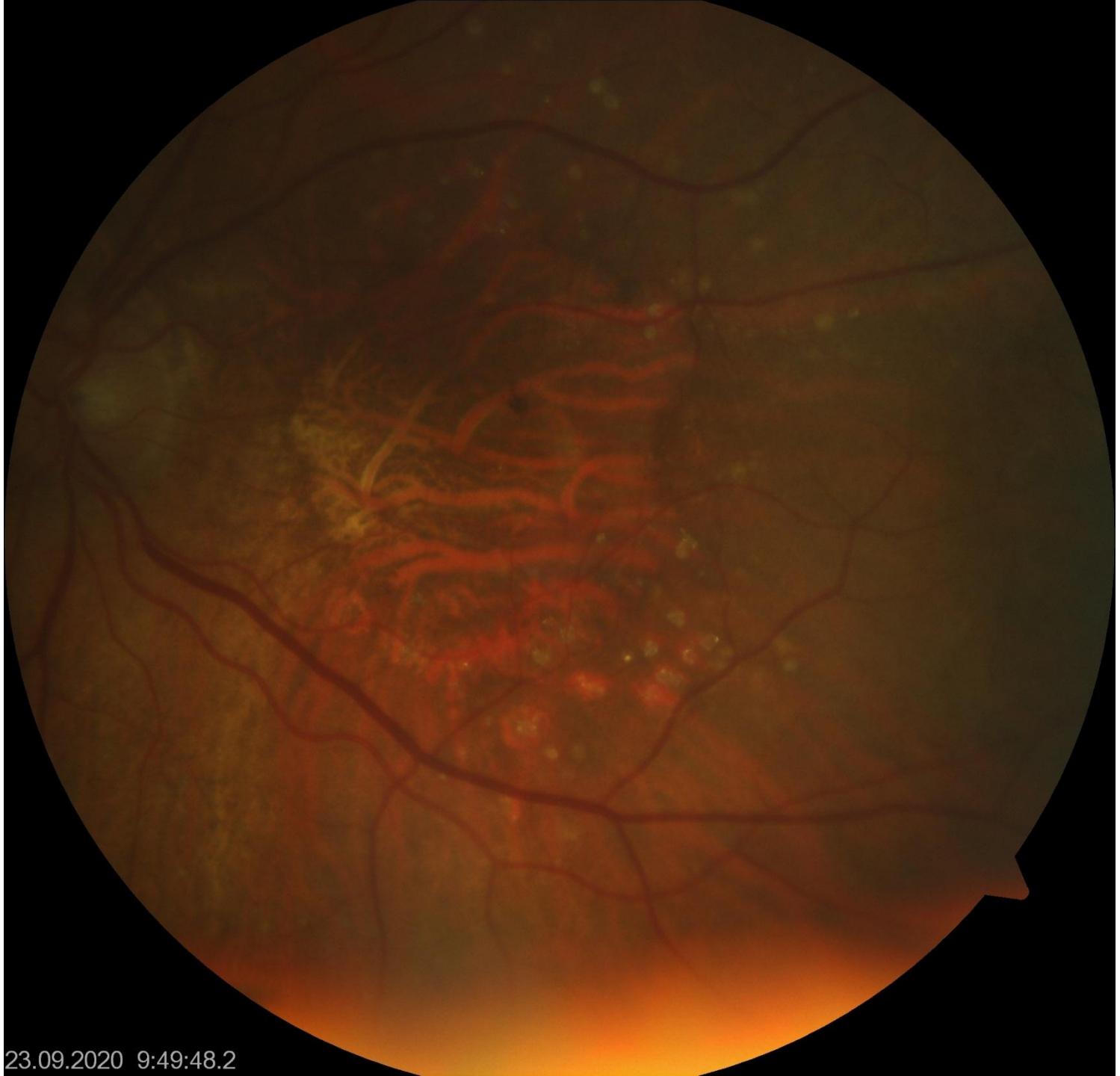


03/04/2016

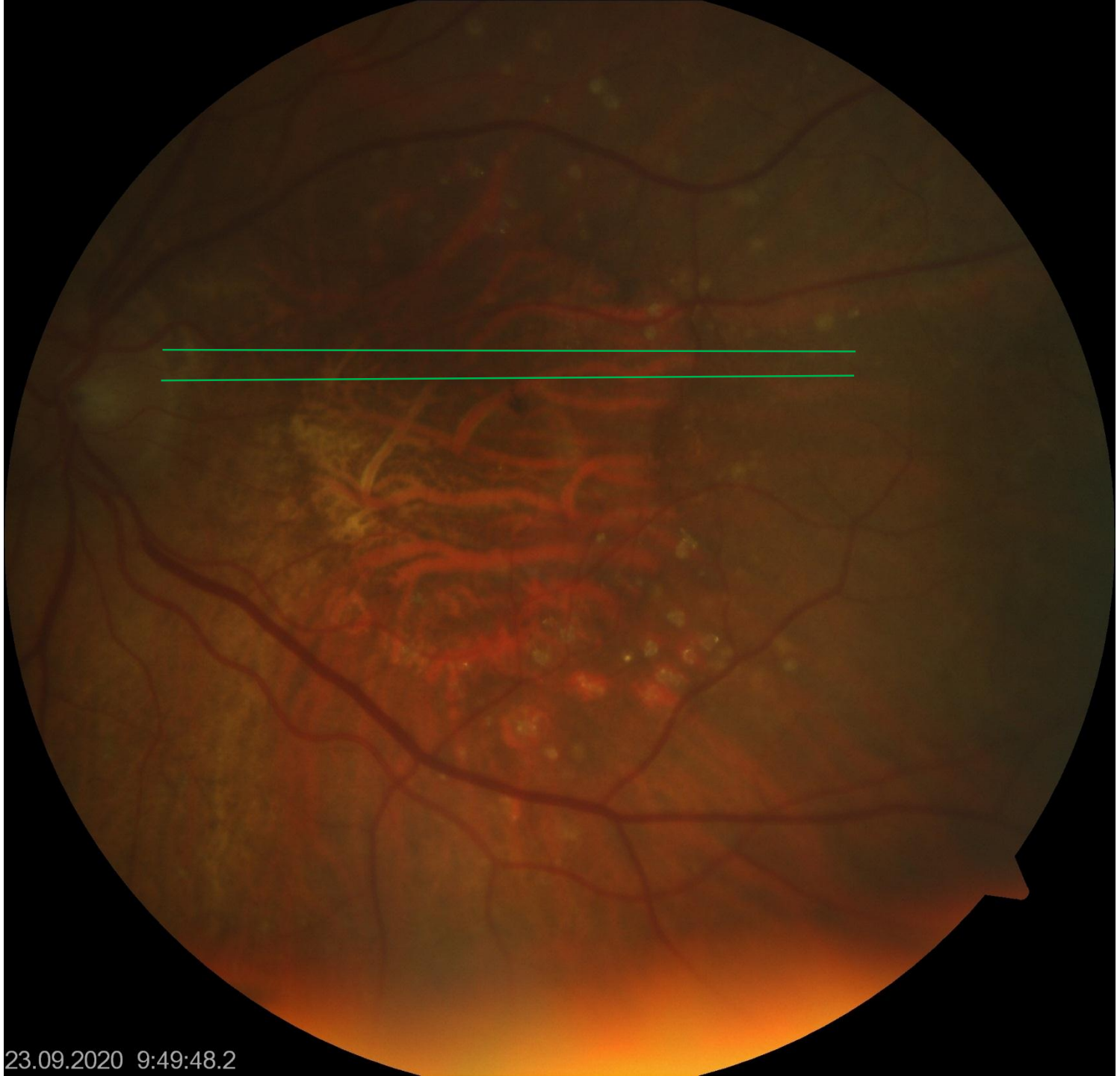


09/23/2016

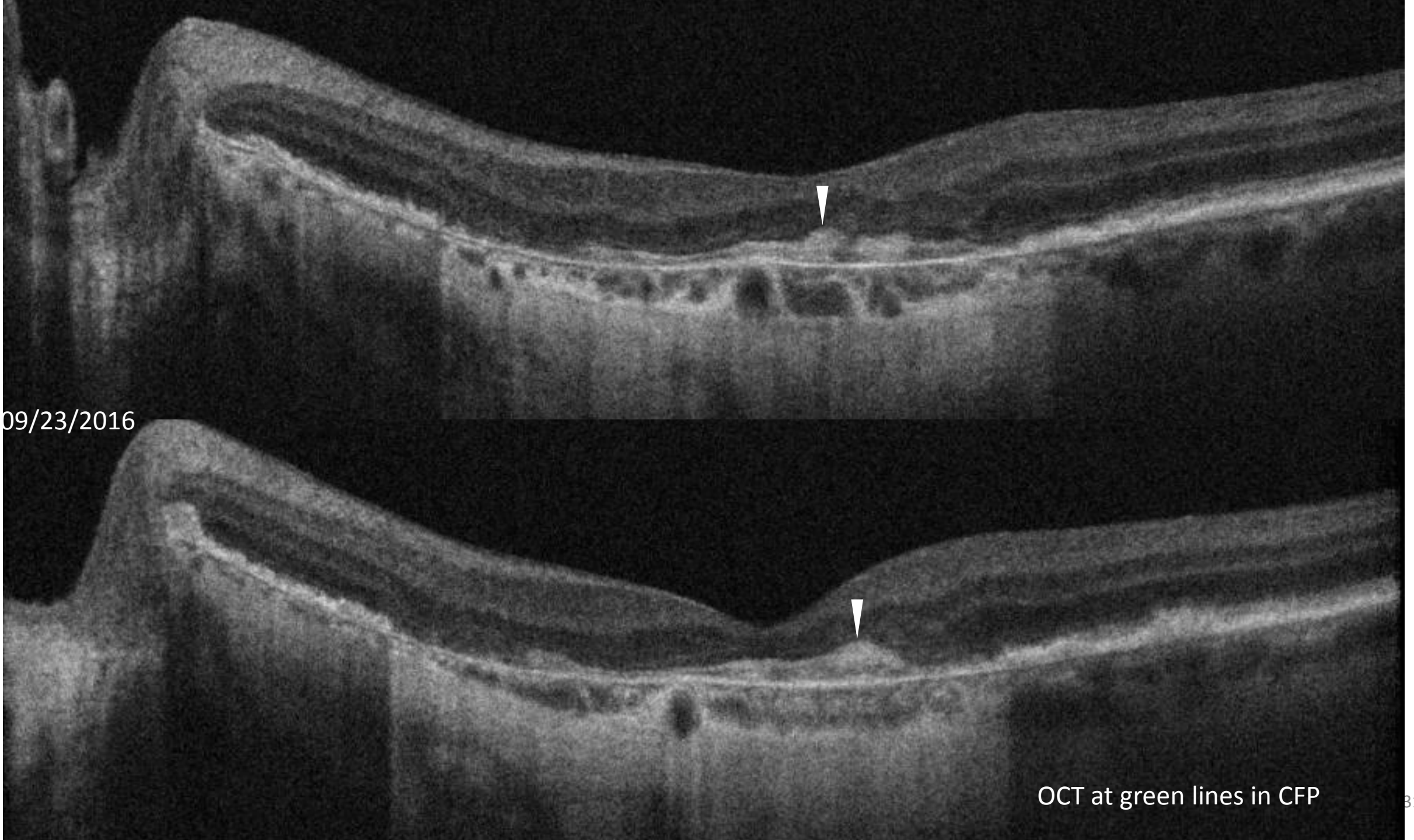


CFP

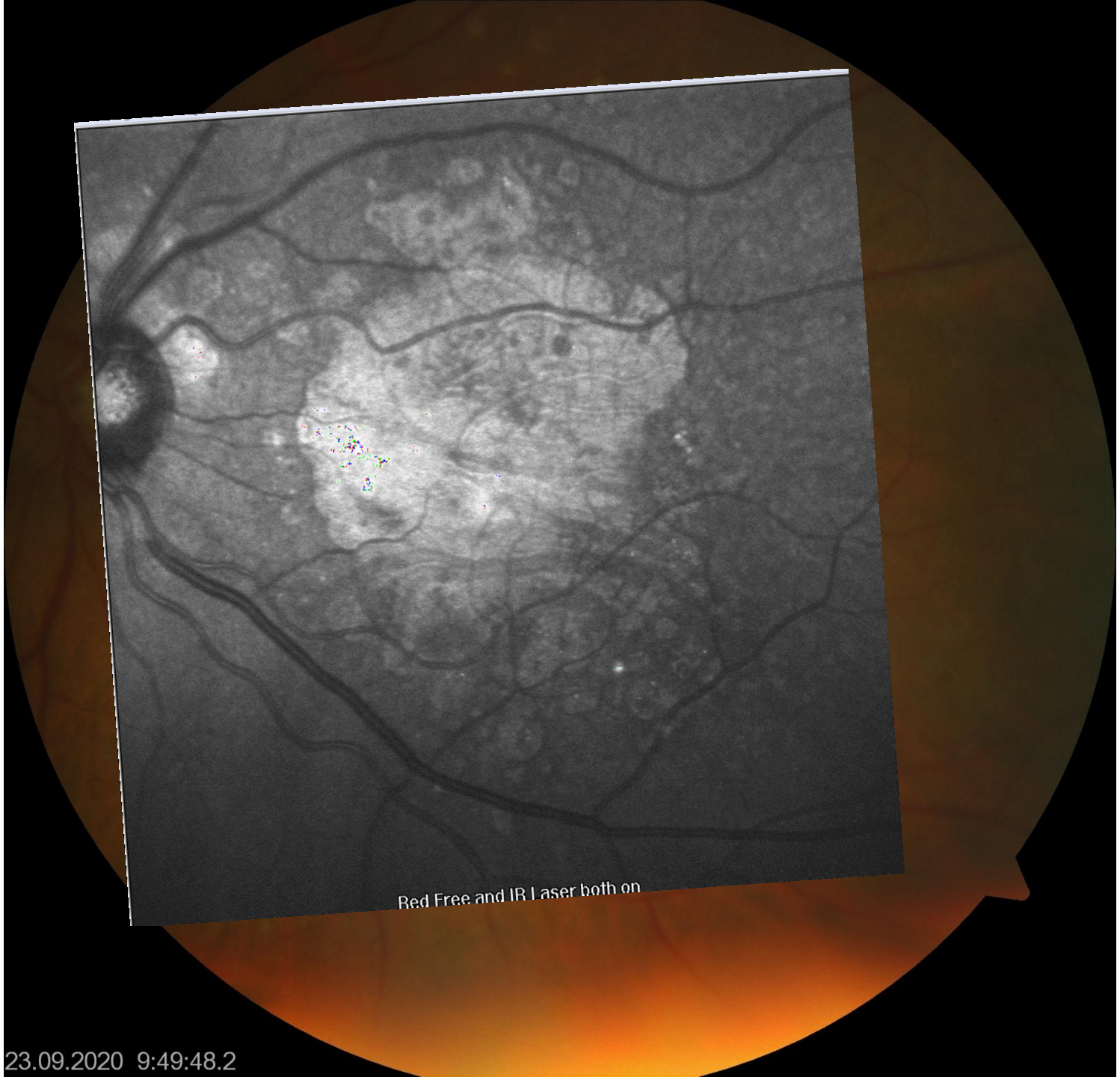
09/23/2016



CFP

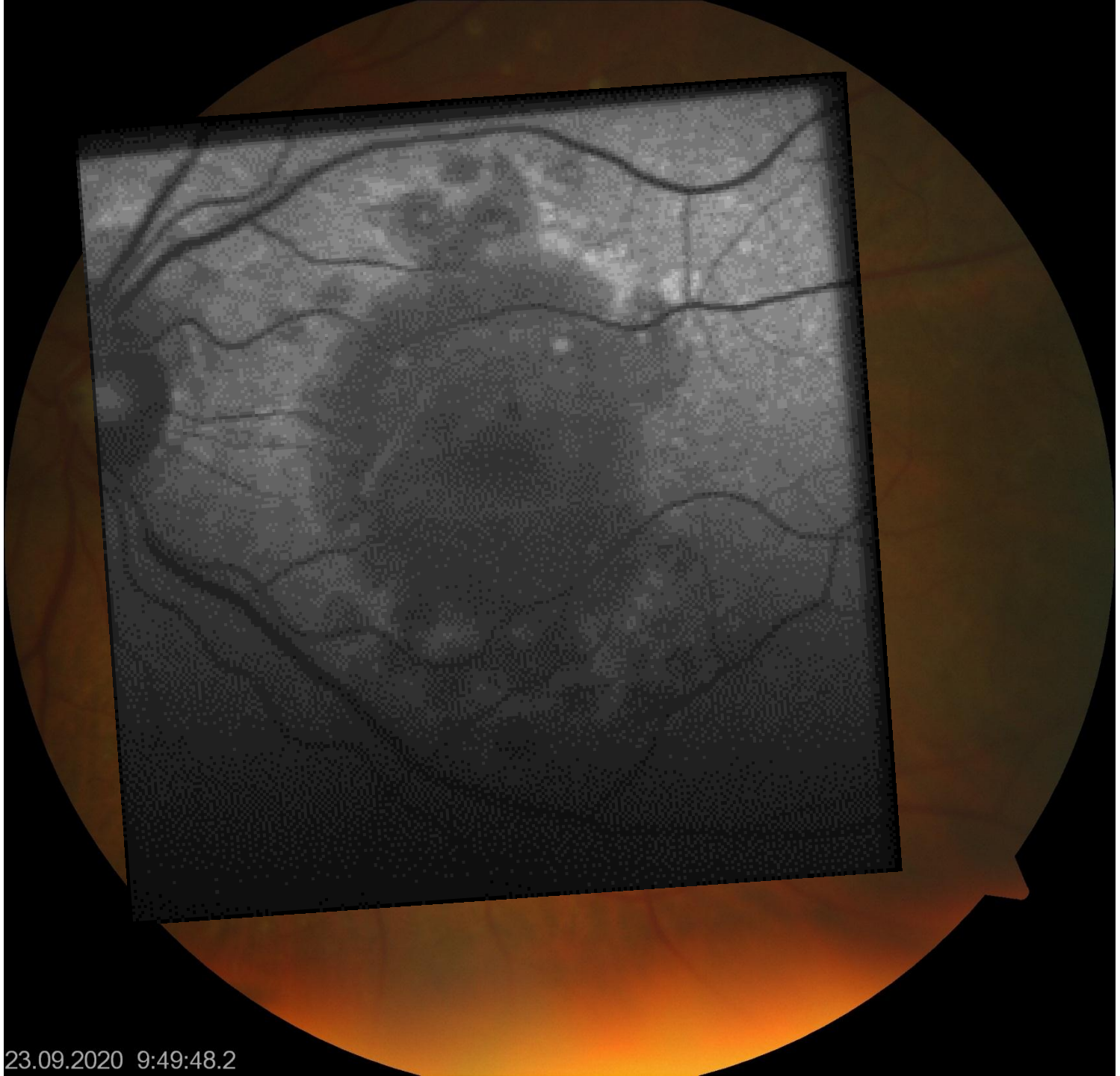

09/23/2016
OCT at green lines in CFP

09/23/2016



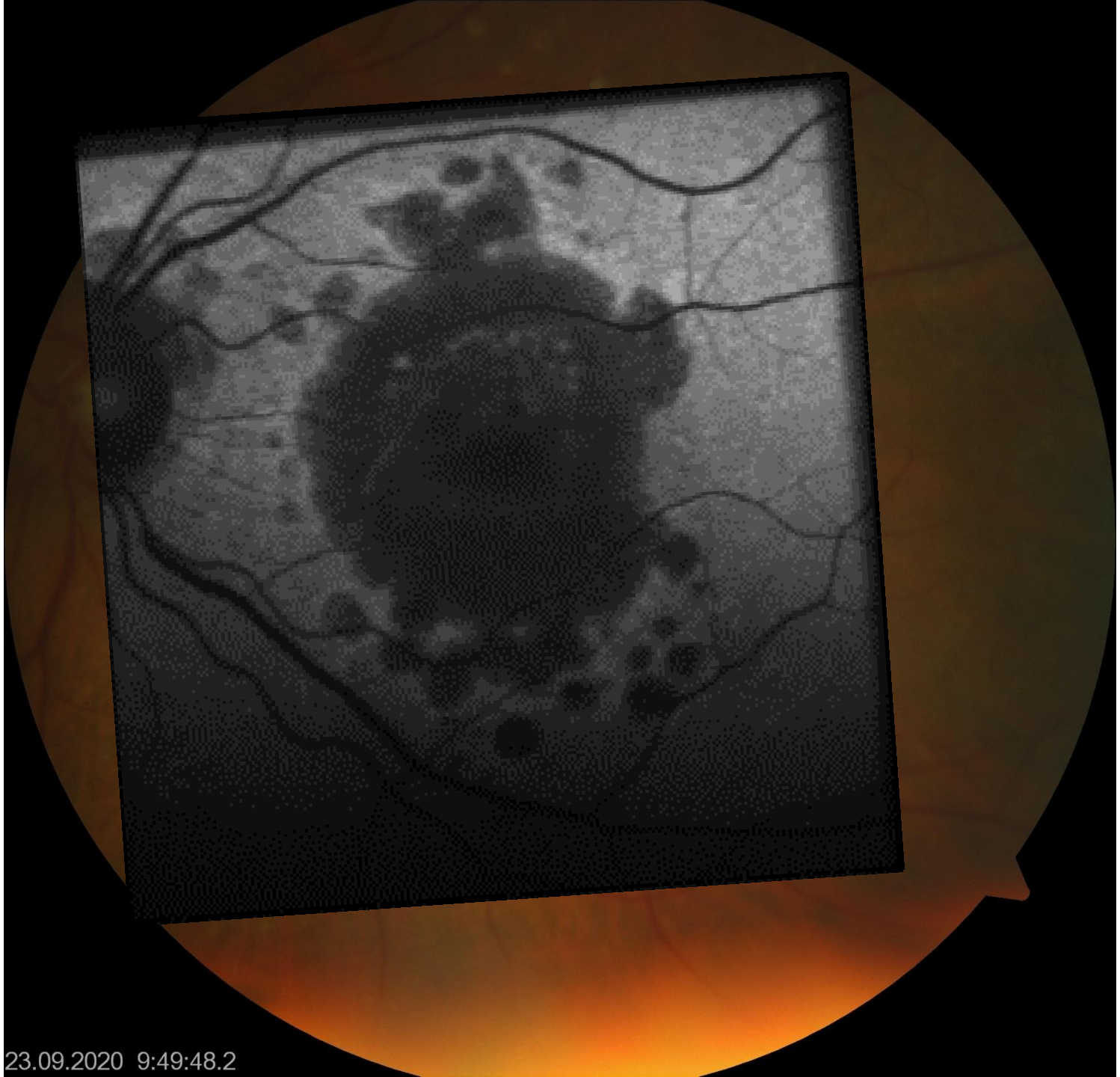
IR

09/23/2016



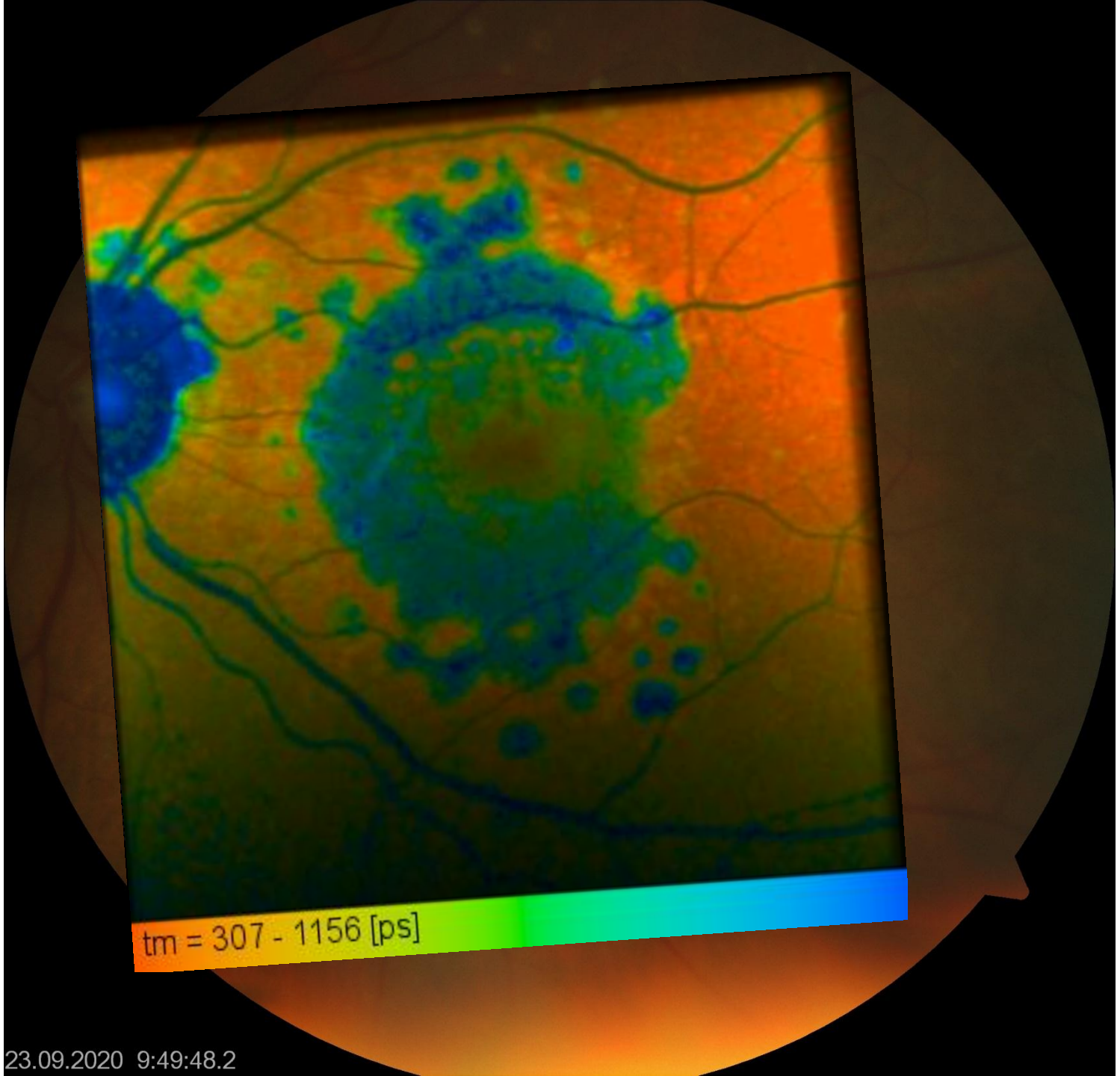
FAF SSC

09/23/2016



FAF LSC

09/23/2016



09/23/2016