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Optical Coherence Tomography (OCT) in the diagnosis and management of retinal abnormalities – wet age-related macular degeneration (AMD) – Part 1

About the author

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Chris Steele graduated from City University in 1988 and was a double prize winner in ocular disease and contact lens examinations. He qualified in July 1989 after his pre-registration year at the Royal East Sussex Hospital, Hastings. He is Consultant Optometrist, Head of Optometry at Sunderland Eye Infirmary (SEI). Over the past 28 years he has continued to develop a wide range of extended roles within his optometry team involving medical retina, cataract, glaucoma, anterior segment, emergency department and paediatric caseloads.

He has authored over 90 publications re: glaucoma, ocular therapeutics, medical retina, specialist medical contact lenses, refractive surgery and clinical risk management and has undertaken numerous presentations both nationally and internationally on these topics. He has authored two books, the first in the Eye Essentials series, Diabetes and the Eye, published by Elsevier in 2007 and Systemic and Ophthalmic Management of Diabetes Mellitus published by JayPee Medical Publishers in 2018.

Chris was a College examiner for pre-registration final exit examinations and postgraduate higher qualifications (diabetes and glaucoma) for many years. Chris was a member of the original NICE Glaucoma Guideline Development Group that produced the NICE glaucoma guidelines (CG85) published in 2009.

In the past 5 years he was a member of the College of Optometrists Medical Retina Development Group that produced the new Medical Retina Higher Qualifications for optometrists. Most recently he was involved in establishing the new Advanced Practice in Ophthalmology MSc Degree Apprenticeship at UCL and Moorfields, London. He continues in his role as a co-editor for Specsavers' CPD.

Outline

Optical Coherence Tomography (OCT) is now commonplace in many optometric practices and its use in the detection and ongoing management of a range of different retinal conditions is well established. This article, the first in a series of five looking at OCT in retinal conditions will explore its use in the diagnosis of AMD and the treatment options available for the condition. The article also explores the importance of OCT in the referral of AMD cases detected in optometric practice to hospital eye services.

Learning objectives

Domain: Clinical practice

Registrants will have up to date knowledge in the use of OCT for the diagnosis of Age-related Macular Degeneration (AMD) and the ongoing management and treatment of the condition (s.5).

Registrant will know how to conduct an OCT assessment, which treatment options are available and how to make appropriate referrals for patients with AMD (s.7).

Background

Many high street optometric practices now have OCT at their disposal, which has revolutionised the way optometrists in practice can diagnose and appropriately manage many ocular anterior and posterior segment conditions such as AMD (**Figure 1a - c**).

Figure 1a: Nidek Retina Scan Duo

Figure 1b: Topcon Triton swept
source OCT, Topcon Healthcare

Figure 1c Spectralis – Heidelberg Engineering

Age-related macular degeneration (AMD) is the term given to ageing changes without any other obvious cause that occur in the central area of the retina, the macula. AMD patients, in whom the structure and function of the macula begins to deteriorate are typically more than 50 years of age. A salient characteristic is the accumulation of extracellular deposits including sub-retinal drusenoid deposits, basal linear, and basal laminar deposits.[1] These eyes may eventually demonstrate neovascularisation or atrophy. Neovascular disease in the macula may result from many causes. In AMD, the neovascularisation may start in the outer retina, and therefore, the term choroidal neovascularisation is no longer considered appropriate for this class (see new classification below). Dry AMD is the most common form of macular degeneration and is the most common cause of visual impairment in the developed world. The Royal National Institute of Blind People (RNIB) reports that AMD is the most common cause of certification for vision impairment. Around 10% of patients with dry AMD will develop macular neovascularisation (MNV), which is the hallmark of wet AMD. Vascular endothelial growth factor (VEGF) drives the development of CNV, which may lead to:

- Bleeding under the retina
- Detachment or atrophy of the retinal pigment epithelium (RPE)
- Sub-retinal or sub-RPE fluid accumulation with associated vision loss

Various ways of classifying AMD have been proposed.[2] The NICE Guideline 82 re: AMD clearly separates its recommendations into those related to 'early' and 'late' disease (see **Table 1**).[3] Within 'late' disease, a distinction is drawn between disease that is:

- **Wet active** - neovascular lesions that may benefit from treatment
- **Wet inactive** - neovascular disease with irreversible structural damage
- **Dry** - non-neovascular disease, including geographic atrophy.
- **Late AMD (indeterminate)** – to include rarer sub-types

AMD Classification	Definition
Normal eyes	· No signs of age-related macular degeneration (AMD) · Small ('hard') drusen (less than 63 microns) only
Early AMD	Low risk of progression: · Medium drusen (63 microns or more and less than 125 microns) or · Pigmentary abnormalities Medium risk of progression: · Large drusen (125 microns or more) or · Reticular drusen or · Medium drusen with pigmentary abnormalities High risk of progression: · Large drusen (125 microns or more) with pigmentary abnormalities or · Reticular drusen with pigmentary abnormalities or · Vitelliform lesion without significant visual loss (best-corrected acuity better than 6/18) or · Atrophy smaller than 175 microns and not involving the fovea
Late AMD (indeterminate)	· Retinal pigment epithelial (RPE) degeneration and dysfunction (presence of degenerative AMD changes with subretinal or intraretinal fluid in the absence of neovascularisation) · Serous pigment epithelial detachment (PED) without neovascularisation
Late AMD (wet active)	· Classic choroidal neovascularisation (CNV) · Occult (fibrovascular PED and serous PED with neovascularisation) · Mixed (predominantly or minimally classic CNV with occult CNV) · Retinal angiomatous proliferation (RAP) · Polypoidal choroidal vasculopathy (PCV)
Late AMD (dry)	Geographic atrophy (in the absence of neovascular AMD) Significant visual loss (6/18 or worse) associated with: · Dense or confluent drusen or · Advanced pigmentary changes and/or atrophy or · Vitelliform lesion (e.g. Adult Best)
Late AMD (wet inactive)	· Fibrous scar · Sub-foveal atrophy or fibrosis secondary to an RPE tear · Atrophy (absence or thinning of RPE and/or retina) · Cystic degeneration (persistent intraretinal fluid or tubulations unresponsive to treatment) Note that eyes may still develop or have a recurrence of late AMD (wet active)

Table 1: Classifying age-related macular degeneration according to NICE Guideline 82 (2018)

This review will discuss the essential role of OCT in the diagnosis of wet AMD and how best to use this technology to make appropriate referrals. Most referrals to the HES are accurate and these patients go on to receive treatment for their conditions.

However, many ophthalmic referrals based mainly around incidental OCT findings are often unnecessary. In such cases, it may be more appropriate for these to be safely monitored in community practice, without referral. This however requires sound knowledge around OCT interpretation and making the appropriate inferences from other collective clinical findings.

The remaining four parts to this medical retina series will include discussion regarding other relatively common retinal abnormalities such as diabetic retinopathy and maculopathy, retinal vein occlusions, central serous chorioretinopathy (CSCR) and vitreo-retinal interface abnormalities (VRIAs) such as epi-retinal membranes (ERM), vitreo-macular traction (VMT) and macular holes, in addition to ARMD discussed here. Common optical coherence tomography (OCT) features for each condition will be discussed with guidance on when to refer and when to consider monitoring certain abnormalities in optometric practice. Following referral, likely further investigations and management options for each condition will be discussed.

Aims

Here in part 1 of this series the following topics will be reviewed:

- The principles of OCT and operational guidance
- Normal retinal anatomy according to OCT
- The benefits of OCT and how to appropriately interpret this clinical data avoiding errors, in order to help make effective medical retina referrals. This article will focus on wet/ dry age-related macular degeneration (AMD)
- Abnormal retinal features specific to AMD that may require referral
- Retinal abnormalities that may be monitored in community practice

Optical coherence tomography (OCT)

Optical coherence tomography (OCT) is a quick, non-invasive and reproducible imaging tool for retinal and in particular, macular lesions and has become an essential part of everyday ophthalmic practice. OCT is similar to ultrasound imaging, but instead of using sound, low coherence interferometry is employed to produce cross sectional images of the retina.

OCT captures optical scattering from the ocular tissue to discern spatial details of tissue microstructures. This is achieved by using infrared light from a super-luminescent diode that is divided into two parts: one of which is reflected from a reference mirror and the other is scattered from the ocular structures. The two reflected beams of light are made to produce interference patterns, to obtain the echo time delay and their amplitude information that makes up an A-Scan. A-Scans that are captured at adjacent retinal locations by a transverse scanning mechanism are combined to produce a 2-dimensional image.[4] A-scans are then repeated at multiple transverse locations and mapped to a grey- or false-colour scale, giving rise to two-dimensional cross-sectional images (termed B-scans). A colour palette is then applied to visualise false-colour B-scans. The colours in the palette are selected to mimic the colours seen in a retinal photograph. They can be adjusted to match a scale indicating the thickness of a particular retinal layer. On OCT false-colour B-scans, highly reflective tissue is usually seen as reddish-white in colour, while hypo-reflective tissue is blue-black in colour.

In original OCT systems, interference patterns generated were varied as a function of time. These devices were referred to as “time domain” OCT (e.g. Stratus OCT, Zeiss). There are now several different types and models of OCT machine available, each of which provides the opportunity to examine results using various images, charts and measurements. In most current OCT devices, the interference patterns generated are varied as a function of frequency, with such devices referred to as “spectral domain” OCT (e.g. Nidek Retina Scan Duo, 3D OCT, Topcon; Cirrus HD-OCT, Zeiss; Spectralis, Heidelberg – **Figure 1a - c**). This results in greatly increased image acquisition speeds.

The newest generation of OCT devices employ tuneable lasers (“swept source” OCT) to further increase image acquisition speed, and employ longer wavelengths of light (~1050nm) to allow improved or enhanced depth penetration (e.g. DRI OCT-1, Topcon Triton). Swept source OCTs allow visualisation of the choroidal thickness as well as the vitreoretinal interface in one scan which is useful for assessing patients with choroidal diseases and those with vitreoretinal interface disease.

Although each OCT machine takes equivalent measurements, the scans which are produced do look slightly different for each. All of the SD-OCT systems commercially available use slightly different scan protocols for macular scanning.

These include:

- Three-dimensional (3D) or volume scans that consist of several horizontal line scans composing a 6 mm × 6 mm, 7 mm × 7 mm or 12 mm × 9 mm rectangular boxes. A three-dimensional view of the image is generated which enables the implementation of advanced, complex analysis e.g. C-scans, topographic maps, cyst volume and gives a holistic view of the macula. These scans are generally centred over the fovea, but can be relocated elsewhere and used to build a 3D computerised tomographical image of the retina.
- 3D scan resolutions are generally lower than single line scan presentations in order to minimise capture time for the patient. Poor patient fixation or patient head movement can significantly reduce the quality of the image produced (**Figure 2**).
  Volume cans allow for a larger area of the retina to be scanned without changing patient fixation and is ideal when screening for anomalies, or to build up an overall impression of the para-foveal area, e.g. the extent of an area of AMD and any associated tomographical retinal changes.
- Radial scans (**Figure 3**) consist of 6–12 line scans arranged in equal angles (star pattern) with a common axis around a point of interest e.g. the fovea. These scan protocols may differ in length, density, or resolution depending on the OCT system used. The results can be viewed as a series of line scans and the thickness map. Radial scans therefore enable scanning a larger area of the macula than a single line scan, while maintaining a high resolution and relatively short patient scanning time. Radial scans however, result in greater gaps between the scan lines in the para-foveal area which may result in lesions being missed and decreased accuracy of thickness values as interpolation of data is required to 'fill the gaps' in between the scan lines. The results can be viewed as a series of line scans or as a thickness map.

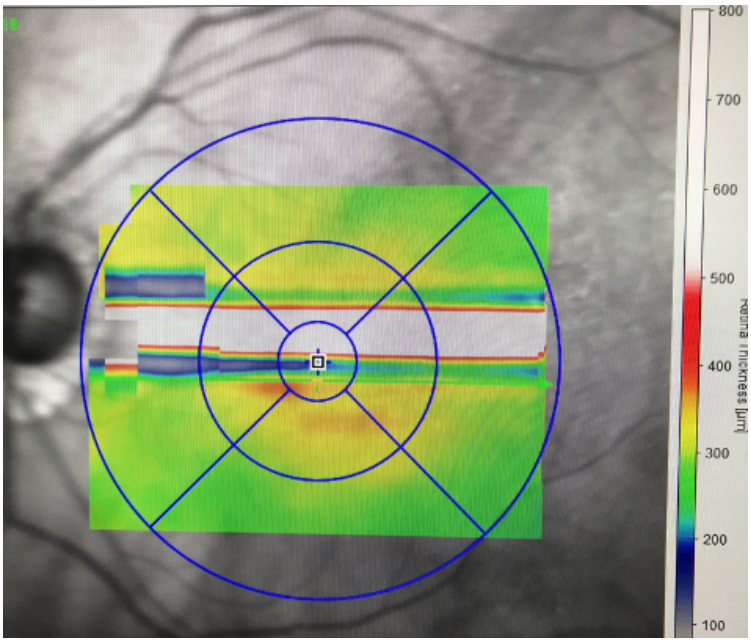

Figure 2: Heidelberg Engineering Spectralis OCT with white band on central retinal thickness map - due to excessive head movement.

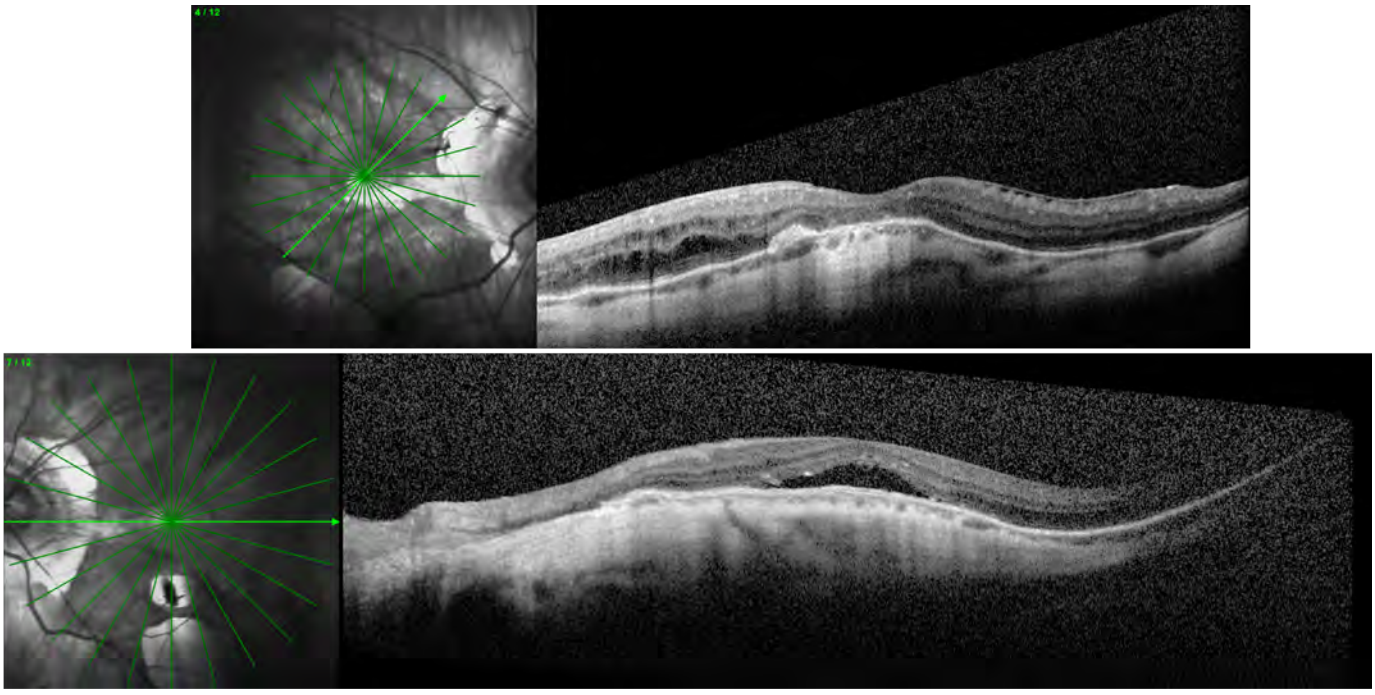

Figure 3: Radial scans consist of 6–12 line scans arranged in equal angles (star pattern) with a common axis around a point of interest e.g. the fovea

- Raster scans consist of a series of parallel line scans that can be oriented in any angle and are of higher resolution.
- Retinal thickness maps provide clinically very useful quantitative information around a specific point of interest such as the fovea. The map can be generated from a volume scan or a radial scan protocol and the information displayed as a colour map where the warmer colours represent increased retinal thickness. These displays can be useful in identifying the extent of diffuse thickening of the para-foveal area in some macular abnormalities e.g. diabetic maculopathy or retinal vein occlusions.

For optimal results a pupil diameter of 3 mm is usually required to obtain a good OCT image, but it is still possible with many systems to obtain adequate images with smaller pupil sizes if needed.

An appropriate scanning protocol is selected to scan the retinal area of interest and a live SD-OCT window is seen. The patient should be instructed to look at the internal fixation target at the centre (for macular scanning) or an external adjustable target where appropriate in e,g,optic nerve head imaging. It is important to ensure the fundus image on the monitor is focused, thus correcting for any refractive errors.

By moving the SD-OCT joy stick backwards or forwards, this will ensure the OCT image is upright and the reference scan pattern is centred on the fovea or the area of interest. Adjust the light entry point across the pupil to get the best signal strength. Repeat the procedure with other scan protocols if necessary. The saved OCT scans are analysed both qualitatively and quantitatively. The OCT view window consists of the analysed SD-OCT images with its corresponding reference fundus image showing the orientation of the OCT scan. The analysis report varies with different OCT systems. In general, all machines have a thickness map displaying the average retinal thickness values in 1 mm, 3 mm, and 6 mm diameter circles divided into sectors with a colour map. Warm colours indicate thicker retinal areas and cool colours indicate thinner retinal areas (**Figure 4**).

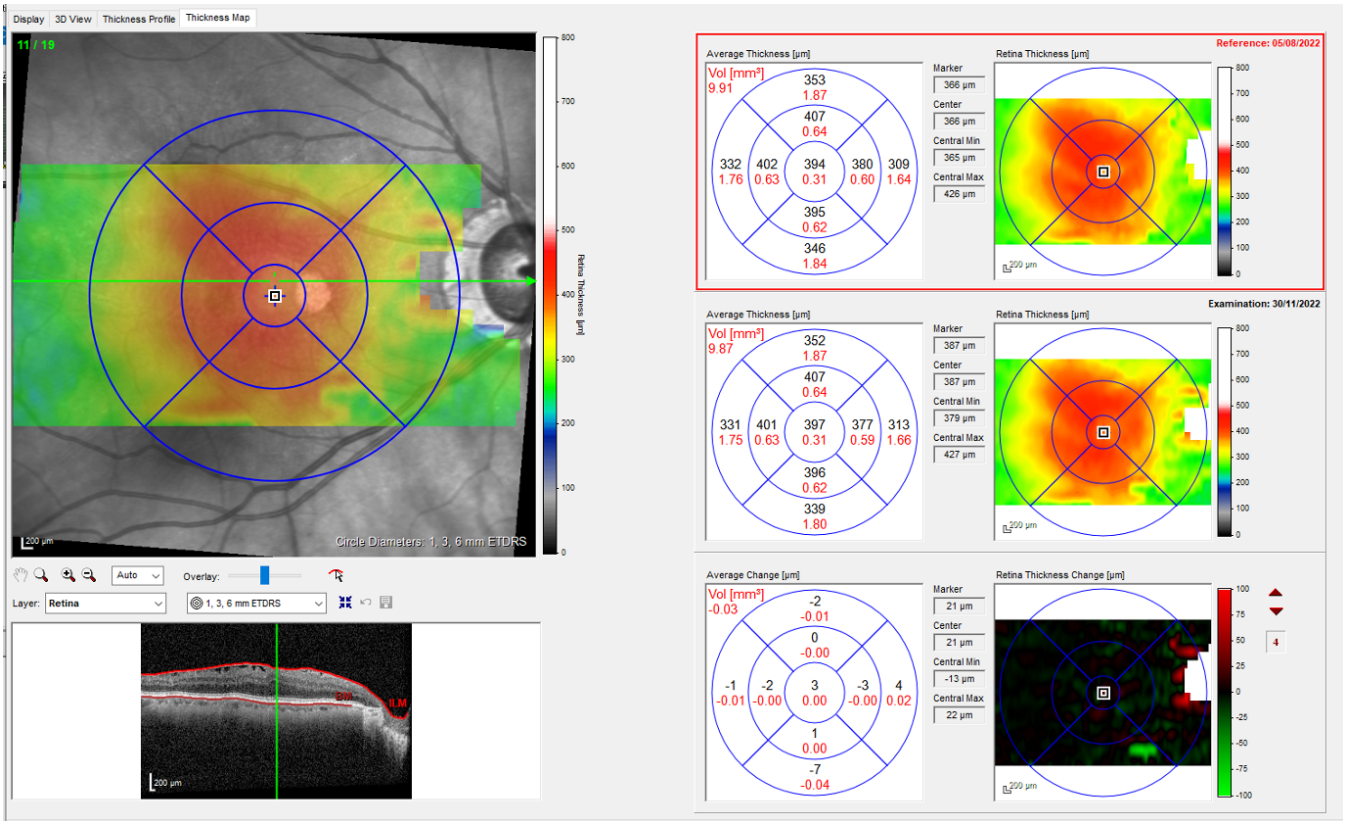

Figure 4: Heidelberg Engineering Spectralis OCT thickness map showing increased thickness (in red) with epiretinal membrane.

The essentials of an effective medical retina referral when incorporating OCT

There are two approaches to OCT interpretation:

Diagnosis – involves accurate pattern recognition based on previous experience of image interpretation and a sound knowledge of what is within normal limits and what is definitely abnormal. This will take many, many hours of exposure to OCT image interpretation in order to build up the necessary experience and expertise to undertake this competently. When unsure of the diagnosis however, it is important to be able to accurately describe an OCT image.

Descriptive – interpreting an OCT scan, by observing that something is not normal, and then being able to consider why it is not normal by describing anything abnormal on the scan. This is best undertaken using descriptive terms that will be understood by other clinicians and is a recommended approach when first getting to grips with OCT image interpretation.

Heidelberg Engineering recently suggested an "Eyeway Code" that aims to act as a method to evaluate scans, as well as providing the common language to discuss the images with other eye care professionals.

Taking inspiration from the Highway Code, with the triangular red signs indicating a 'hazard,' the Eyeway Code" features 10 hazard signs that can be used to describe shapes and patterns in the OCT scan[5] (**Figure 5**).

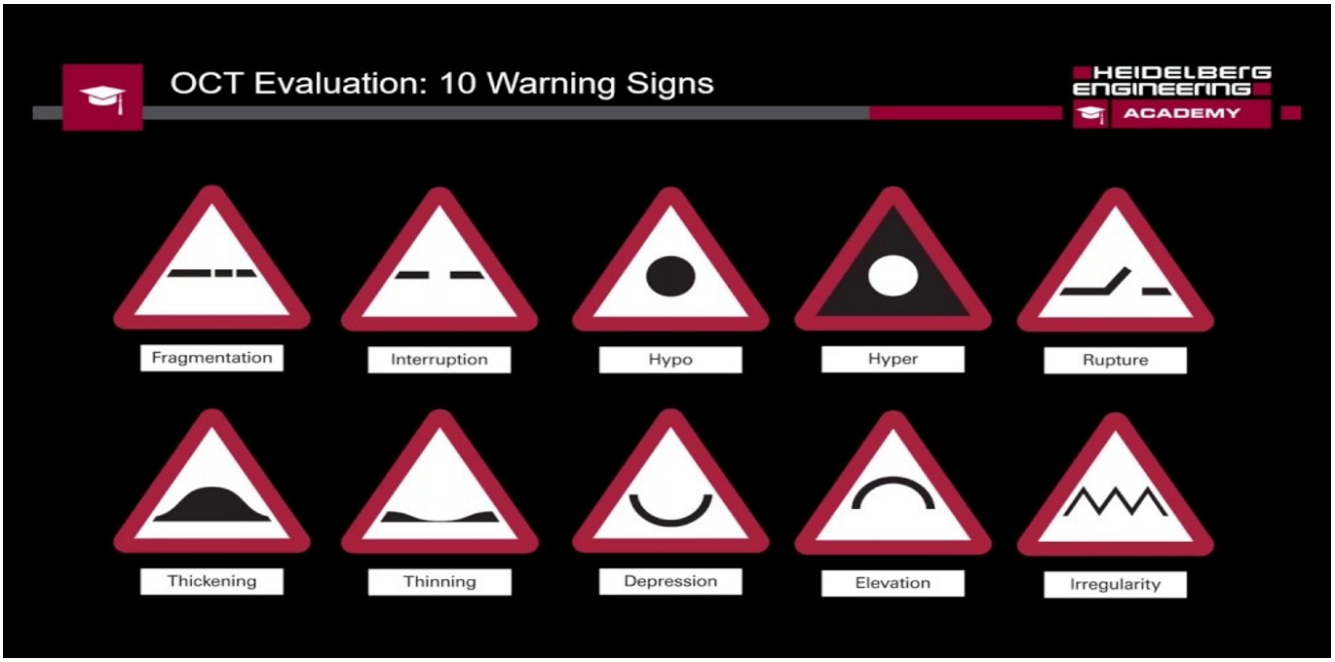

Figure 5: The Eyeway code By Tim Cole and Mark Holloway – Heidelberg Engineering

The signs include:

- Depression
- Elevation
- Fragmentation
- Interruption
- Irregularity
- Rupture
- Thinning
- Thickening
- Hyper-reflectivity
- Hypo-reflectivity.

Furthermore, using commonly understood medical terminology and abbreviations to describe the signs observed in OCT scans all help in improving quality and accuracy of referrals. In other words, even if a clinician is unsure of a diagnosis, being able to provide an accurate description of an OCT scan is essential. In order to accurately describe and interpret OCT images it is important to consider the following for each OCT acquisition:

- Ensure the image is appropriately centred with high enough resolution to provide sufficient image quality. A poorly centred scan may result in erroneous various statistical indices being calculated which may directly influence any decision to refer or perhaps even treat an eye. Retinal thickness for example, is a reproducible and common quantitative measurement that is used to monitor the disease process or treatment response using OCT. It is important to remember that the normative value for retinal thickness is dependent on which OCT machine is used, as well as the ethnic background of the individual being examined.
- Artefacts, particularly motion detection due to head/ eye movement during the scan acquisition, are still quite possible (despite inbuilt eye tracking software) (**Figure 2**). Where these occur, these may be eliminated by repeating scans or by changing to a different scan protocol. Other common artefacts are related to decentred scans and poor focussing prior to image acquisition. Software-related artefacts are mostly due to failed segmentation algorithms giving rise to inaccurate identification of the inner and outer retinal boundaries, as well incomplete segmentation of other layers in between (see below).
- Note the observed retinal profile and signs of normality such as foveal pit being present or absent, as well as e.g. a well-defined inner limiting membrane (ILM). All OCT instruments take the ILM as the inner retinal border. The outer retinal border may be taken as one of the three hyper-reflective outer retinal layers. For example, Spectralis and Cirrus high-definition OCT take the outer most layer, RPE; whereas others take the inner border of the second hyper-reflective line - the inter-digitation zone.
- Check each identifiable layer of the OCT image and carefully assess for signs of abnormality. Some OCT machines can delineate individual layers of the retina including the outer segments of the photoreceptors and retinal pigment epithelium (RPE).
- Beware that shadow artefacts caused by overlying blood vessels are inevitable. However, any other irregular shadows identified should be compared with the fundus reference image.
- When using radial or volume scans, check each line scan carefully for any abnormalities.
- Review all the available data outputs to gain more information, e.g. central retinal thickness (CRT).

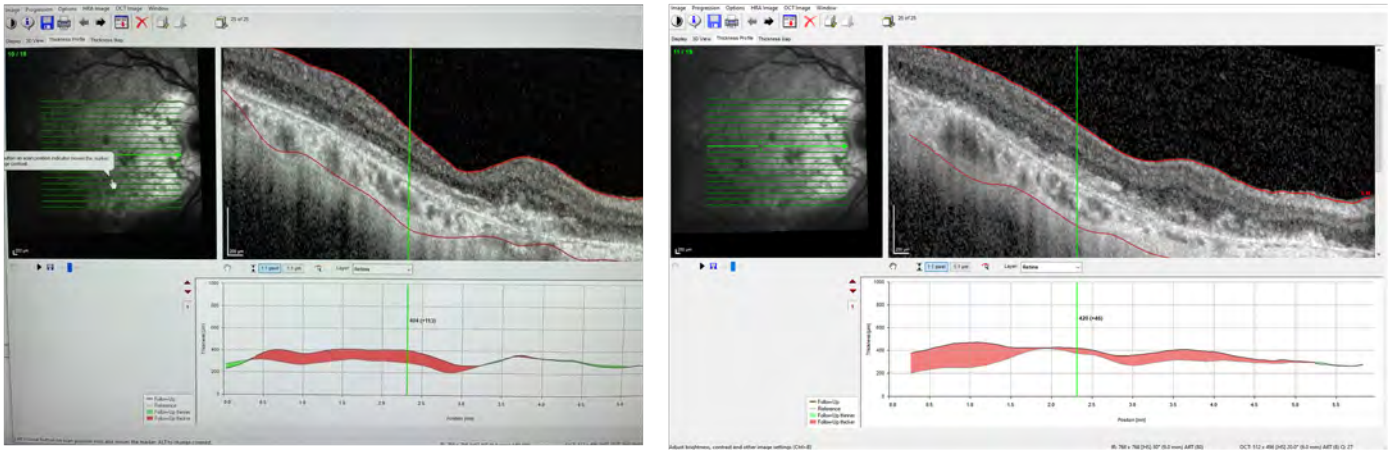

Figure 6a and **6b**: **Poor segmentation of OCT image – indicated by red lines. External limiting membrane (ELM) wrongly located in the choroid!**

- Always check the segmentation is correctly identified on the images, as every OCT system has an inbuilt segmentation algorithm that identifies the differences in reflectance of the retinal layers and borders (**Figure 6a** and **6b**). Lines are then added over the inner and outer borders of the retina (see above). This is really important to check to ensure e.g. accurate central retinal thickness measurement. Most OCT machines allow for manual correction of segmentation lines. This is particularly useful when analysing images involving e.g. age-related macular degeneration (AMD) and vitreo-macular traction (VMT).

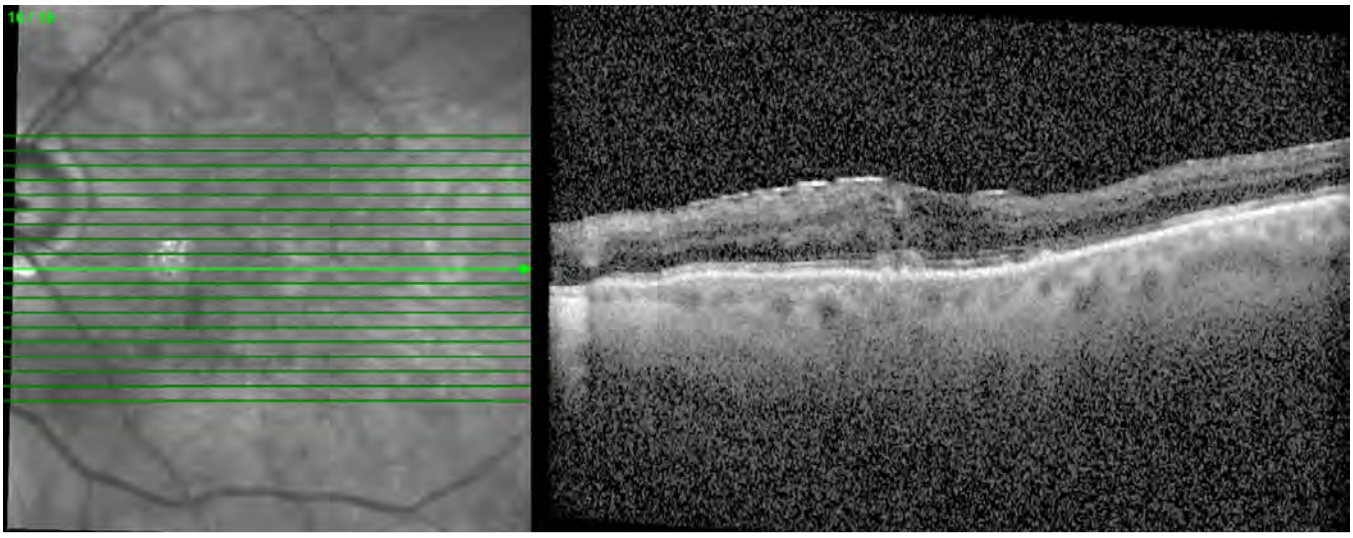

Figure 7a: "Noisy" is where the S:N ratio decreases, but details can still be evaluated.

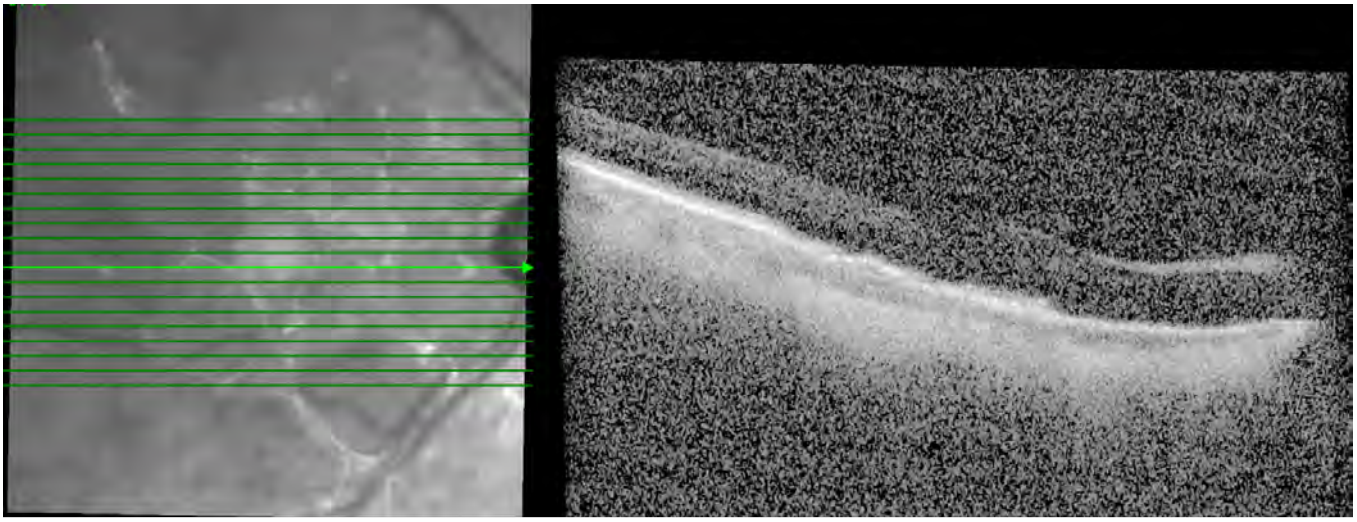

Figure 7b: Insufficient" is where no clear image is obtainable on OCT and it can only be assumed that there is any alteration in the area of interest.

- Note the scan quality by valuating the signal-to-noise (S:N) ratio which can be simply classed as either "good," "noisy", or "insufficient":
  - "Good" image quality means the scan offers a sufficient resolution of details with a high S:N ratio
  - "Noisy" is where the S:N ratio decreases, but details can still be evaluated (**Figure 7a**)
  - "Insufficient" is where no clear image is obtainable and it can only be assumed that there is any alteration in the area of interest (**Figure 7b**).

- The infra-red (IR) image offers an excellent overview of the extent of retinal alterations and gives very useful overview regarding any underlying disease. An alteration on the IR image is very likely to reveal a morphological change in the cross-sectional scan, and both images supplement each other well.
- In a healthy patient, the IR image has a smooth monotone appearance and an evident foveal depression. Soft gradients from light to dark as well as a lighter appearance of the peripapillary region can also occur in healthy individuals and can be considered normal (**Figure 8**).
  - Dark areas signify light absorption, resulting from any fluid or blood accumulation in the sub RPE, subretinal, intraretinal, or epiretinal space. Round and sharply demarcated hypo-reflective spots often represent microaneurysms (MA) blurred, flame-like alterations represent retinal haemorrhages.

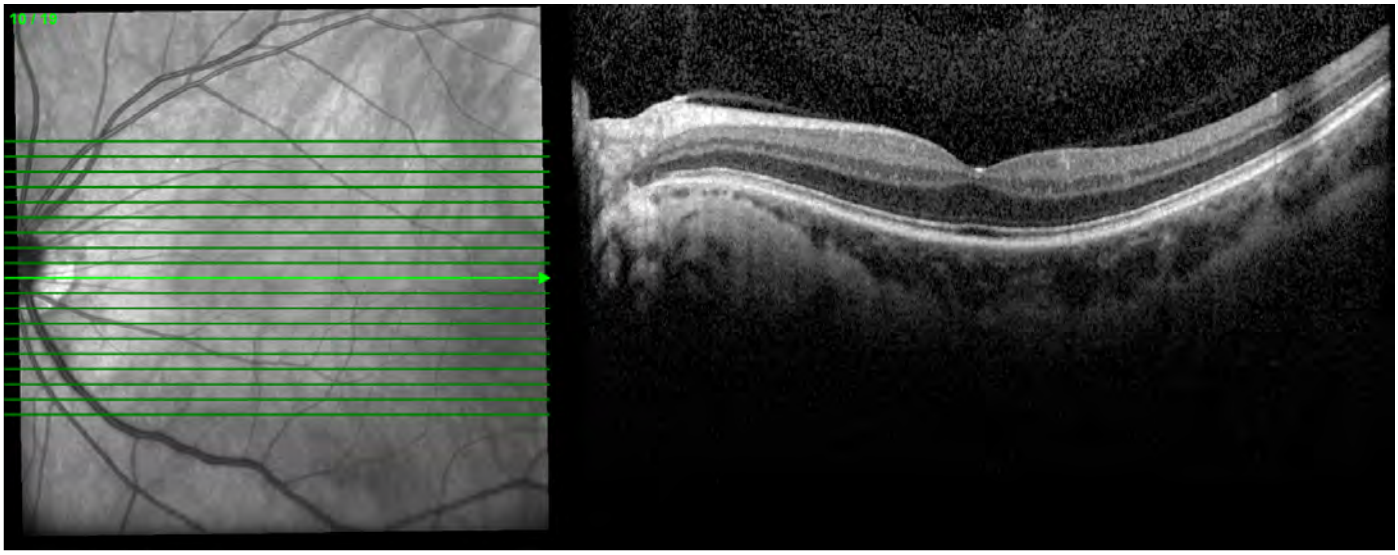

Figure 8: Normal infrared image (left) and Spectralis Heidelberg Engineering OCT – myope

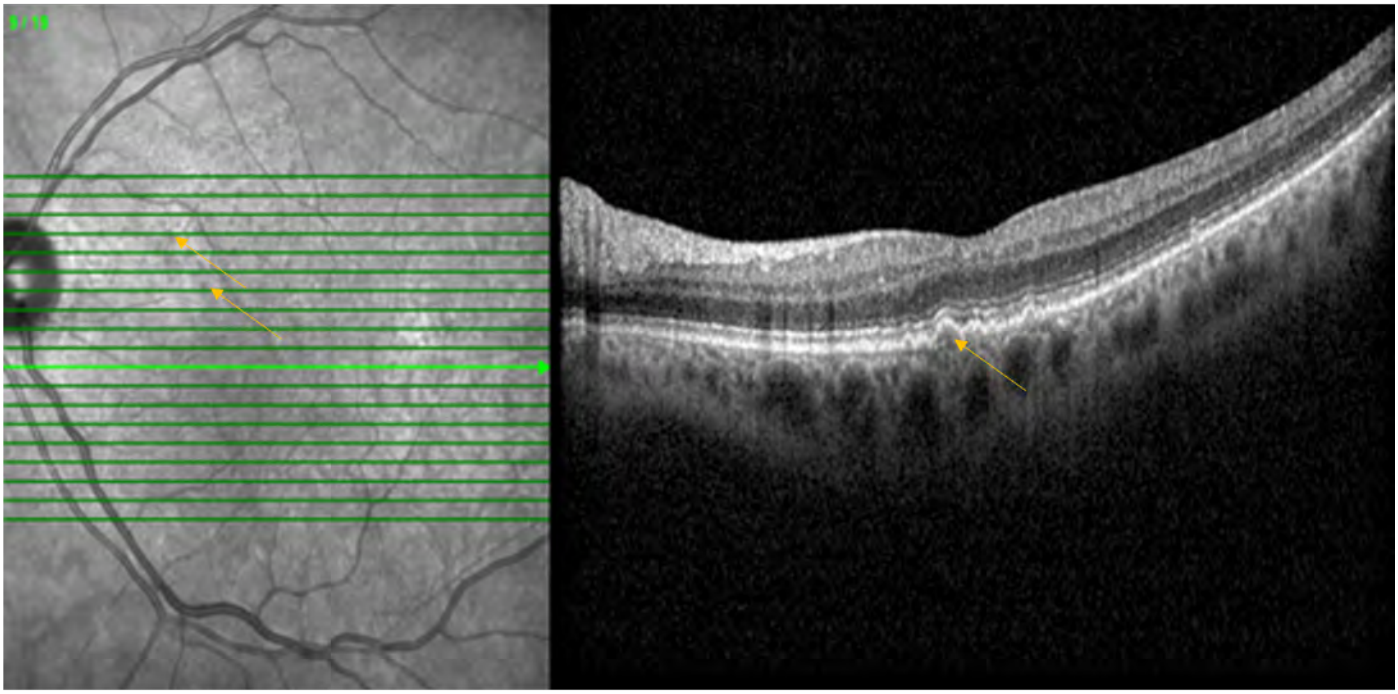

Figure 9: Drusen - Hyper- or hypo-reflective, especially in the macular region. Therefore any suspicion of drusen should be checked on the cross-sectional OCT scan.

- Bright spots are signs of increased reflectivity usually caused by e.g. exudates. Conversely, drusen or pigmentary alterations may present with a hyper-reflective appearance. Areas of RPE atrophy appear bright, often with a visualisation of the underlying choroidal vessels.
- The appearance of drusen (**Figure 9**) may be either hyper- or hypo-reflective, especially in the macular region. Therefore any suspicion of drusen should be checked on the cross-sectional OCT scan. The IR image has shown to be particularly helpful for detecting sub-retinal drusenoid deposits (SDD) also called reticular pseudo-drusen (see later section) (**Figure 10a - g**). They often appear as a network of medium to hyper-reflective alterations with a hypo-reflective halo.

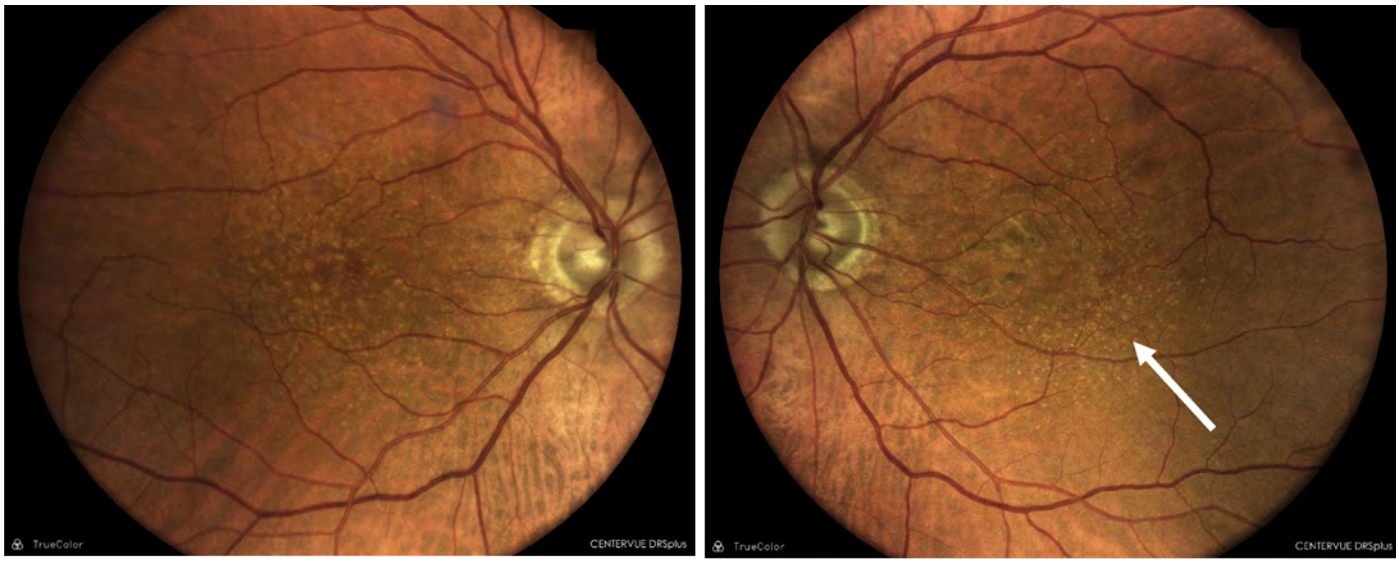

Figure 10a: Sub-retinal drusenoid deposits (SDD) or reticular pseudo-drusen

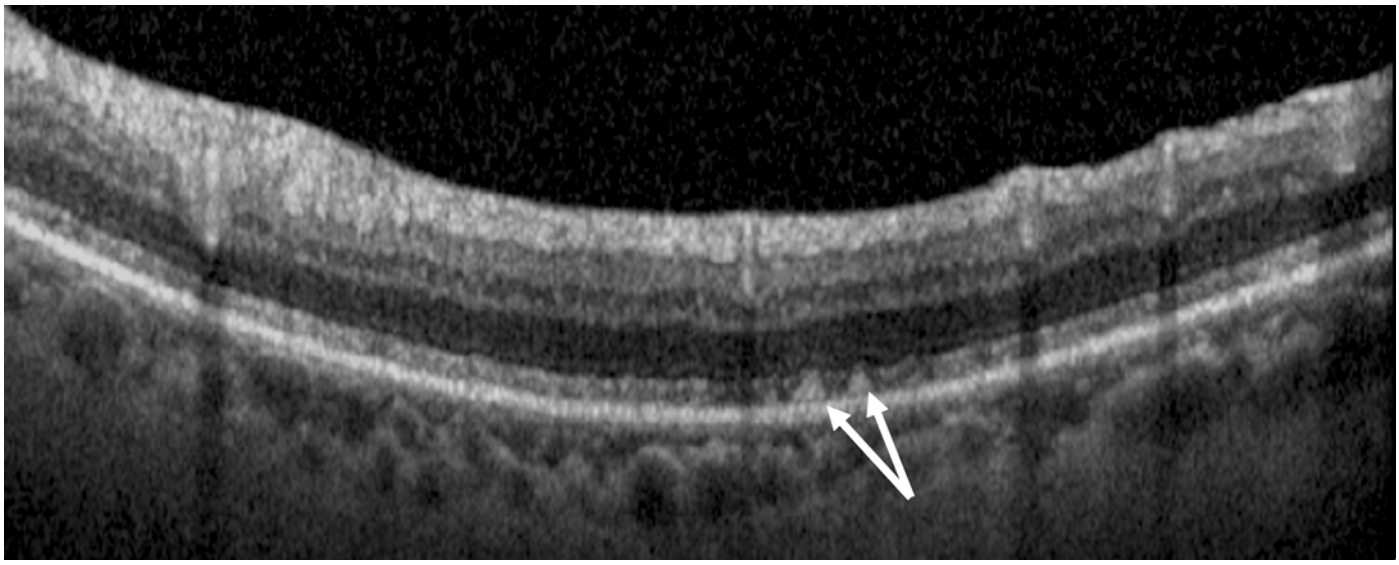

Figure 10b: Sub-retinal drusenoid deposits (SDD) or reticular pseudo-drusen – with permission Heidelberg Engineering

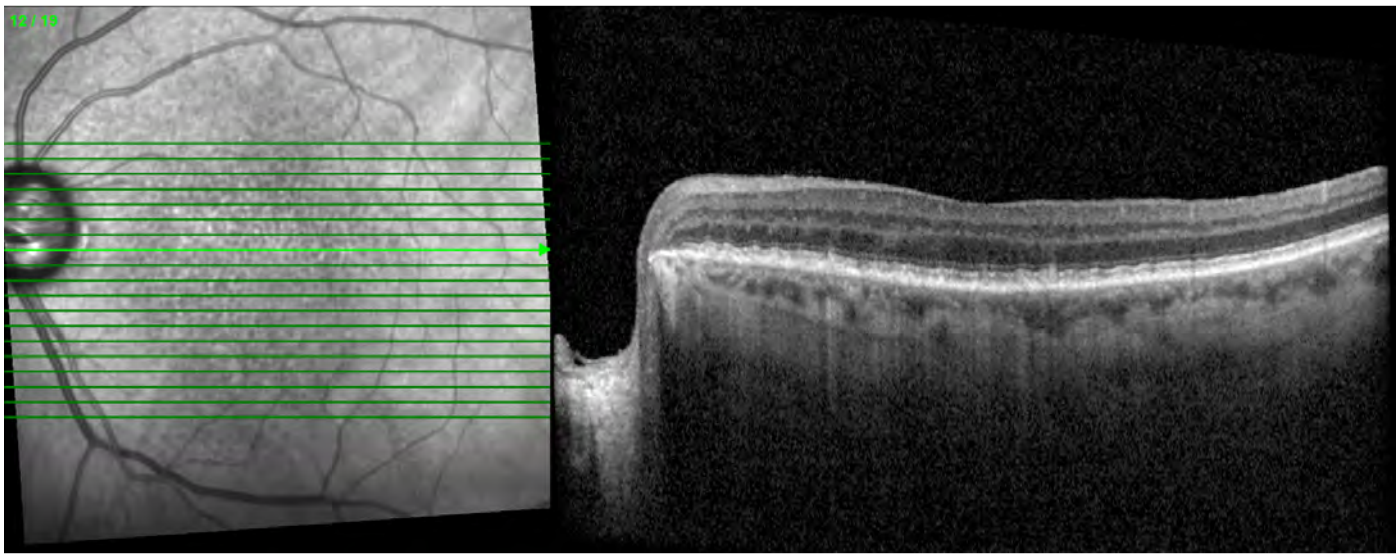

Figure 10c: Sub-retinal drusenoid deposits (SDD) or reticular pseudo-drusen

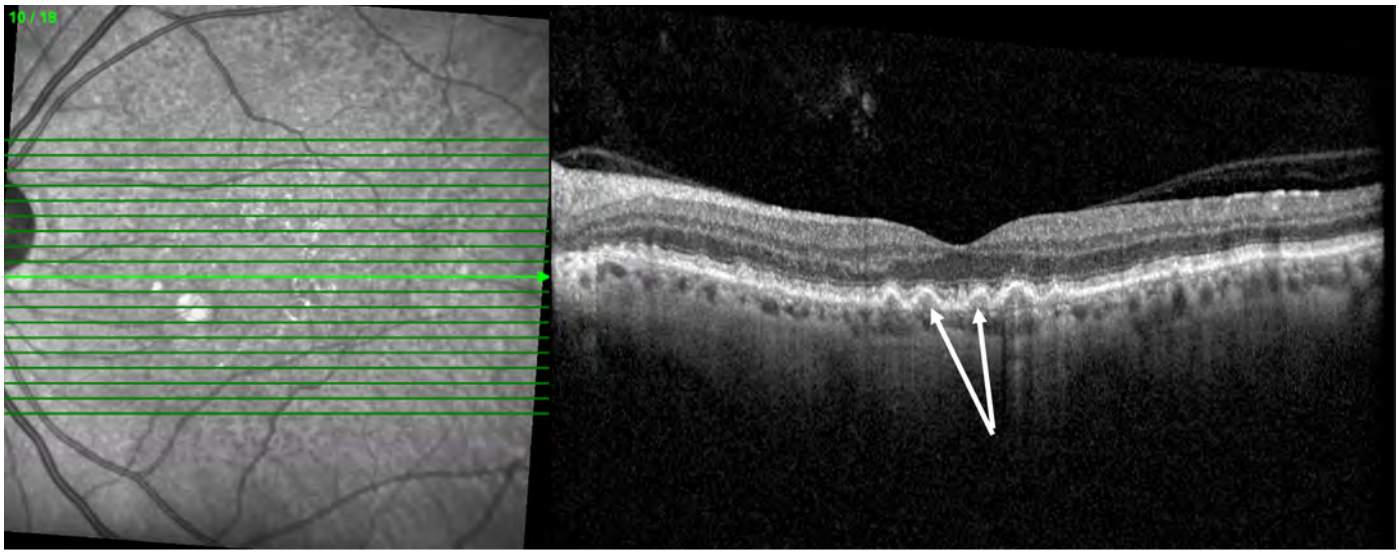

Figure 10d: Cuticular drusen

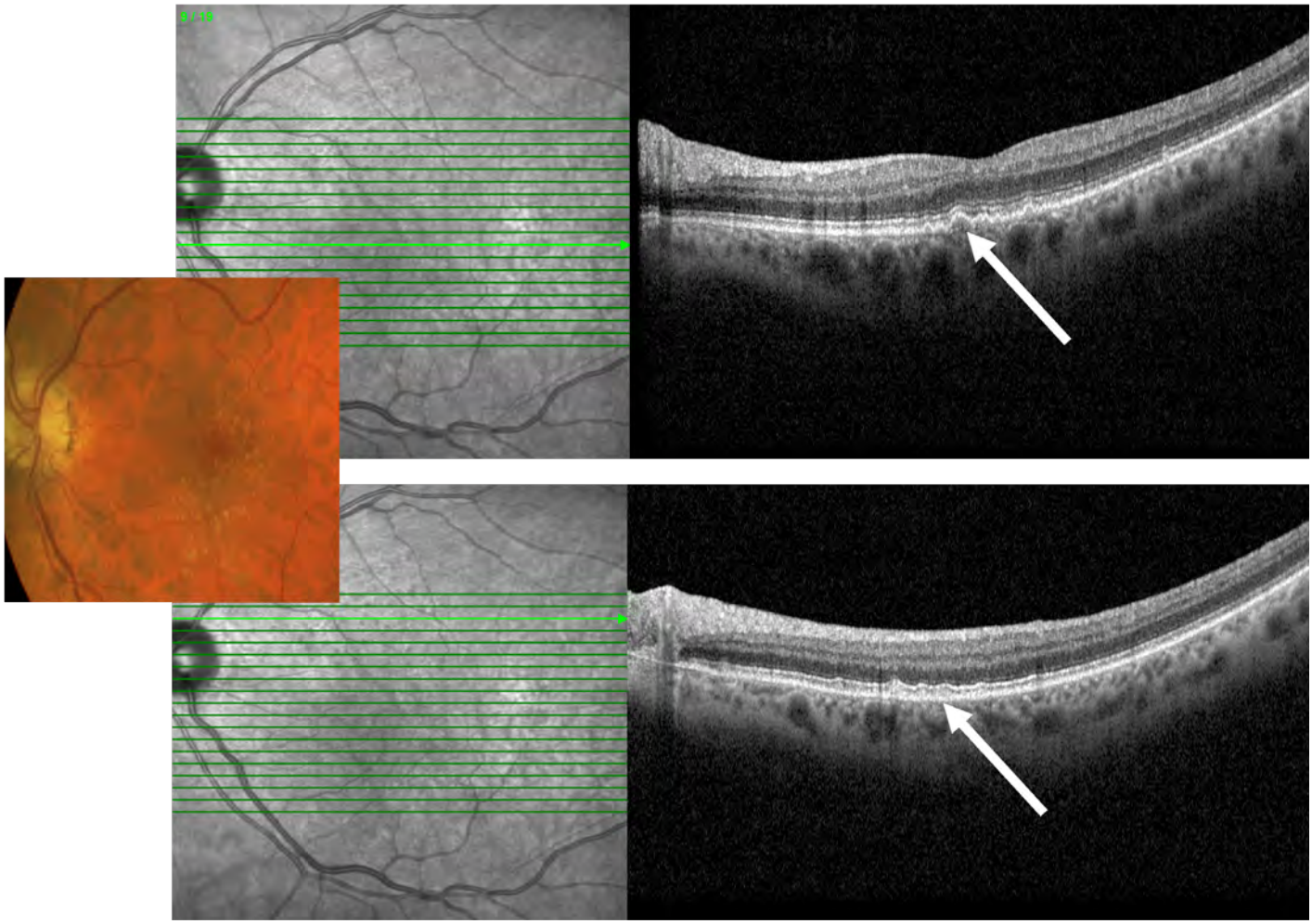

Figure 10e: Medium drusen

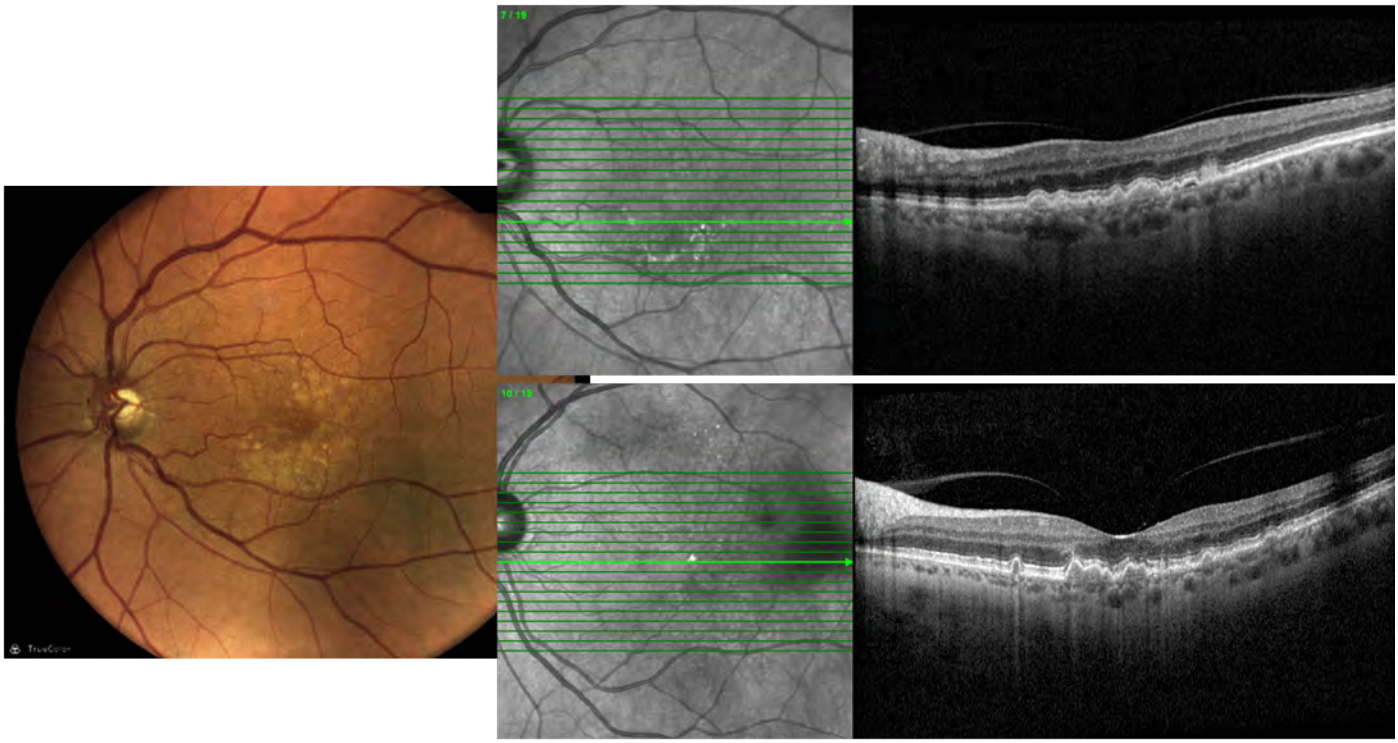

Figure 10f: Examples of large drusen

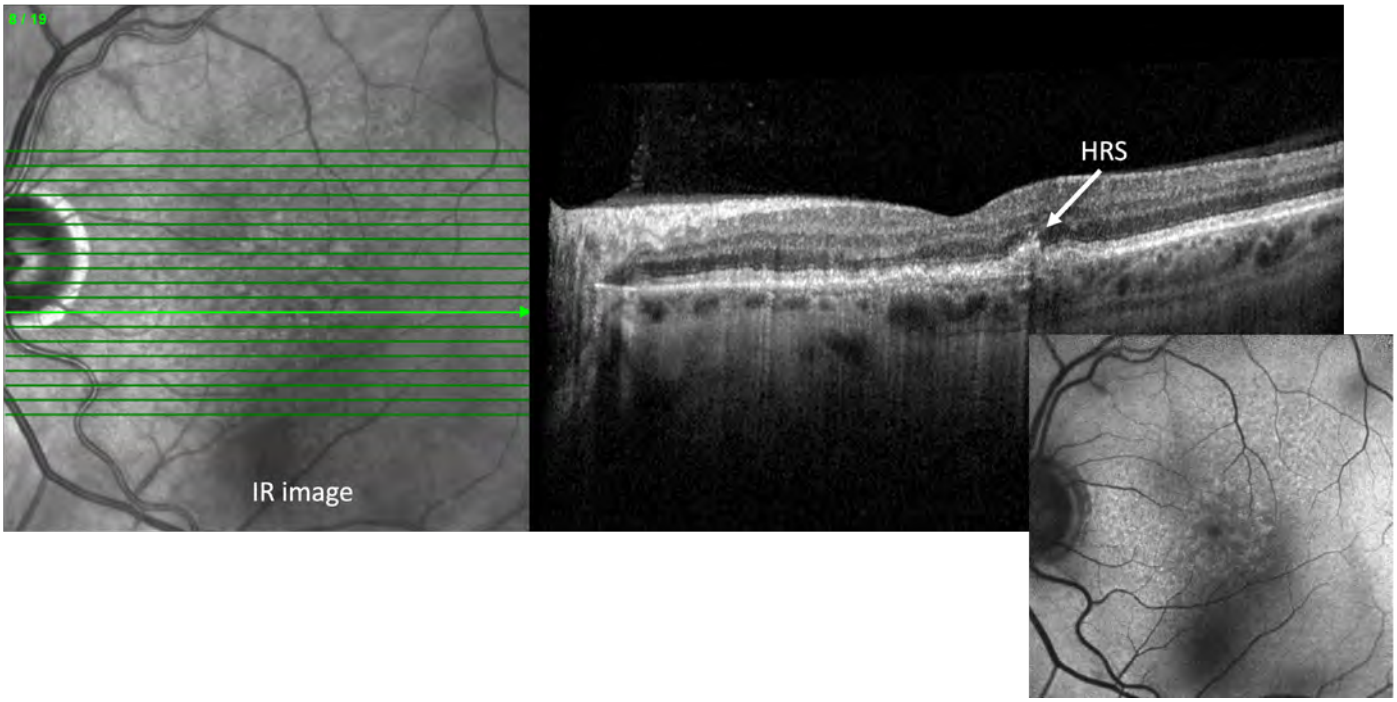

Figure 10g: Drusen with overlying hyper-reflective spots

The normal retina according optical coherence tomography (OCT)

In 2014 the International Nomenclature for OCT Panel provided a classification of anatomical landmarks identifiable on SD-OCT in the normal macula[6] (**Figures 11** and **12**). This panel consisted of an international panel with expertise in retinal imaging.

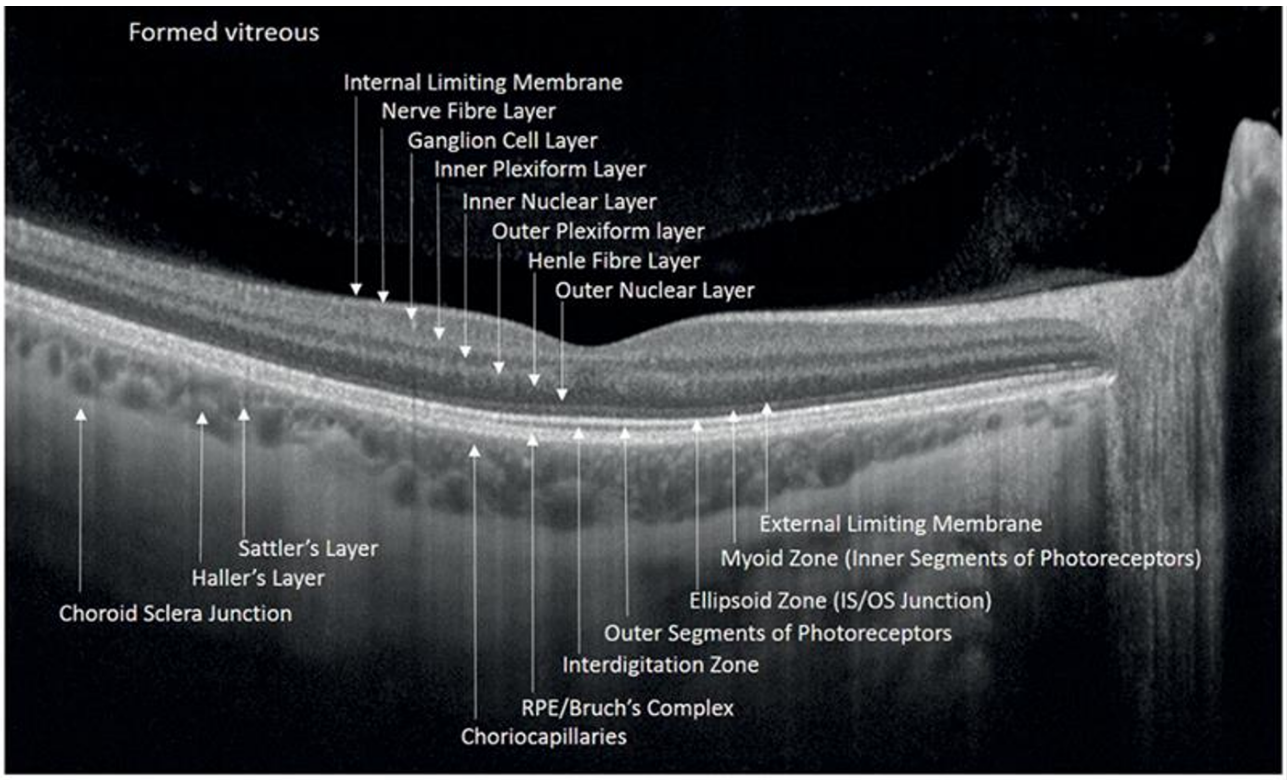

Figure 11: Normal retinal OCT image – with permission Heidelberg Engineering

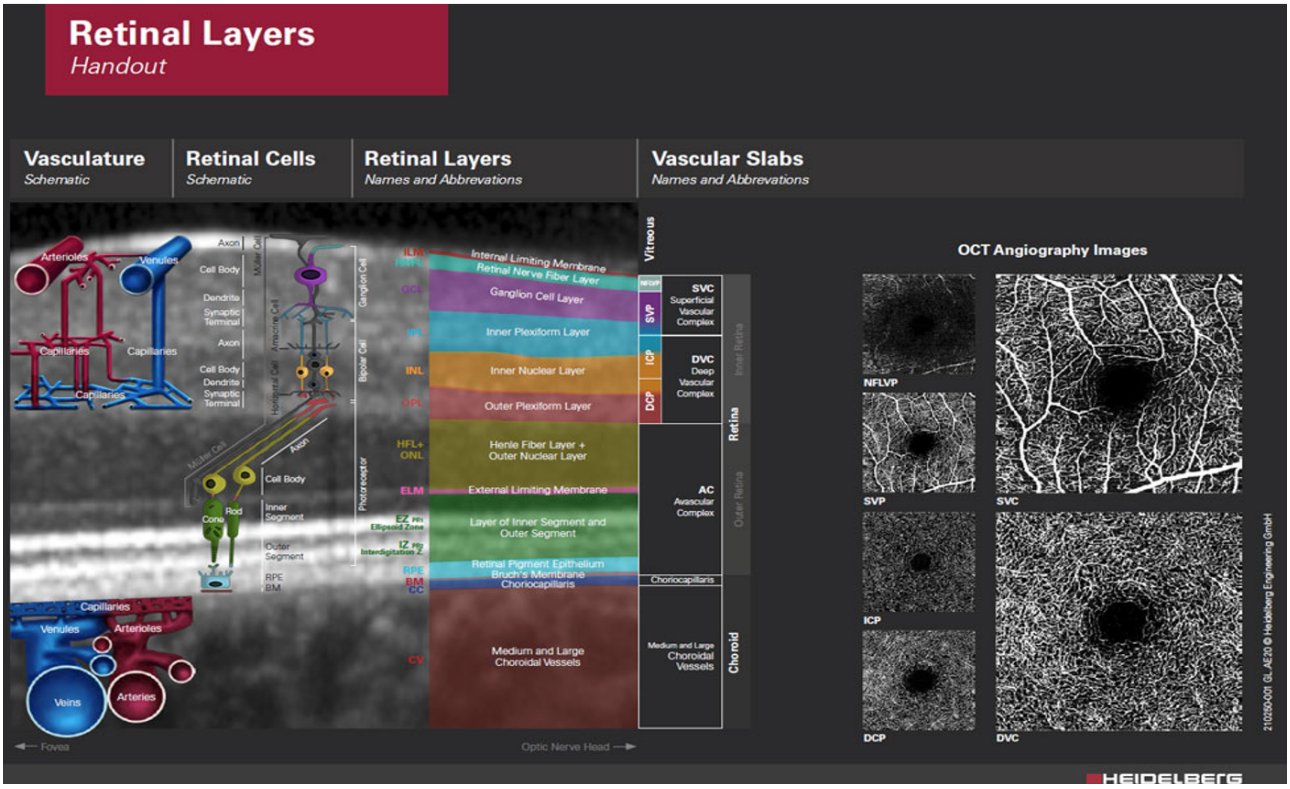

Figure 12: Retinal layers – with permission Heidelberg Engineering

In summary, according to this agreed international classification, the retinal and choroidal layers are divided into (see **Table 2**):

- A number of zones with specific reflective structures, which may be either hyper-reflective or hypo-reflective. These are not clearly defined by distinct margins and they are not associated with any specific histologic evidence.
- Discrete bands or layers that are well defined that correspond to proven histological evidence. These may also be hyper or hypo-reflective in appearance. One of these layers demonstrates variable reflectivity and this is the choroido-scleral junction.

Hyper-reflective Bands	Hyper-reflective Zones	Hypo-reflective Bands	Hyper-reflective Zones
Posterior cortical vitreous	Ellipsoid zones		Myoid zone
Nerve fibre layer (NFL)	Inter-digitation zone	Ganglion cell layer	Choriocapillaris
Inner-plexifom layer (IPL)		Inner nuclear layer	Inner and outer choroidal layers
Outer-plexiform layer (OPL)		Henle's nerve fibre layer and outer nuclear layer	
External limiting membrane (ELM)		Outer segments of photo-receptors	
RPE/Bruch's membrane complex			

Table 2: Normal anatomical retinal structure based on OCT and varying reflectivity

In the early phases of AMD, vision may remain relatively unaffected despite visible changes on clinical examination. Over time, vitelliform deposits may develop, pigment may migrate into the retina, drusen size may increase, and hypo-pigmentation and hyper-pigmentation of the RPE may develop. Late phases of the disease include atrophy of the outer retina, thinning and loss of the RPE, and neovascularisation (MNV) may develop (see below). Various common retinal abnormalities are identifiable by recognisable, but abnormal OCT reflectivity. Some of these are summarised in **Table 3**.

Reduced reflectivity	Increased reflectivity
Sub-retinal fluid (SRF)	Hard exudates
Sub-RPE fluid	Hyper-reflective foci (spots)
Intra-retinal fluid (IRF – retinal oedema)	Calcification
	Haemorrhages
	Sub-hyaloid reflective material (SHRM)
	Fibrosis
	Epi-retinal membranes (ERM)
	Vitreous membranes
	Choroidal neovascularisation (CNV)
	Retinal pigment epithelial (RPE) hyperplasia
	RPE atrophy resulting in choroidal hyper-reflectivity

Table 3: Reflectivity of common pathological changes seen on OCT

Optical coherence tomography (OCT) and neovascular (wet) macular degeneration (nAMD)

Traditionally, wet AMD or nAMD was considered as choroidal neovascularisation (CNV) and was divided into occult (type 1) and classical (type 2). Type 1 CNV referred to neovascular vessels confined to the sub-RPE space, and Type 2 referred to vessels proliferating above the RPE in the sub-retinal space. Recently, this classification has been renamed as macular neovascularisation (MNV) and classified into type 1 MNV, type 2 MNV, and type 3 MNV for retinal angiomatous proliferation (RAP).[7] This new classification is based on the location of the neovascularisation determined by OCT and OCT angiography (OCTA) imaging. Although MNV is usually viewed as an abnormal or pathological change, there is some evidence to suggest it is also an ocular protective response by the choriocapillaris to regenerate new blood vessels in order to improved oxygen and nutrient supply to the retina.[8]

Type 1 MNV (previously termed Occult CNV)

This includes ingrowth of vessels initially from the choriocapillaris into and within the sub-RPE space giving rise to varying types of PEDs. These neovascular complexes arising from the choroid may be imaged with OCT as an elevation of the RPE by material with heterogeneous reflectivity and where vascular elements may be seen. OCTA shows vessels below the level of the RPE. Accompanying the ingrowth is a varying amount of additional cellular elements including fibroblasts, myofibroblasts, and macrophages that seem to participate in the disease process and can lead to formation of fibrotic tissue. Leakage from vessels, haemorrhage, or associated proliferation of fibrotic tissue can cause expansion of a fibrovascular pigment epithelial detachment (PED).

On OCT, it presents as a heterogeneous, hypo- to medium reflective elevation of the RPE (representing fibrovascular material) (**Figures 13** and **14**). If leakage is substantial, it might present as, or be accompanied by, a serous PED. With fluorescein angiography it is described as "occult" or poorly defined MNV.[9] "Occult" (type I) MNV have two patterns:

- Fibrovascular PEDs as seen on OCT
- Leakage from an undetermined source, characterised by an ill-defined focus of choroidal leakage without identifiable classic CNV or fibrovascular PED.

In indocyanine green angiography it presents as a low-intensity hyper-fluorescence.

Type 1 MNV usually presents a fairly mature neovascular tissue that may incompletely respond to anti-VEGF therapy.

This is the most common type seen in dry AMD and other conditions with diffuse RPE/Bruch's membrane abnormalities such as cuticular drusen, familial dominant drusen (also known as Doyne honeycomb retinal dystrophy or Malattia Leventinese), and polypoidal choroidal vasculopathy (PCV).

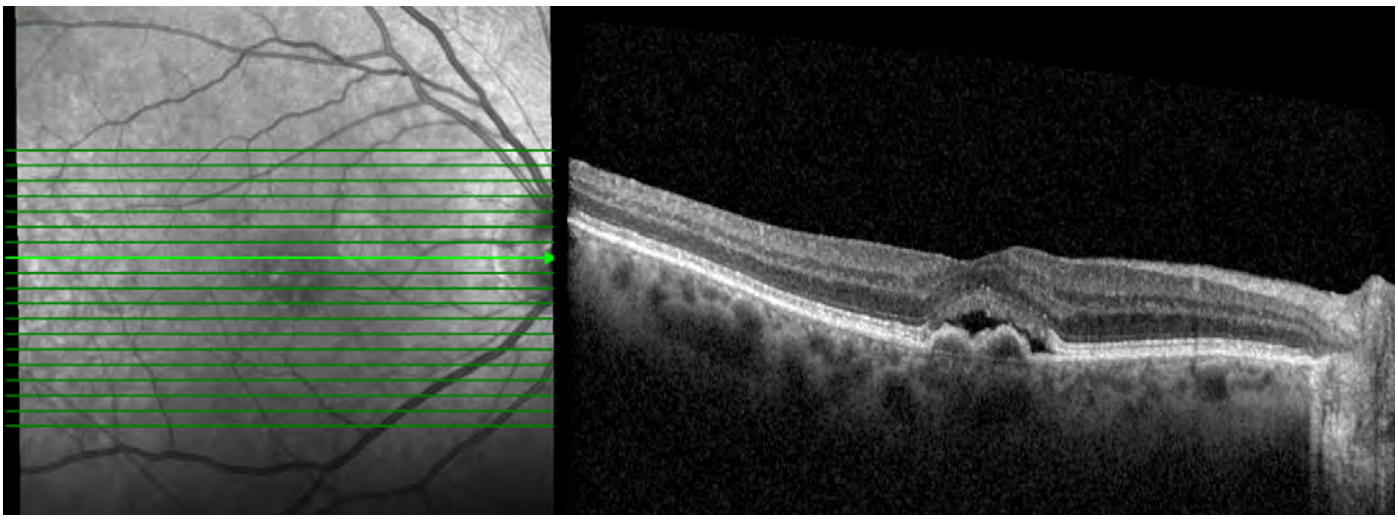

Figure 13: Type 1 MNV – small PEDs with medium reflective content and overlying SRF.

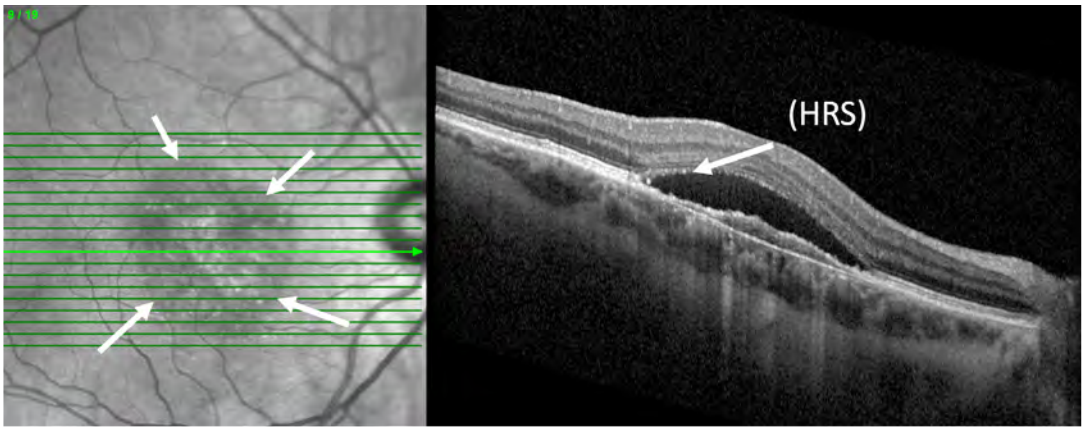

Figure 14a: RE Type 1 MNV – shallow PED with hypo – medium reflective elevation of RPE and overlying sub-retinal fluid (SRF). Note hyper-reflective spots (HRS) along border of SRF – a sign of activity

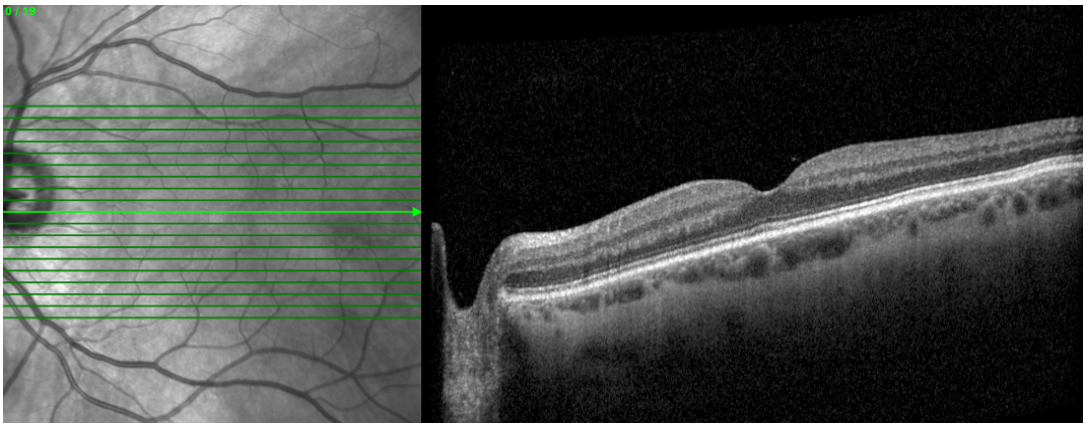

Figure 14b: Normal fellow LE eye

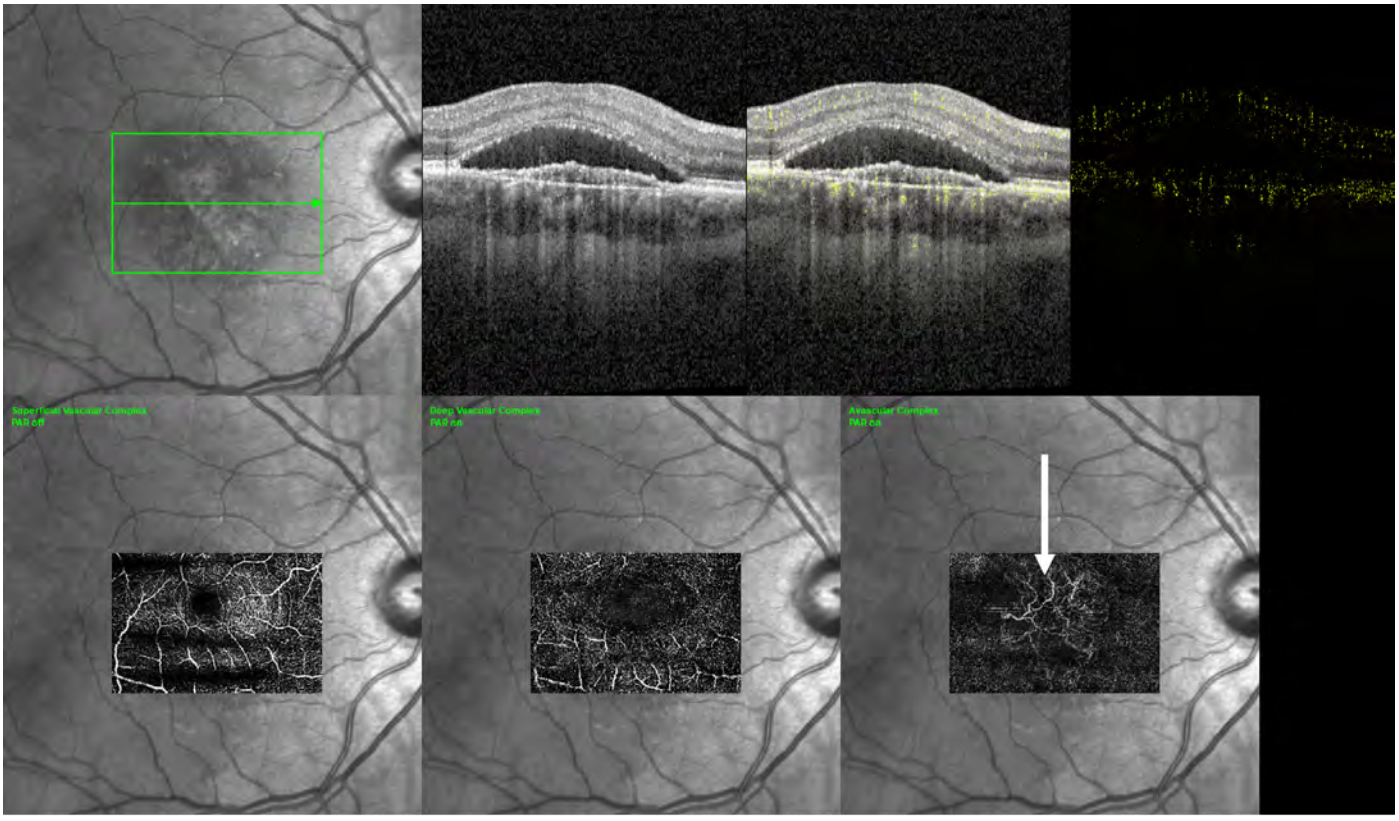

Figure 14c: Type 1 MNV - Shallow PED & SRF. Optical coherence tomography angiography (OCTA) – CNVM present in avascular zone

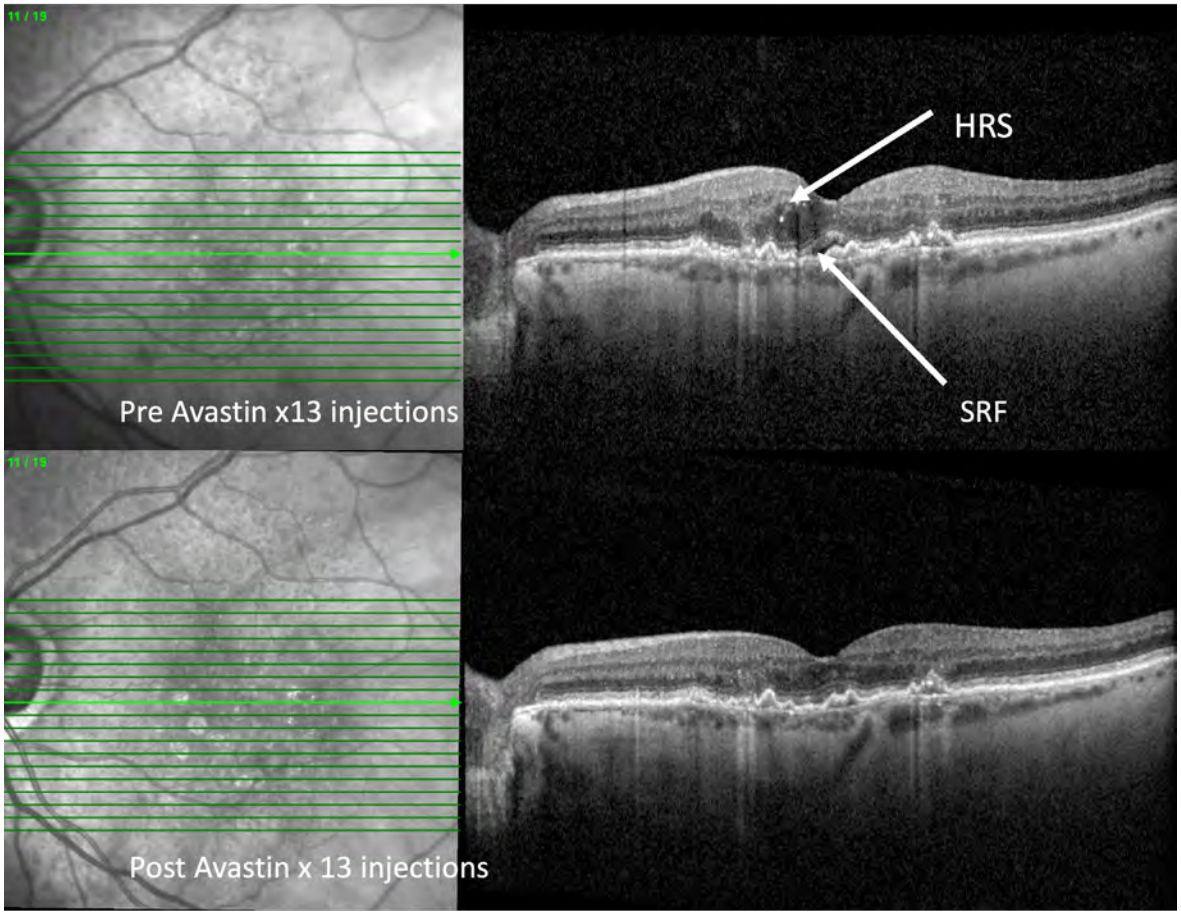

Figure 14d: RType 1 MNV LE. Pre and post Avastin x13 injections (note hyper-refective spots (foci))

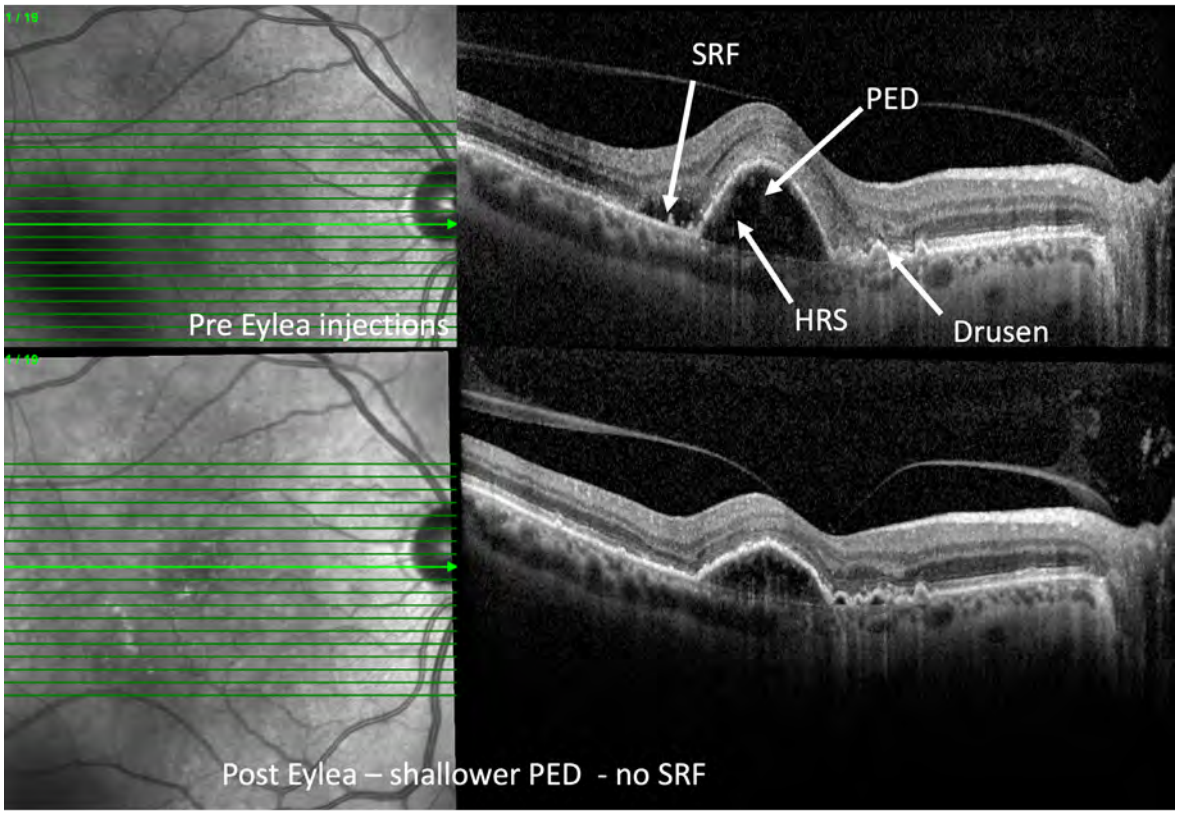

Figure 14e: Type 1 MNV pre and post Anti-VEGF intravitreal Eyea x 3 injections

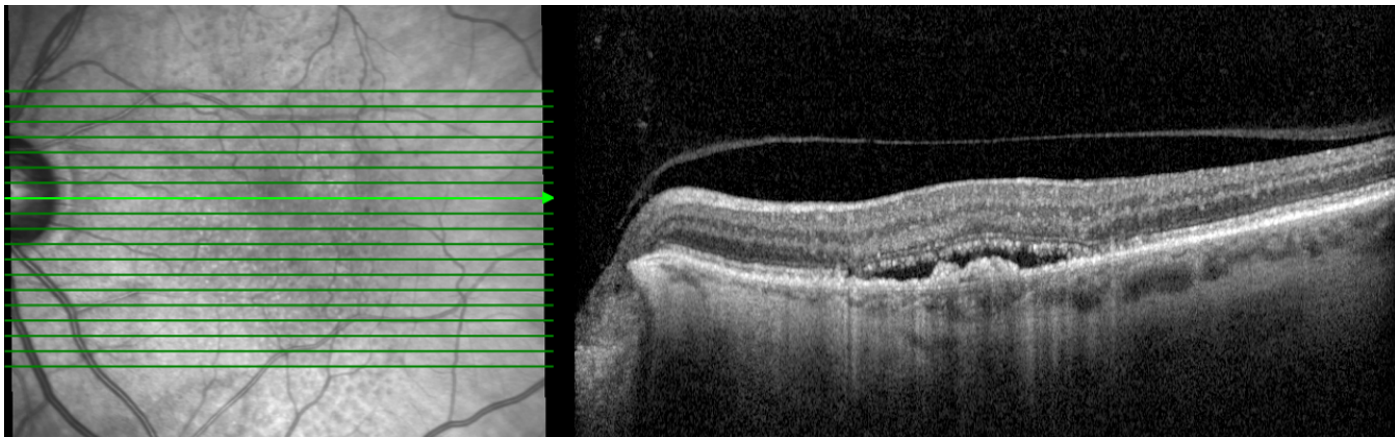

Figure 15a: Type 2 MNV well-defined hyper-reflective tissue above the RPE on OCT

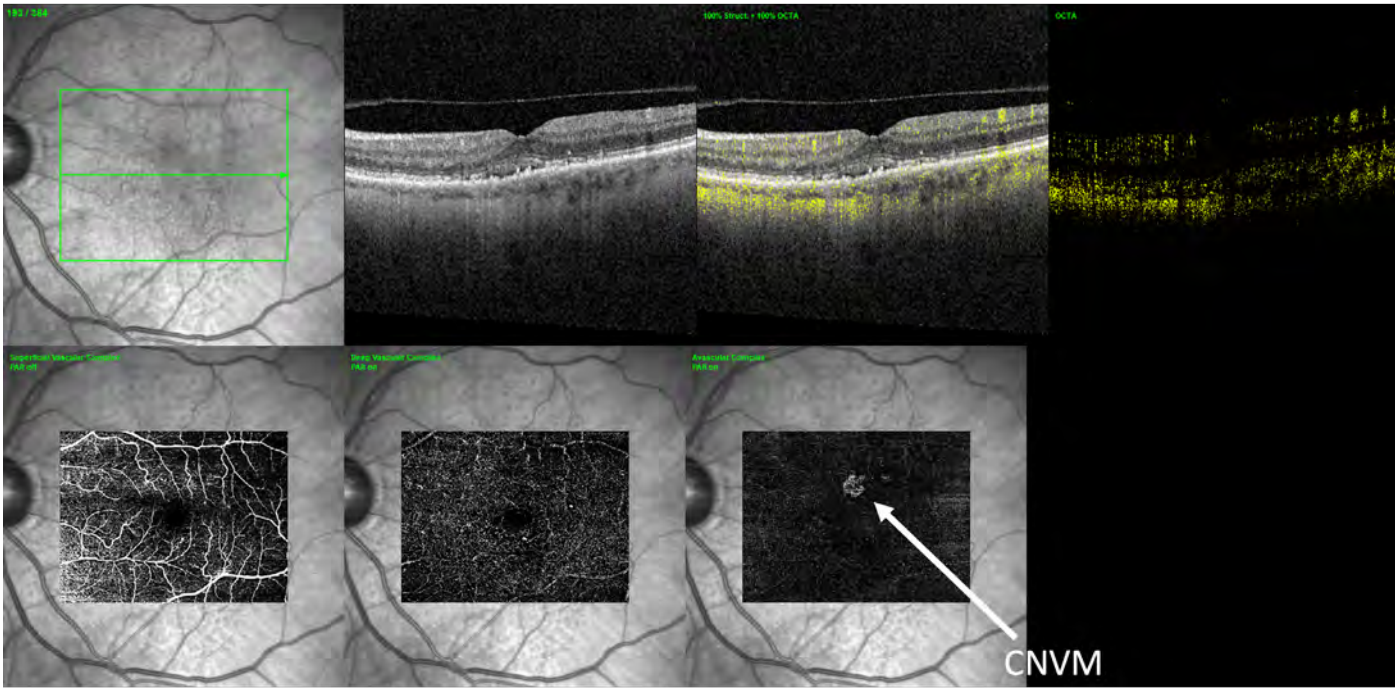

Figure 15b: OCTA – CNVM in avascular zone

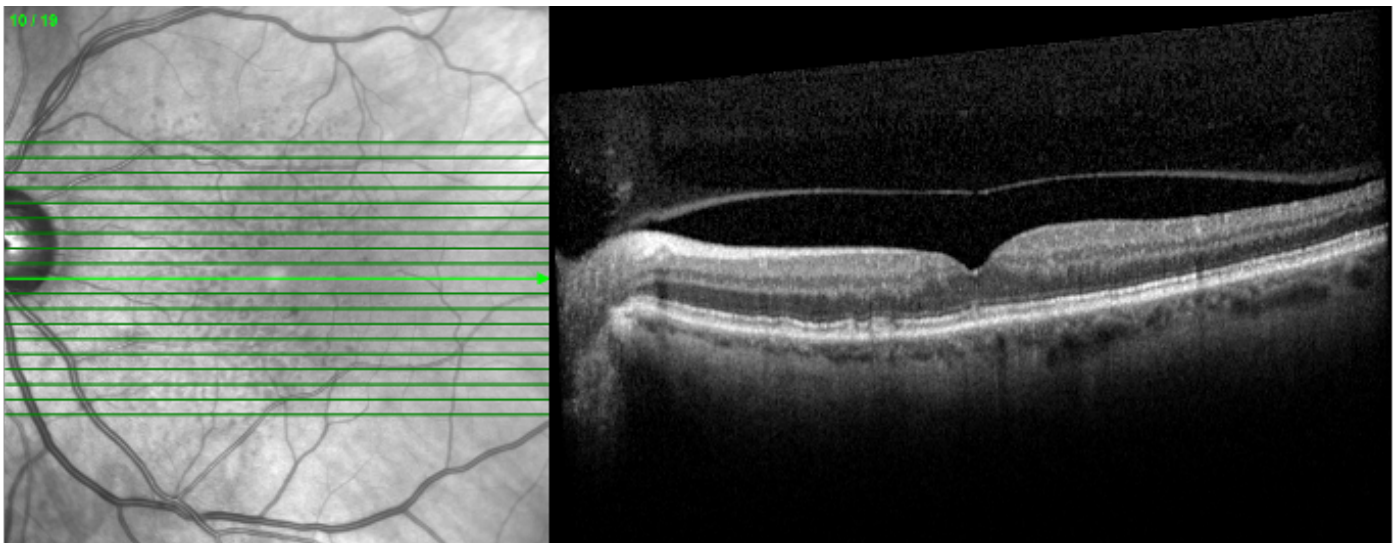

Figure 15c: Same eye after loading phase Eylea (3 injections)

Polypoidal choroidal vasculopathy

PCV is an important sub-type of neovascularisation identifiable by the formation of branching vascular networks and nodular vascular masses, called polyps.[10,11] In PCV, these branching vascular networks may have visible large vessels on clinical fundus examination and some can be as large as the retinal arcade vessels. At the outer border of these vascular lesions, nodular vascular elements may be seen with a similar appearance to aneurysms. As alternate term, *aneurysmal type 1 neovascularization*, has recently been proposed for PCV, but this remains controversial as there is uncertainty as to whether polyps are simple aneurysms or more complicated vascular structures.[12]

PCV occurs more often in younger patients, may not always be associated with drusen, and often contains a haemorrhagic component. Although not commonly seen in white Caucasian populations (most usually females, affecting both eyes with peripapillary neovascular lesions), it accounts for approximately half of MNV seen in Asians (usually unilateral presentation, mainly in males).[13,14] Large polyps may be visible on clinical examination, but fundoscopy, OCT, OCTA and fluorescein angiography are often not able to distinguish PCV from typical AMD. Indocyanine green (ICG) angiography (see later section) is the definitive method to image the polypoidal vascular structures in the choroid.

Type 2 MNV (previously termed "classic CNV)

This is characterised by the proliferation of the neovascular tissue above the RPE, in the sub-retinal space and between the RPE and photoreceptors. It may be associated with subretinal hyper-reflective material and separation of the neurosensory retina from the RPE (**Figure 15a - c** and **16a - b**). OCT angiography demonstrates vascular elements above the level of the RPE. The typical clinical appearance is a grey-green lesion beneath the retina with overlying retinal thickening, with well-defined hyper-reflective tissue above the RPE on OCT. With fluorescein angiography it is described as "classic" with a well-demarcated area of hyper-fluorescence that manifests in the early phases of the angiography. In the intermediate and late phases, there is typically intense leakage from the lesion that pools in the subretinal space. Neovascular elements may be detected in the very early phase of the angiogram. It may be more difficult to identify on indocyanine green angiography (ICGA) due to the hyper-fluorescence of the background choroidal circulation.

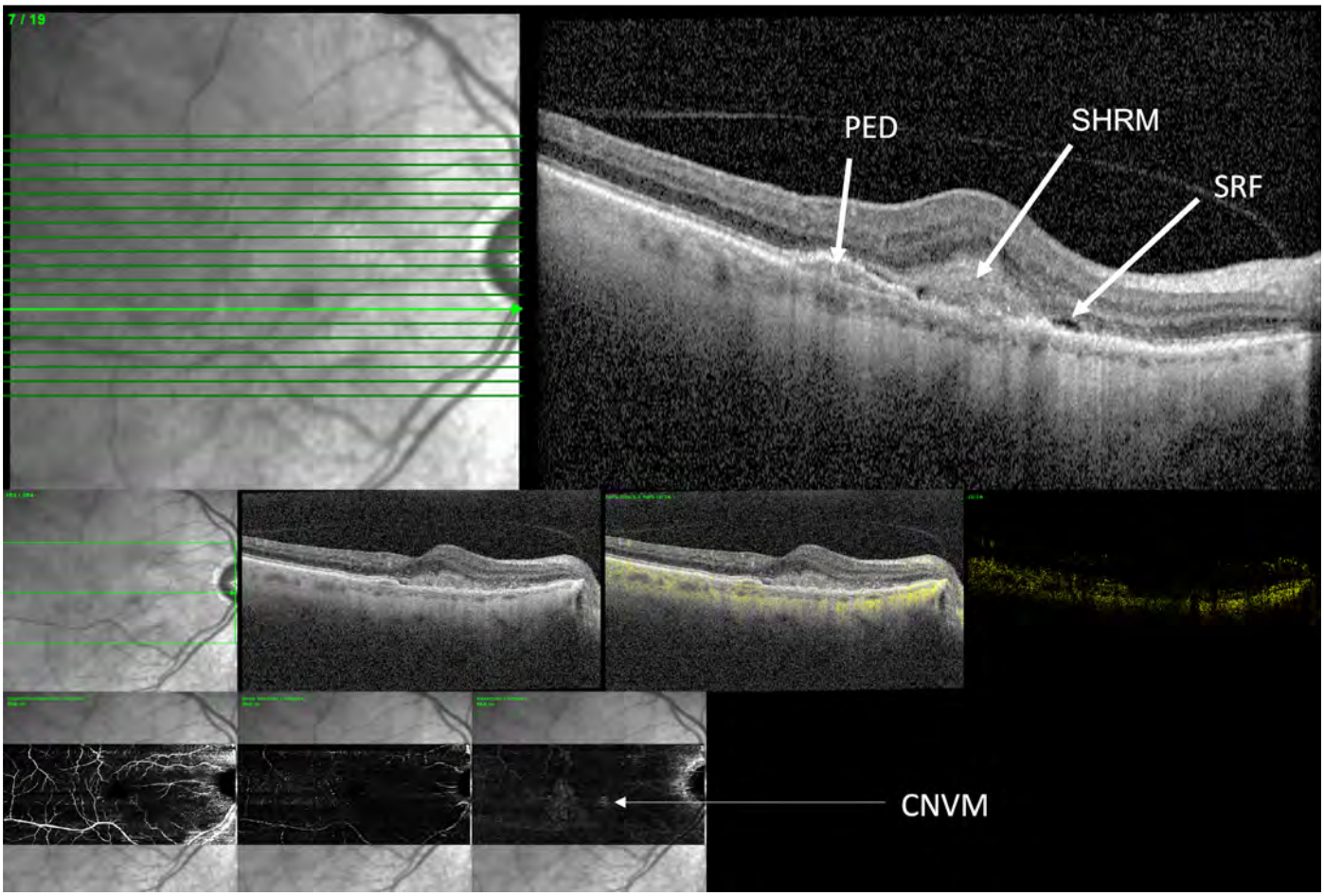

Figure 16a: Type 2 MNV TYPE 2 with sub-retinal hyper-reflective material (SHRM) and SRF

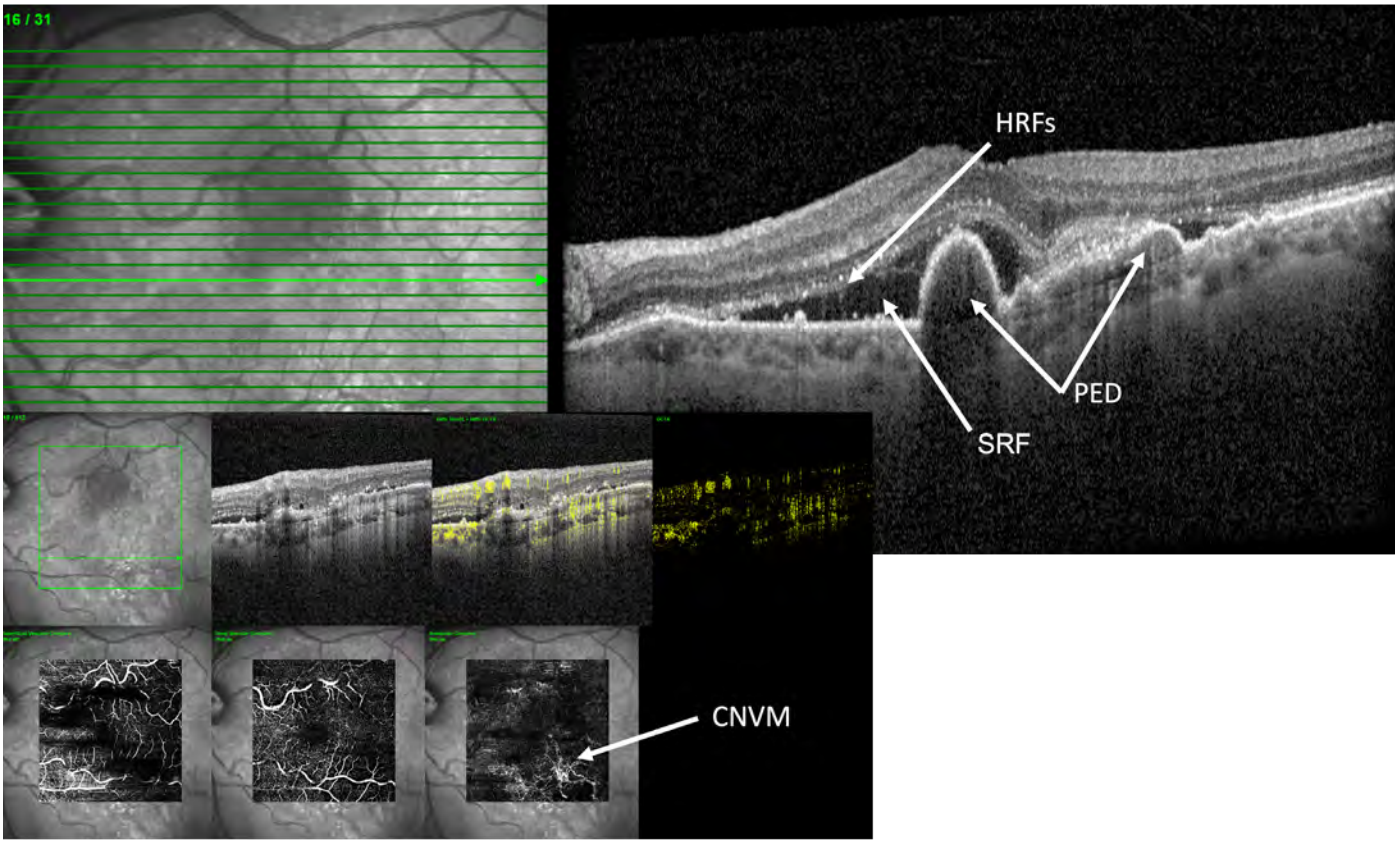

Figure 16b: Type 2 MNV – note multiple hyper-reflective foci (spots) HRF and CNVM in avascular zone on OCTA

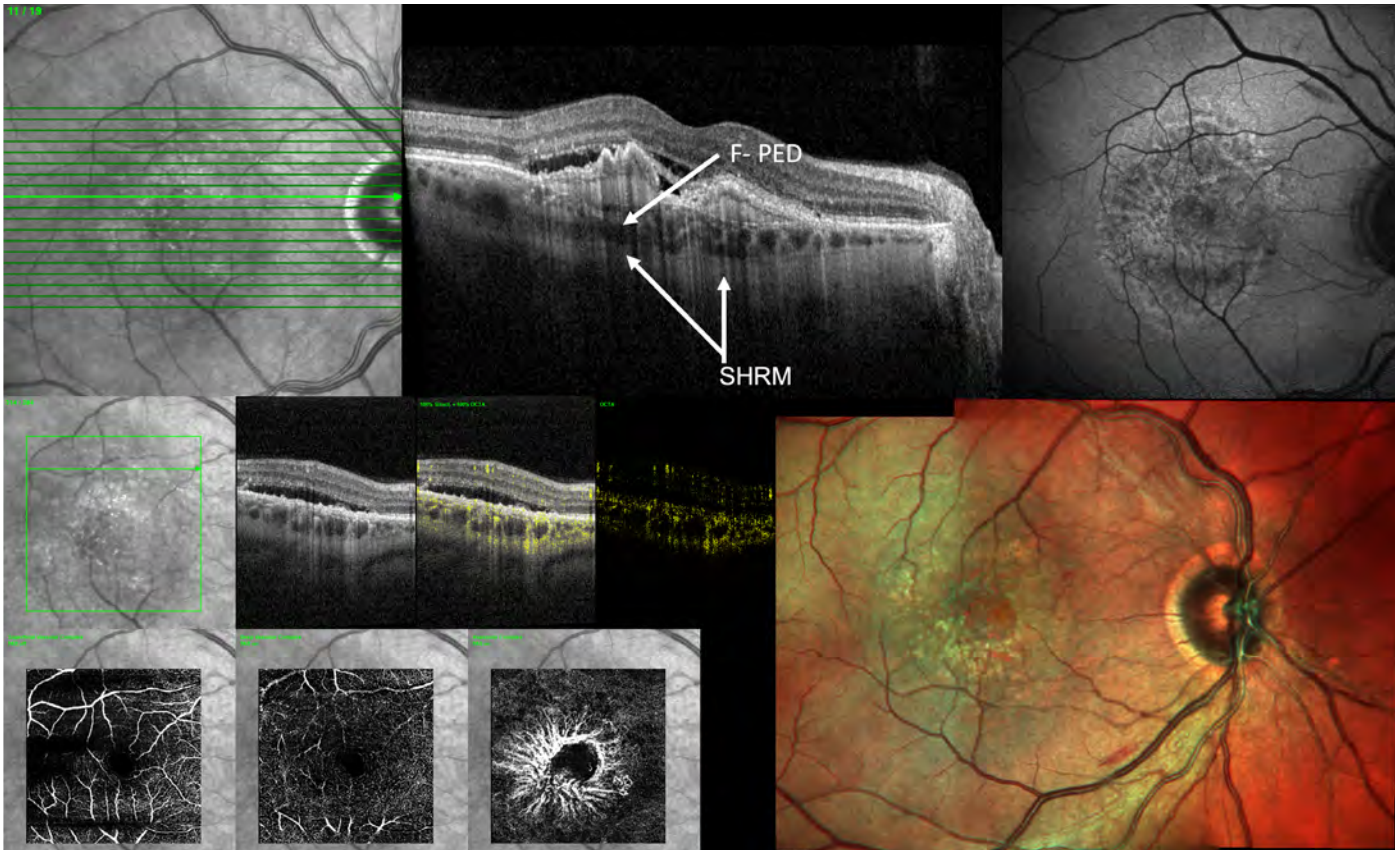

Figure 17a: Type 2 MNV – Fibrovascular PED (F-PED) with significant SHRM. Note large CNVM on OCTA

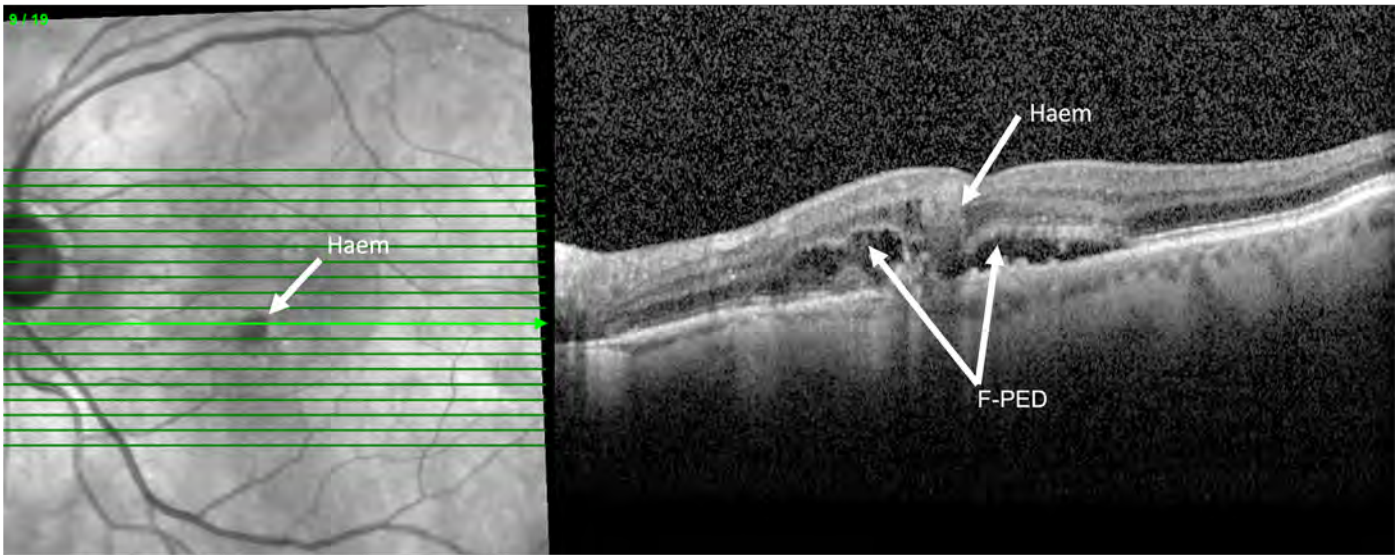

Figure 17b: Type 2 MNV – Fibrovascular PED (F-PED) with central intra-retinal haemorrhage

On OCT, eyes with type 2 MNV often present with SHRM and fluid accumulations, representing the fibrovascular tissue and exudation in this area (**Figure 17a - b**). SD-OCT localises the vessels between the RPE and the photoreceptor outer segments. Disruption of the inner/outer segment photoreceptor junction and intra-retinal cystic spaces is often observed. The type 2 vessels often respond well to anti-VEGF therapy. Sub-retinal and intra-retinal fluid, lipid exudates, and subretinal haemorrhage may be commonly seen in types 1 and 2 MNV.

This presentation is seen with more localised damage to the RPE/Bruch's membrane complex in e.g. pathologic myopia with lacquer cracks, punctate inner choroidopathy, multifocal choroiditis, panuveitis and choroidal rupture. It may also be seen in pattern and vitelliform (e.g. Adult Best) macular dystrophies, ABCA4/ Stargardt disease, reticular pseudodrusen (see below), and pseudoxanthomaelasticum.

Mixed type 1 and type 2 MNV (previously termed minimally "classic" CNV)

This is where OCT findings of both type 1 and type 2 MNV are found together. OCTA demonstrates neovascularisation in the sub-retinal pigment epithelial and sub-retinal compartments. On fluorescein angiography early hyper-fluorescence with late leakage and a larger surround of stippled hyper-fluorescence that also shows late leakage. This is often difficult to differentiate from type 3 neovascularization.

Type 3 MNV – (Retinal angiomatous proliferation)

This is characterised by extension of hyper-reflectivity from the middle retina toward to level of the RPE associated with intra-retinal oedema, haemorrhage, and telangiectasis (**Figure 18**). OCTA shows the downgrowth of new vessels toward or even penetrating the level of the RPE. With fluorescein angiography local hyper-fluorescence associated with intraretinal staining is observed. It often shows fluorescence from deeper layers suggestive of occult CNV. The neovascularisation however is not necessarily CNV.

This is commonly referred to as retinal angiomatous proliferation (RAP).[15] Clinically, RAP lesions are commonly characterised by intraretinal haemorrhage overlying a retinal vessel that acutely dives in toward the RPE. Lesions are often located in the paramacular area. Angiomatous tissue with surrounding capillary telangiectasia and dilated perfusing arterioles or draining venules can be seen in early stages. Sub-retinal fluid and/or haemorrhage are seen if type II neovascularisation has occurred. It is characterised by intra-retinal neovascularisation and has a distinct presentation on OCT with intra-retinal hyper reflective foci, as well as intra-retinal cystoid changes. As the type 3 MNV originates in the retinal capillary plexus, it also presents on OCT as an accumulation of fuzzy-looking intraretinal hyperreflective material (IHRM) with associated intra-retinal fluid (IRF). Imaging characteristics include sub-RPE CNV with intra-retinal angiomatous change along with sub-retinal neovascularisation and cystic change. Type 3 neovascularisation is mostly seen in nAMD and responds well to anti-VEGF therapy. It may also be present in e.g. macular telangiectasia Type 2 (idiopathic perifoveal telangiectasia).[16]

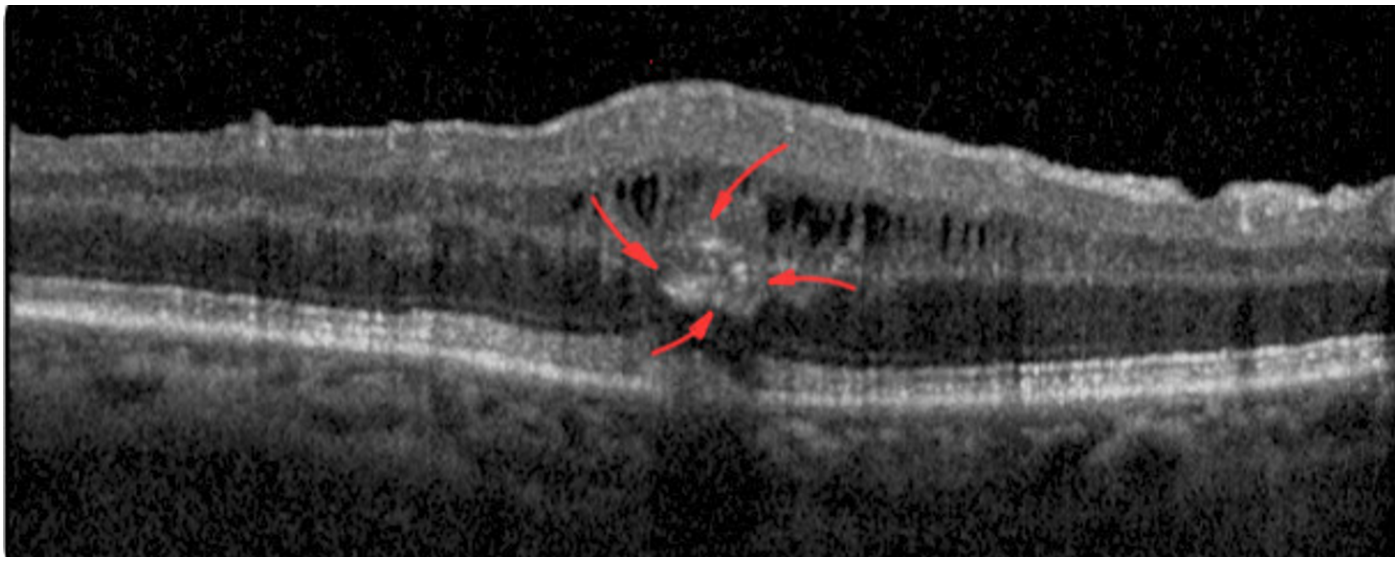

Figure 18: Type 3 MNV – with permission Heidelberg Engineering

Other OCT features

- **Drusen** - These are white or yellow deposits, of lipid rich material in Bruch's membrane of the choroid under the retina. They are often associated with macular degeneration, and the presence of drusen increases a person's risk of developing AMD. They can be described as hard or soft depending on their hard or soft edges noted on fundoscopy. Hard drusen are usually less than ~63 microns in diameter, where as soft drusen may be up to 1000 microns. Intermediate drusen are between 63-125 microns (see **Table 1**). They appear as hyper-reflective deposits above the RPE on SD-OCT in addition to their size, shape as a result of their internal consistency (**Figure 10g**).

  Cuticular drusen (CD) (**Figure 10d**) are part of the spectrum of age-related macular degeneration (AMD and have been described as triangular shaped hyper-reflective elevations of the RPE-basal lamina on OCT. When patients with CD are followed longitudinally, there was a significant risk of progression to GA or CNV. Patients with CD are commonly first diagnosed in the fifth decade of life, and there is a female predominance. Multimodal imaging is essential for characterising such lesions, using a combination of colour fundus photographs, optical coherence tomography (OCT), fluorescein angiography (FA), and fundus autofluorescence (FAF).[17] CD are often densely packed together giving rise to a "sawtooth" appearance on OCT.

- **Reticular pseudo-drusen** - Reticular pseudo-drusen (or sub-retinal drusenoid deposits - SDD) represent a sub-phenotype of AMD that was first identified on blue-light (red-free) fundus photography.[18] (**Figure 10b** and **10c**). They clinically appear as yellowish, faint, interlacing networks that most commonly occur along the arcades, and do not fluoresce on fluorescein angiography. OCT imaging of reticular pseudo-drusen shows numerous drusenoid deposits above the RPE in the subretinal space.[19,20] The origin of these lesions remains unclear, but may represent direct photoreceptor damage. Reticular pseudo-drusen were initially associated with neovascular AMD but recent studies show that they also represent a risk factor for progression to geographic atrophy.[21,22]

  Reticular means "netlike" and describes the appearance of reticular pseudo-drusen. Sub-retinal drusenoid deposits (SDD) on the other hand is a newer term for the same alteration matching the subretinal localisation in the OCT scan.

- **Central retinal thickness (CRT)** - Although an important feature in certain conditions such as diabetic maculopathy or retinal vascular occlusions (see later parts in this series), this is not as significant in CNV management.

- **Sub-retinal fluid (SRF)** - This is a separation of the neurosensory retina from the RPE by fluid which is easily detected using OCT. This is a clear sign of activity in all types of MNV and is often the first sign of activity in Type 1 MNV (**Figures 13** and **14**). Visual acuity improvements are often the most significant in cases where SRF was clearly identified at the commencement of treatment. Therefore, SRF identification is a key feature to accurately identify when deciding to refer patients with MNV for further assessment and treatment . It has been recently suggested that eyes manifesting activity by SRF only in treat & extend anti-VEGF regimens for wet AMD seem to exhibit rather low rates of macular atrophy during long-term follow-up. Therefore, SRF might be an indicator of a more benign form of wet AMD.[24]

- **Intra-retinal cystoid fluid spaces** - These are most often associated with Type 2 or 3 MNV where the lesion is above the retinal pigment epithelium (RPE). In Type 1 or sub-RPE MNV, the intra-retinal fluid appears in the later stages.
- **Exudative intra-retinal fluid** - This presents as large circular or oval shaped hypo-reflective spaces overlying a pigment epithelial detachment (see below) as well as Type 2 and 3 CNV. These respond well to anti-vascular endothelial growth factor (VEGF) therapy. However, the presence of intra-retinal cystic spaces at baseline is associated with a poorer visual outcome.
- **Degenerative intra-retinal fluid** - This can be identified by clearly defined hypo-reflective spaces in the inner retina overlying areas of RPE atrophy or scarring. These do not respond to anti-VEGF therapy and are often a sign of chronicity and may persist throughout treatment, no matter how long the duration of treatment is. Similar degenerative changes in the outer retina are referred to as outer retinal tubulations.
- **Outer retinal tubulations (ORT)** - Outer retinal tubulation usually seen in advanced stages of AMD and may sometimes be mistaken for fluid accumulations . ORT represent areas of permanent photoreceptor damage which, on OCT are recognized by a hypo-reflective lumen, with a hyper-reflective ring that consists of the PRs mitochondria and the ELM.[26] ORT is located within the avascular outer nuclear layer (ONL) and should not be mistaken for microaneurysms, which are alterations of the precapillary arterioles of the central retinal artery and lie within the inner retinal layers. ORT often overlie fibrous scars and should not be confused with intra-retinal cystoid spaces (above). The presence of ORT in AMD are a sign of chronicity and poor prognostic outcomes (**Figure 19a - b**).

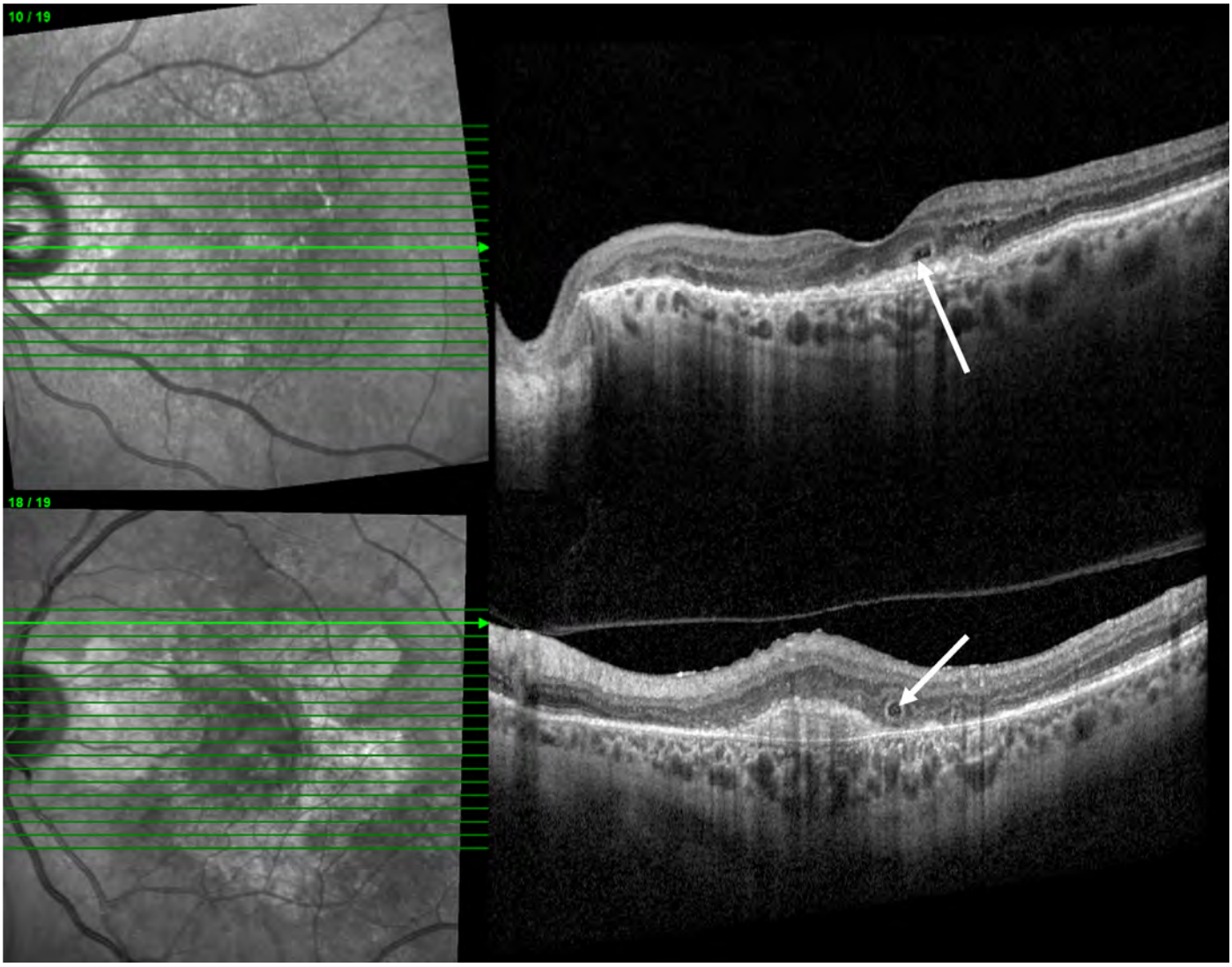

Figure 19a: Outer retinal tubulations (ORT)

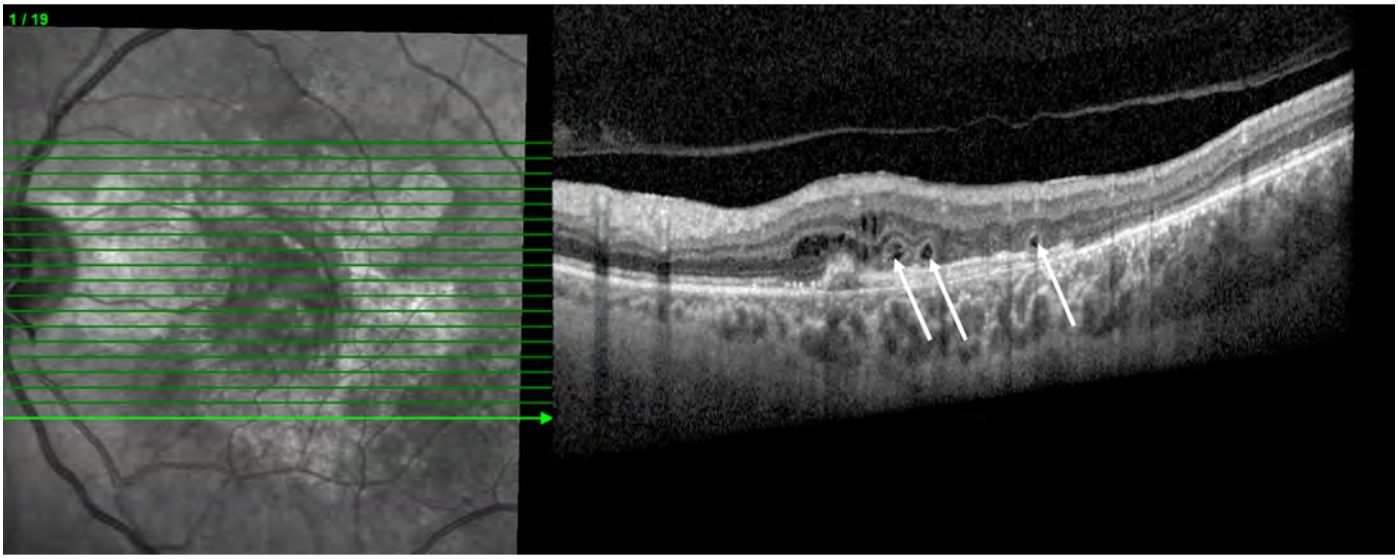

Figure 19b: Multiple outer retinal tubulations (ORT)

- **Sub-retinal hyper-reflective material (SHRM)** – Subretinal hyper-reflective material (SHRM) is the result of exudation into or under the retina of material excluding red blood cells (**Figures 16** and **17**). The material is detected by OCT and appears as regions of featureless accumulations of relatively uniform increased reflectivity compared with fluid. The material may include serum, fibrin, and inflammatory cells. SHRM is not hyper-autofluorescent, as opposed to vitelliform material, which is hyper-autofluorescent. SHRM can resolve, but fibrosis can subsequently develop and the presence of SHRM is therefore associated with poorer visual outcomes.[27] Reappearance of SHRM is a sign of recurrent exudative activity resulting from neovascularisation.[28] The reflectivity on OCT of SHRM intensifies with time as fibrosis may develop. SHRM is common and often persists after anti–vascular endothelial growth factor treatment. At 2 years, eyes with scarring have been reported to be more likely to have SHRM than other eyes. Greater SHRM dimensions are associated with worse VA. In eyes with neovascular AMD, SHRM is an important morphologic biomarker.[29]
- **Lipids (hard exudates)** – These are seen as accumulations of yellow-white globular material in or under the retina. OCT shows hyper-reflective foci (HRF) or spots (HRS) in the retina (see below). Hard exudates are a sign of blood-retinal barrier breakdown. Smaller HRS, which cannot always be detected on a fundus image, are thought to be tiny accumulations of lipids or lipid-laden macrophages and therefore precursors of hard exudates.[30] Exudates are usually located in the inner retinal layers or below the OPL as they originate from the retinal capillary plexus.
- **Hyper-reflective spots (HRS)** – These are a more recently described entity often seen scattered within the neurosensory retina (**Figures 14** and **16**). Why they appear is not yet understood, but they are thought to be possibly migrating RPE cells, pigment filled macrophages, microglial cells or lipid. They are often associated with on-going activity often leading to further disease progression. HRS on structural SD-OCT often represent a precursor to Type 3 MNV.[31] HRS are a sign of intraretinal lipoprotein exudation, pigment migration (occurs in up to 50% of eyes with AMD and is mainly associated with drusen where usually the choriocapillaris is also compromised), retinal degeneration or photoreceptor debris.[32] As photoreceptors are detached from the RPE layer due to, e.g., SRF accumulation, the shed discs and other cellular debris are no longer recycled by the RPE cells, and they accumulate around the photoreceptor tips where macrophages ingest them. So, HRS around the photoreceptor tips can usually be interpreted as photoreceptor debris or macrophages at work.
- **Shallow irregular RPE elevation (SIRE)** – This is where Bruch's membrane and RPE appear as separate lines (double layer sign – DLS) (**Figure 20a - b**). They may or may not be associated with exudation. Where there are signs of pachychoroid spectrum disease (choroid >270μm) and a SIRE, these features are frequently seen in type 1 MNV.
- **Retinal pigment epithelial (RPE) atrophy** – This is another poor prognostic sign particularly if already evident at the start of anti-VEGF therapy and is mostly associated with Type 2 or 3 MNV (**Figure 21a - c**). RPE atrophy is mostly found in advanced stages of AMD (geographic atrophy (GA)) with progressive and irreversible photoreceptor, RPE, and choriocapillaris loss. In 2020, a consensus on GA grading was reached, introducing two new terms:[33]
  - iRORA (incomplete RPE and outer retinal atrophy)
  - cRORA (complete RPE and outer retinal atrophy)

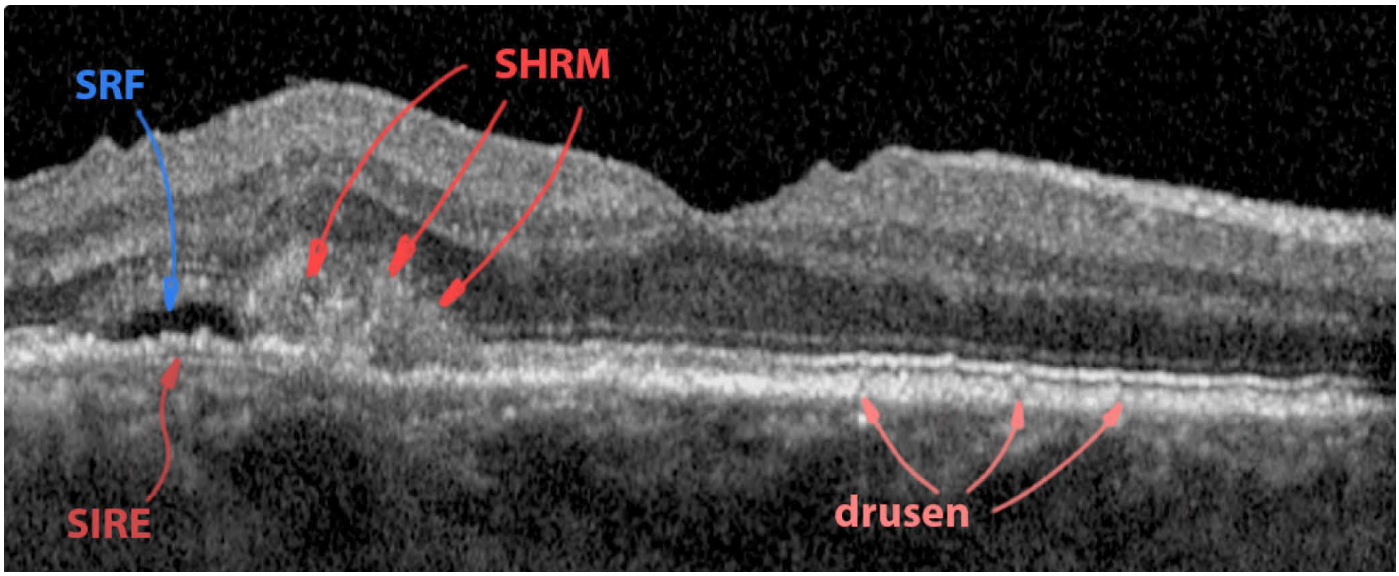

Figure 20a: Shallow irregular RPE elevation (SIRE) where Bruch's membrane and RPE appear as separate lines (double layer sign – DLS) – with permission from Heidelberg Engineering

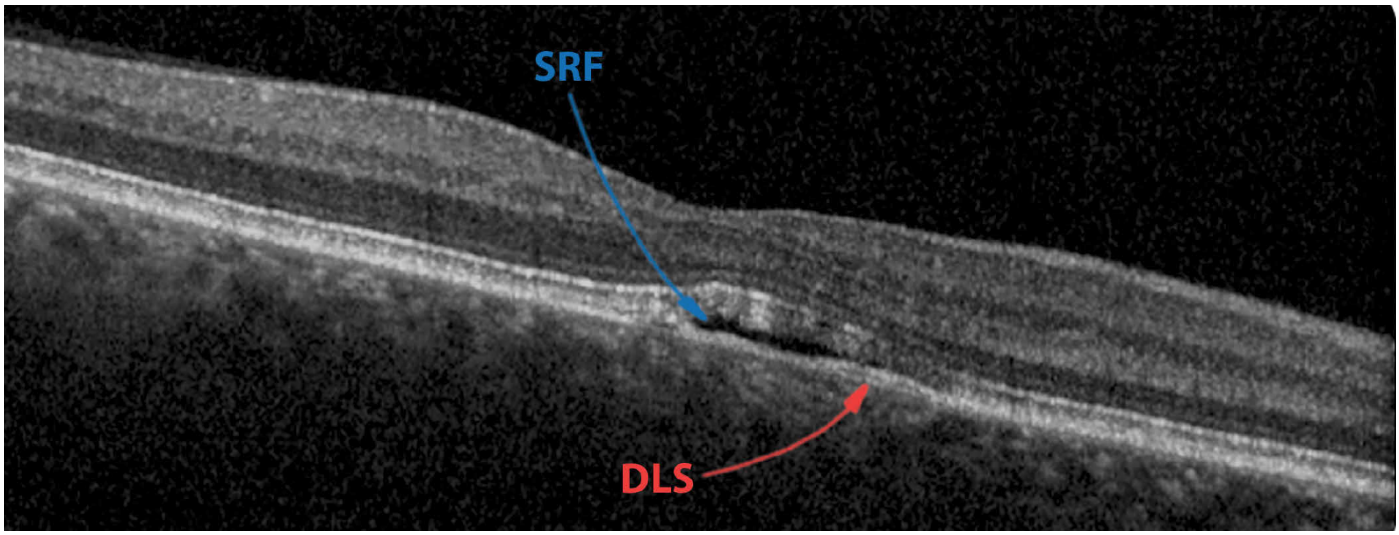

Figure 20b: Double layer sign (DLS). With permission Heidelberg Engineering

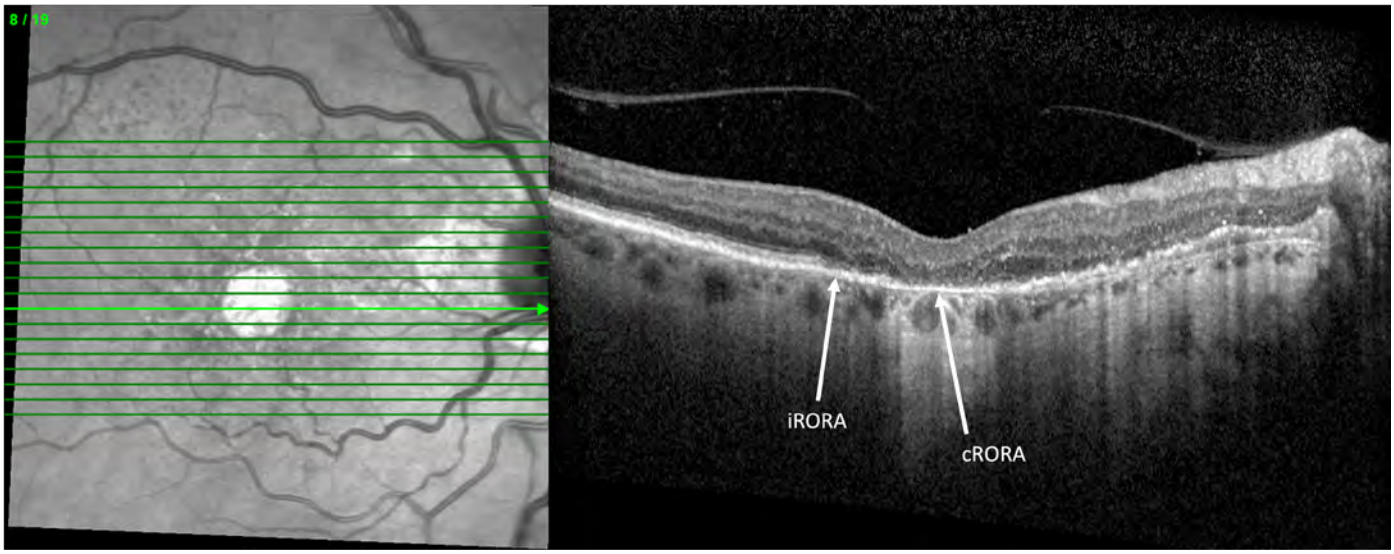

Figure 21a: Geographic atrophy (GA) - Incomplete RPE outer retinal atrophy (iRORA) and complete RPE outer retinal atrophy (cRORA)

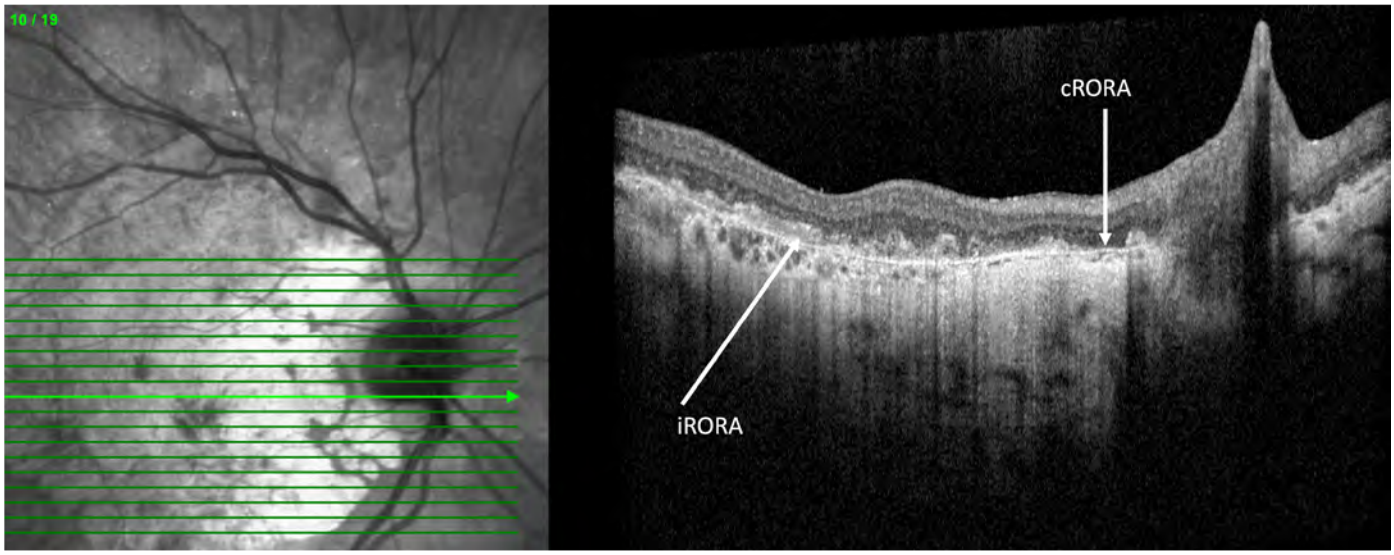

Figure 21b: Geographic atrophy (GA) - iRORA and cRORA – incompete and complete RPE and outer retinal atrophy

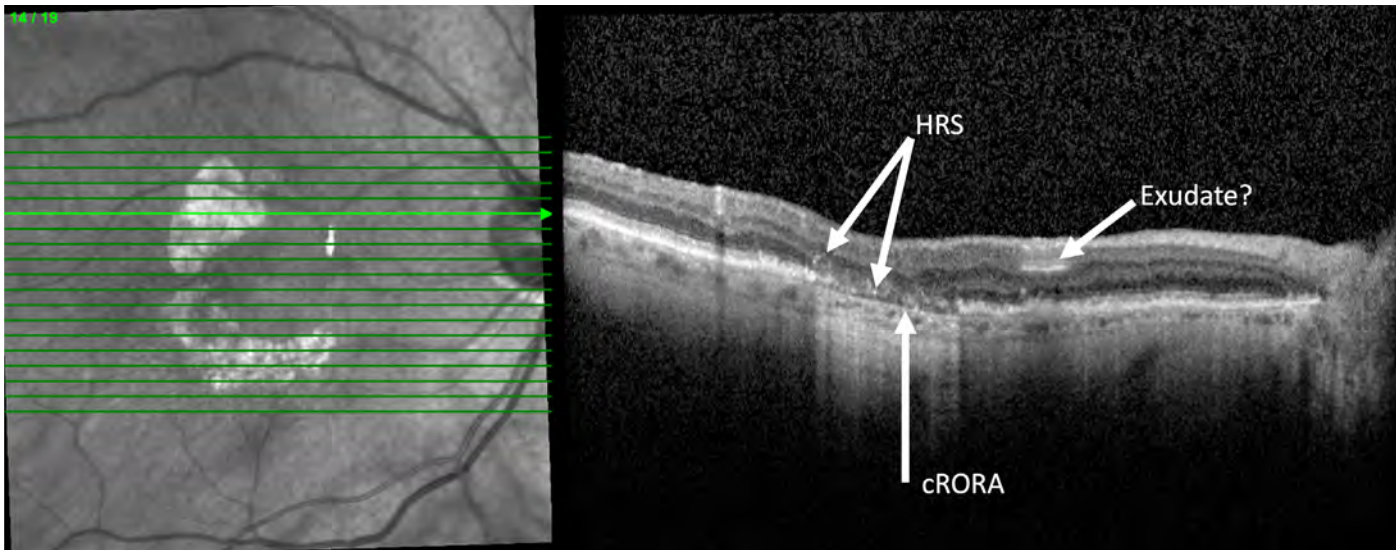

Figure 21c: Geographic atrophy - cRORA - complete RPE and outer retinal atrophy

- **Outer retinal atrophy (ORA)** - Outer retinal atrophy refers to changes seen in the outer nuclear layer with loss of visualisation and intensity of reflection from outer retinal bands, particularly the ellipsoid zone as imaged by OCT.[34] Outer retinal atrophy can be seen in areas overlying large drusen, over areas of neovascularisation, fibrosis, and in regions affected by subretinal fluid. This is as a result of the loss of photoreceptor outer segments, retraction and widening of the inner segments, and loss of the number of nuclei in the outer nuclear layer. In complete outer retinal atrophy (cORA), the ellipsoid zone (EZ) and the inter-digitation zone are not visible, the external limiting membrane (ELM)[26] may not be detectable, and the outer nuclear layer becomes thinner. Within five years 74% of eyes with iRORA progress to cRORA.[35]
- **Geographic atrophy** - This is a late presenting feature of non-neovascular AMD and is clinically characterised by a sharply delineated area of depigmentation revealing the underlying choroidal vasculature (**Figure 22a - b**). Most cases of geographic atrophy develop in areas previously noted to have large drusen. The life cycle of long-standing drusen is commonly characterised by the initial development of hyper-pigmentation, followed by hypo-pigmentation as the drusen regress, and finally geographic atrophy. The spread of atrophy can progress around the fovea in a continuous ring or in several patches located peri-foveally. Geographic atrophy usually does not affect the centre of vision (fixation) until later stages.

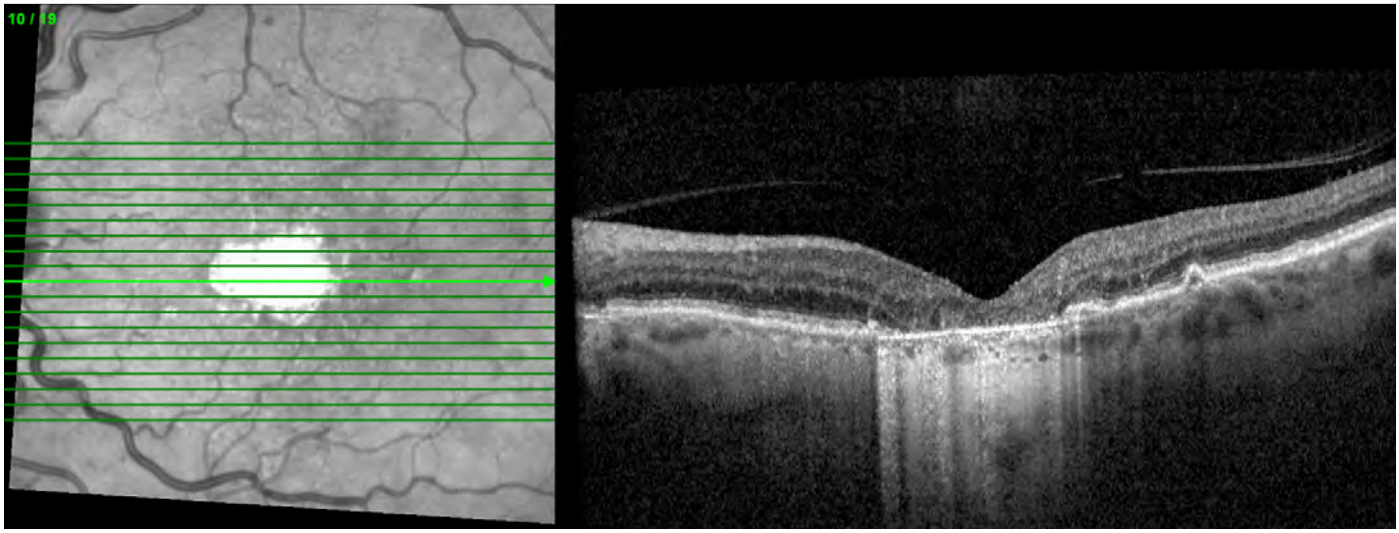

Figure 22a: Geographic atrophy - cRORA

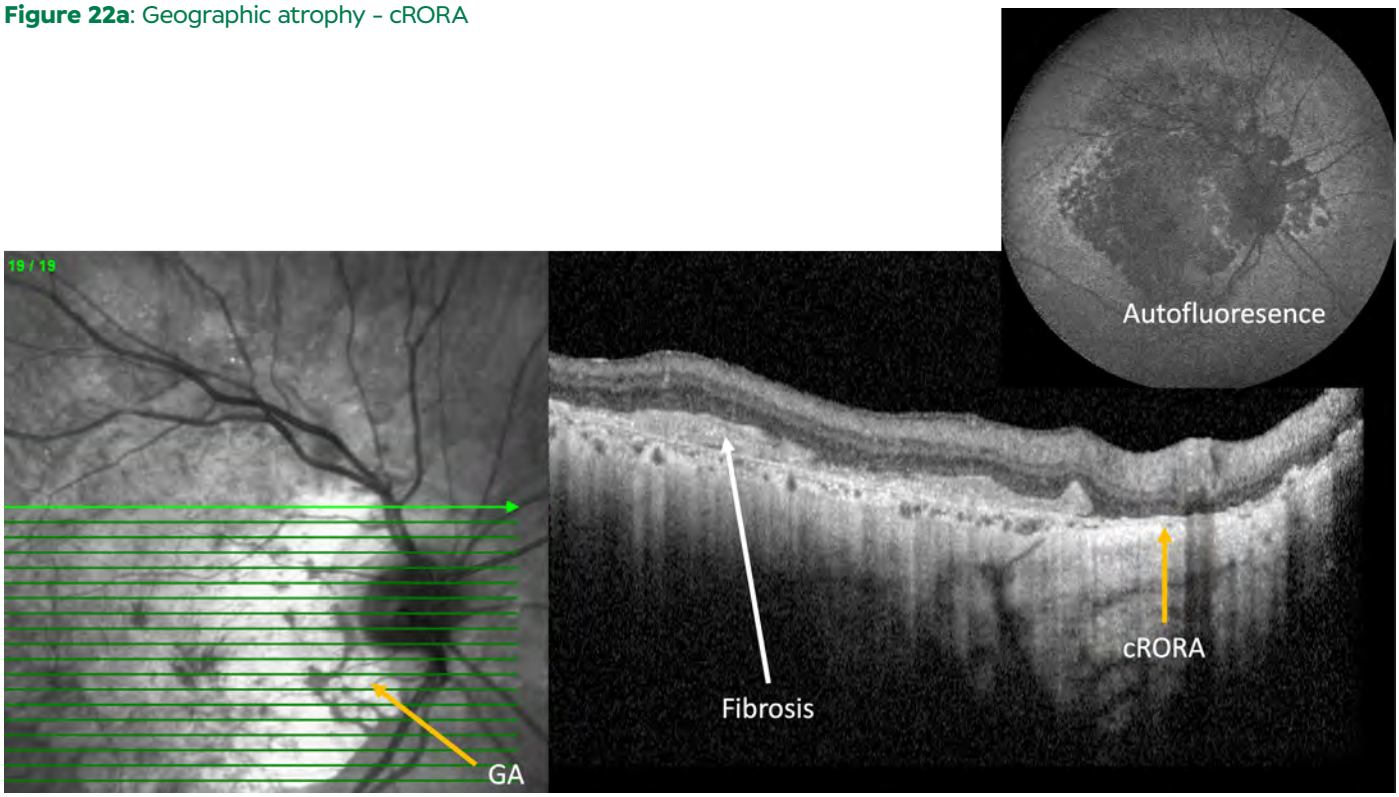

Figure 22b: Geographic atrophy (GA)

On OCT, areas of geography atrophy are characterised by loss of the external limiting membrane (ELM), inner/outer segment junction (or ellipsoid zone - EZ), and the RPE-Bruch's membrane complex. Because of the lack of RPE, there is often enhancement of the underlying choroidal reflectivity.

Fundus autofluorescence (**Figure 22b**) is a useful, accurate method for monitoring the progression of geographic atrophy (see later section). In geographic atrophy, the atrophic areas appear as sharply demarcated areas with a loss of autofluorescence, corresponding to areas of RPE loss and therefore lack of lipofuscin.

- **Pre-choroidal cleft** - This is an accumulation of fluid just below the Bruch's membrane and above the choroid. It occurs mainly in patients with nAMD who were repeatedly treated with anti-VEGF agents for MNV.
- **Vitelliform lesions** - Vitelliform (egg-yolk coloured, as viewed on colour fundus photography) material consists mainly of photoreceptor debris, caused by malfunctioning RPE phagocytosis process.[36] (**Figure 23a - c**). As viewed on OCT, the medium to hyper-reflective material accumulates between the RPE and the retina. It often has a cloudy inhomogeneous medium to hyper-reflective appearance, which can be associated with SRF and could be mistaken as a neovascularisation. The underlying conditions include Best vitelliform macular dystrophy (young patients) and various pattern dystrophies such as the adult-onset foveo-macular vitelliform dystrophy. These often get confused with wet AMD and are referred urgently, which is usually not necessary.

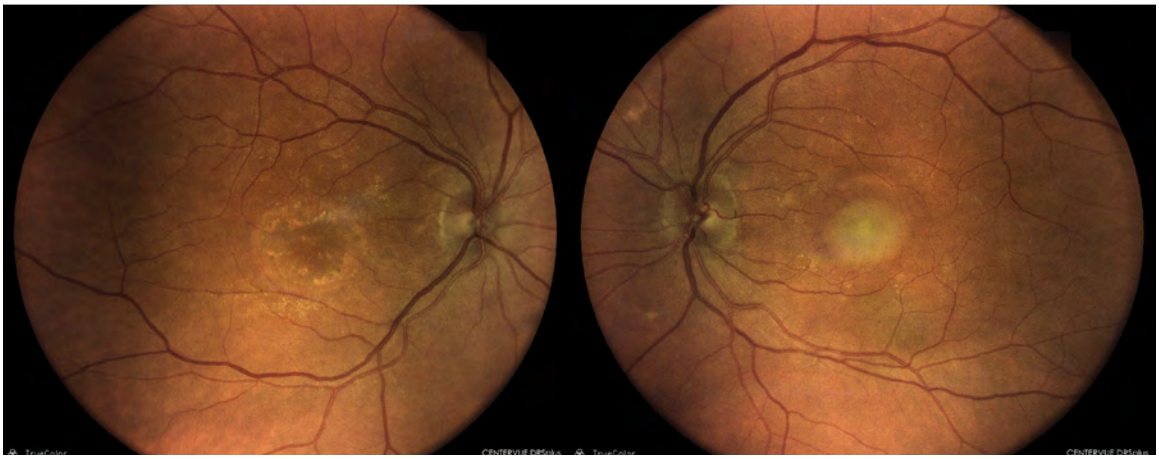

Figure 23a: Adult Best Vitelliform maculopathy

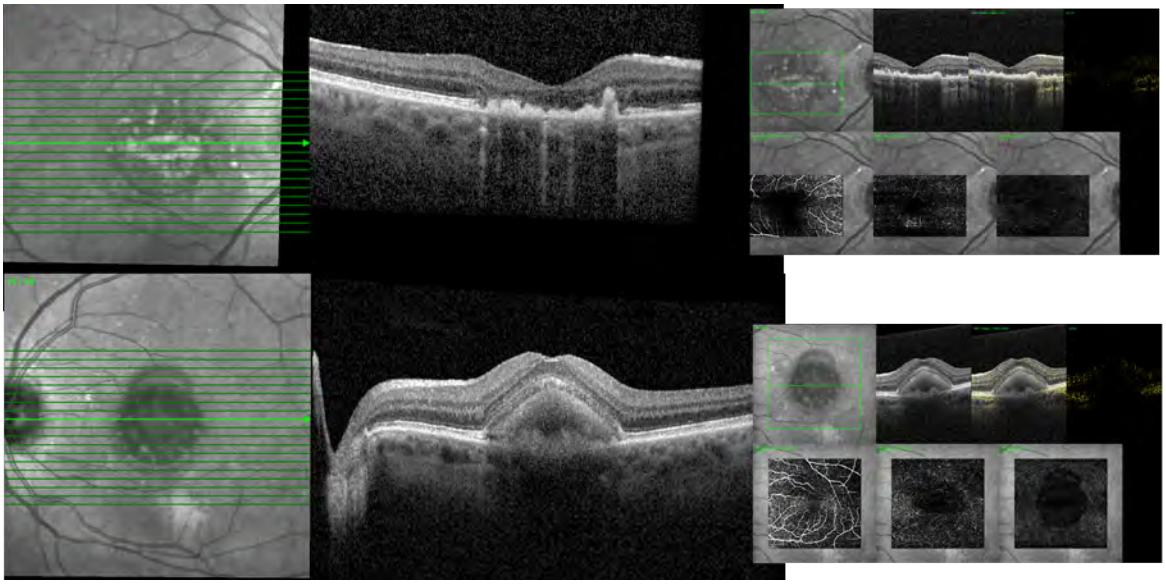

Figure 23b: OCT images of Adult Best Vitelliform maculopathy – note no CNVMs on OCTA

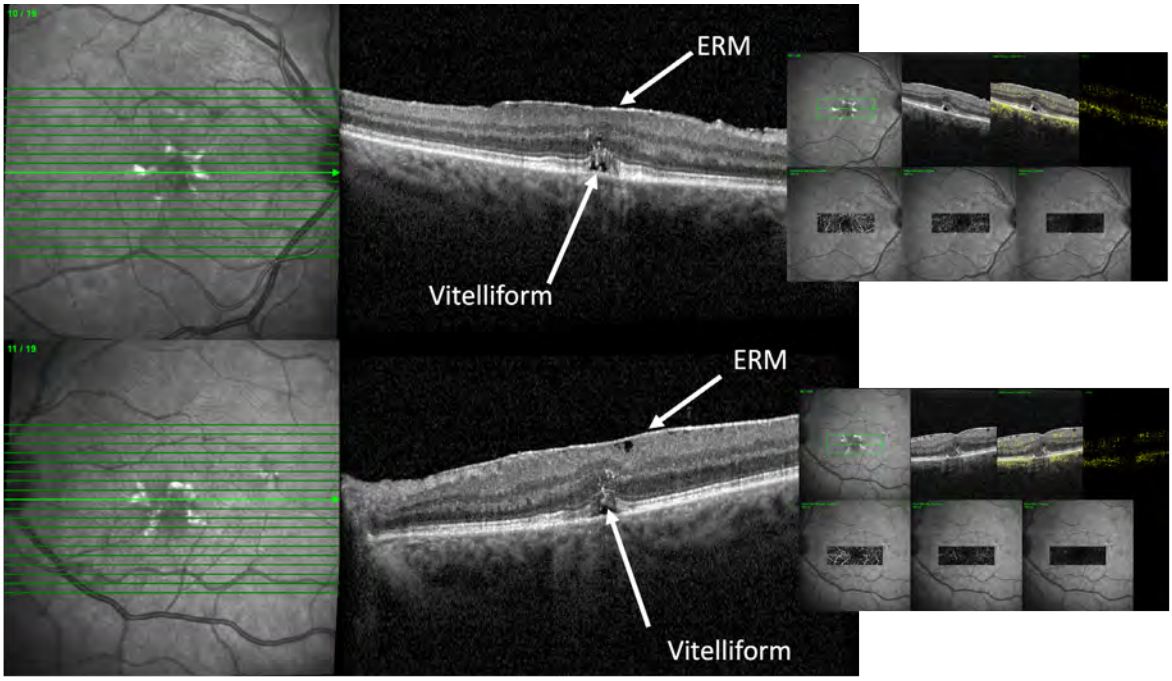

Figure 23c: Adult Best Vitelliform maculopathy with overlying epiretinal membranes

Pigment epithelial detachments (PEDs)

PEDs are caused by structural splitting within the inner aspects of Bruch's membrane separating the retinal pigment epithelium (RPE) from the Bruch's membrane.

PEDs may be classified as follow:[37]

- **Serous** – These are sharply demarcated, dome-shaped RPE elevations, characterised by clear optically empty sub-RPE spaces, which usually respond well to anti-VEGF treatment (**Figure 24**). They appear as homogenous, signal free abrupt elevations of the RPE. The most common underlying conditions are AMD or central serous chorioretinopathy (CSCR – see part 4). Serous PED in AMD is associated with neovascularisation but only accounts for around 1% of cases of neovascular AMD (nAMD). When assessing serous PEDs, an increased choroidal thickness is an important marker for CSCR (pachychoroid), while drusen are often present in AMD.

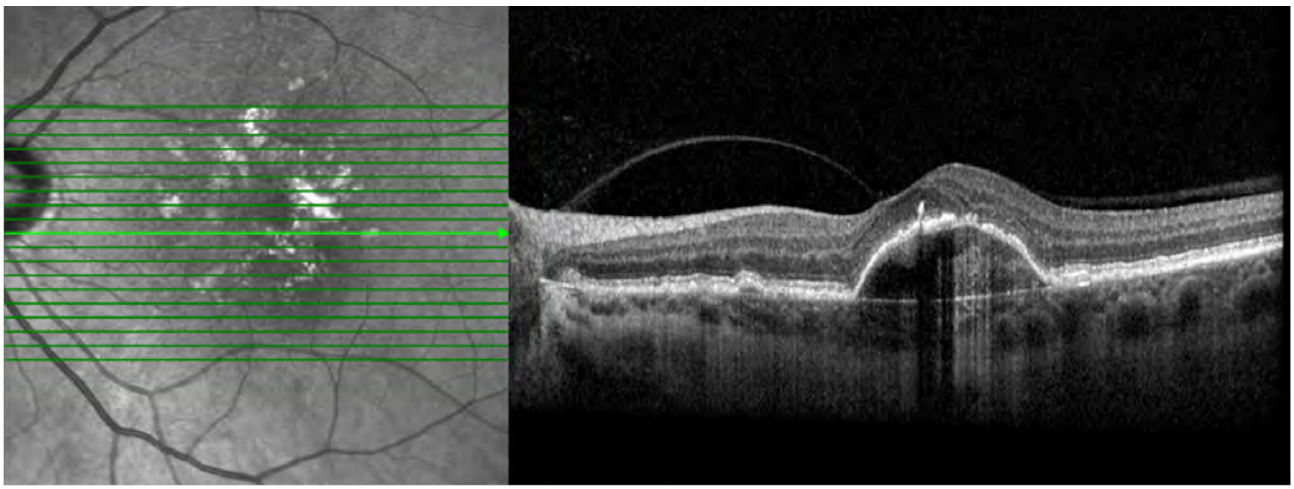

Figure 24: Serous pigment epithelial defect (PED)

- **Drusenoid** – With age, the RPE loses its capacity to phagocytise and metabolise the shed photoreceptor outer segments and other cellular components. This reduced function leads to the accumulation of lipoproteins and other cellular debris between the RPE and the Bruch's membrane, resulting in the so-called drusen – a hallmark of age-related macular degeneration (AMD) (**Figure 25**). Drusen usually have an homogenous, hypo- to hyper-reflective appearance on OCT. They can vary in size and have either distinct borders or they can be confluent. There are also different types of drusen. Once the size of a drusen increases drastically, or multiple drusen merge and reach a size of 433μm, it is called a drusenoid PED (**Figure 25**). These are slowly enlarging; shallow elevations of the RPE, where sub-RPE spaces are filled with partially reflective material. They often exhibit scalloped borders that are thought to develop from the coalescence of soft drusen. They have better short-term visual outcomes compared to the other types of PEDs.

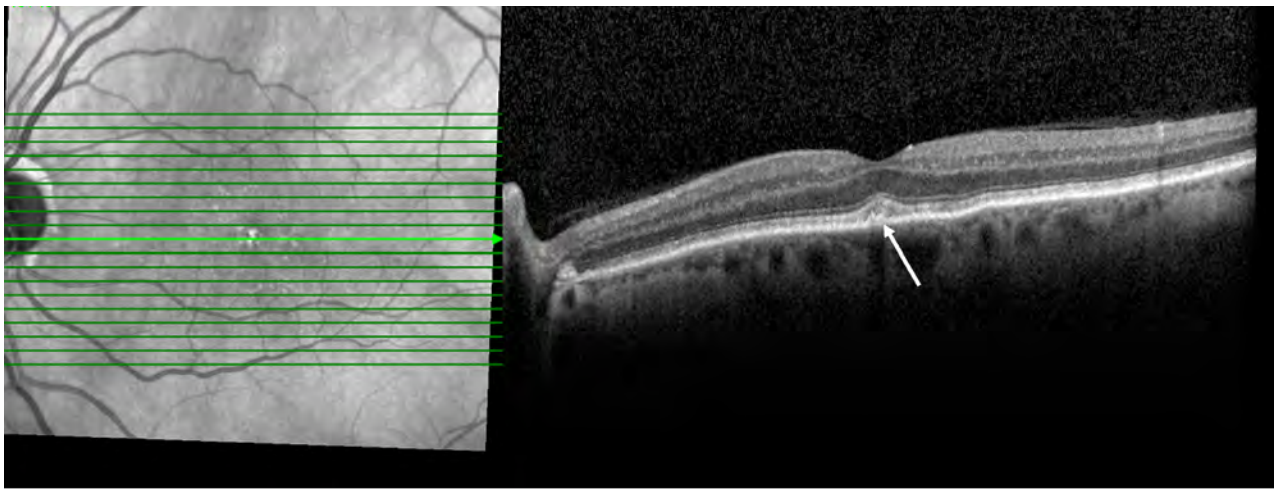

Figure 25: Drusenoid pigment epithelial defect (PED)

- **Fibro-vascular** - The Bruch's membrane is the final membrane of collagenous tissue separating the RPE from the choriocapillaris of the choroid. Degeneration and defects of the Bruch's membrane are associated with the formation of MNV amongst several other conditions. The newly formed vessels proliferate between the Bruch's membrane and the RPE, resulting in a fibrovascular PED (**Figures 15-17**). These PEDs have a heterogeneous, hypo- to medium-reflective appearance on OCT. This is typically seen in type 1 MNV but also Type 3 MNV. The juvenile vessels also tend to leak or bleed and so a fibrovascular PED is often accompanied by sub-retinal fluid (SRF), which is a sign of active exudation. This type of PED is prone to recurrences because neovascular tissue tends to persist, even though treatment may reduce the height of the lesion.
- **Haemorrhagic** - where the surface appears highly reflective with shadowing due to the blood, thus obscuring any view of the deeper layers.
- **Mixed**

The natural history of all types of PEDs involves eventual collapse. Geographic atrophy can develop after the collapse of PEDs, especially with large drusenoid PEDs. This can lead to RPE rips.

Retinal pigment epithelial (RPE) rips

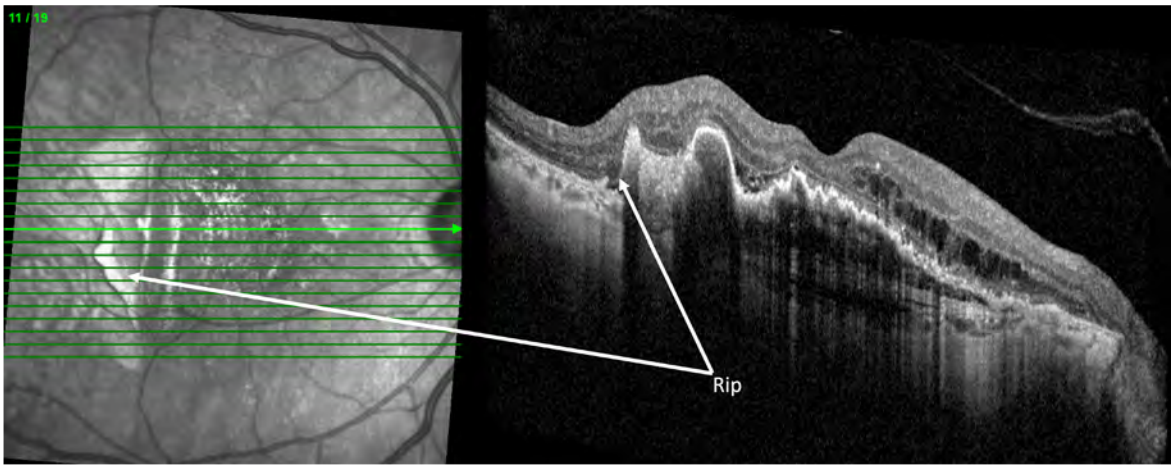

Figure 26a: Retinal pigment epithelial (RPE) rip

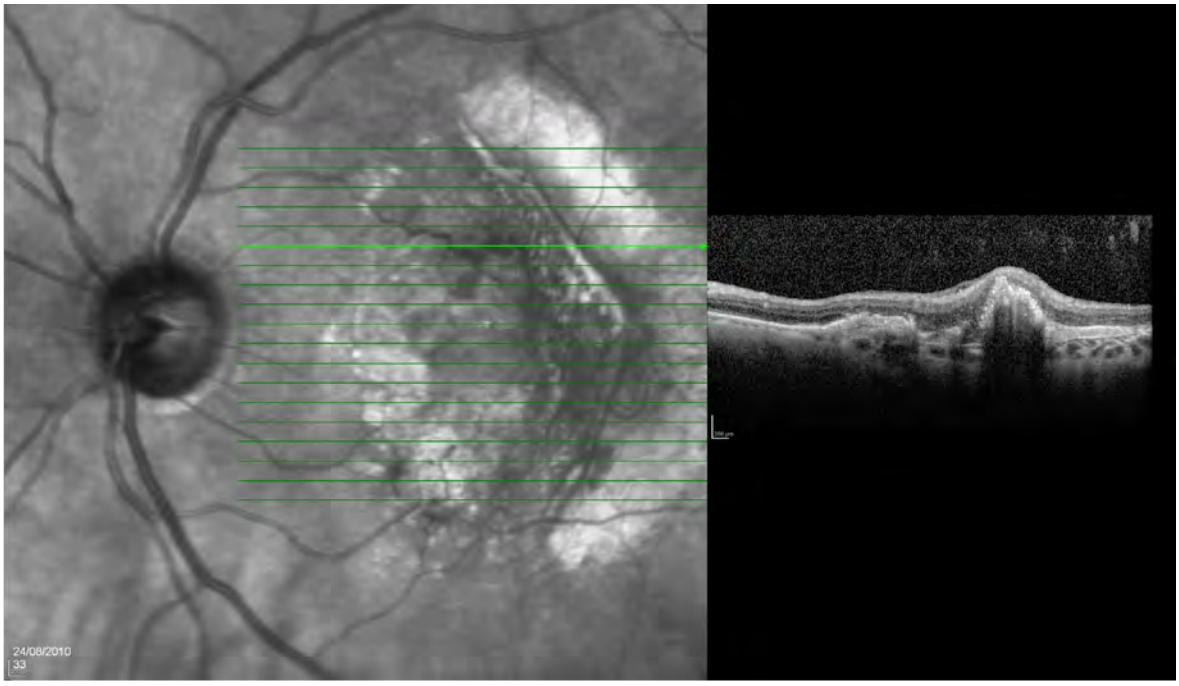

Figure 26b: Fibrosed RPE rip

An additional feature of neovascular AMD is RPE tears or rips. Rip (or tear) of the RPE is caused by a tractional dehiscence of the RPE monolayer. Tensile forces greater than the structural strength of the RPE monolayer[38] can lead to a tear. They are known to occur spontaneously as a feature of the disease and as a result of anti-VEGF therapy. With OCT, a tear in the RPE is characterised by a focal defect in the RPE with scrolling at the borders along with pleating of the adjacent continuous RPE. RPE rips are often associated with the presence of a collapsing dome shaped PED. Where RPE rips develop, this is normally associated with a poor prognosis[39] (**Figure 26a - b**).

Disciform scarring

End-stage AMD can be visualised as a disciform scar made up of hyper-reflective tissue in the same location as the inciting CNV accompanied by retinal atrophy[40] (**Figures 27** and **28a - b**).

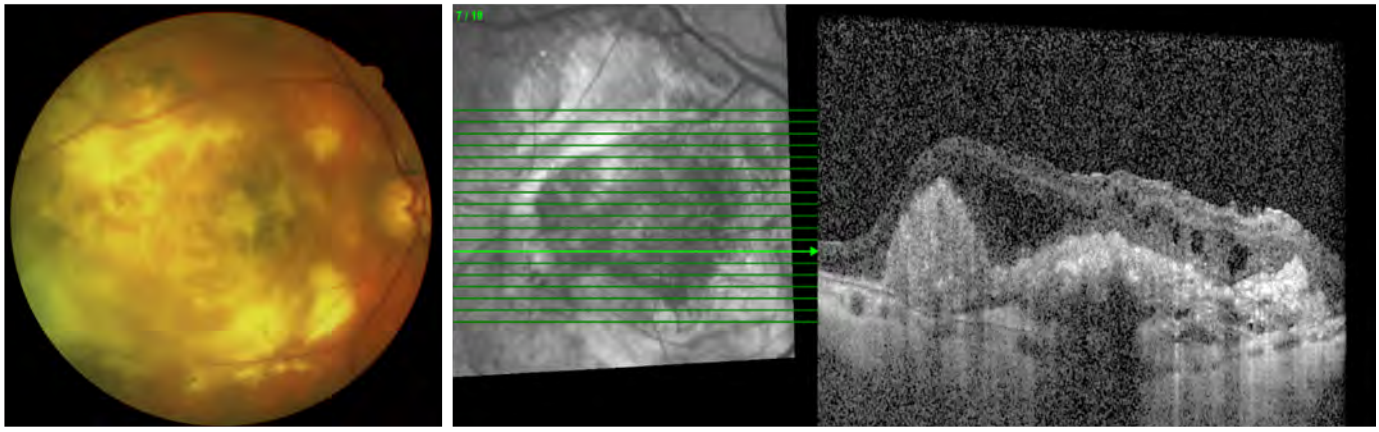

Figure 27: Disciform scarring

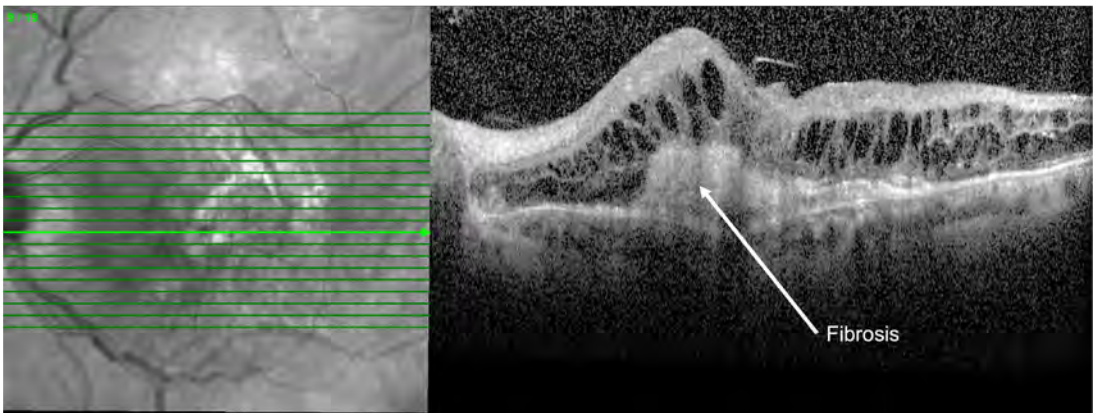

Figure 28a: Disciform scar with fibrosis

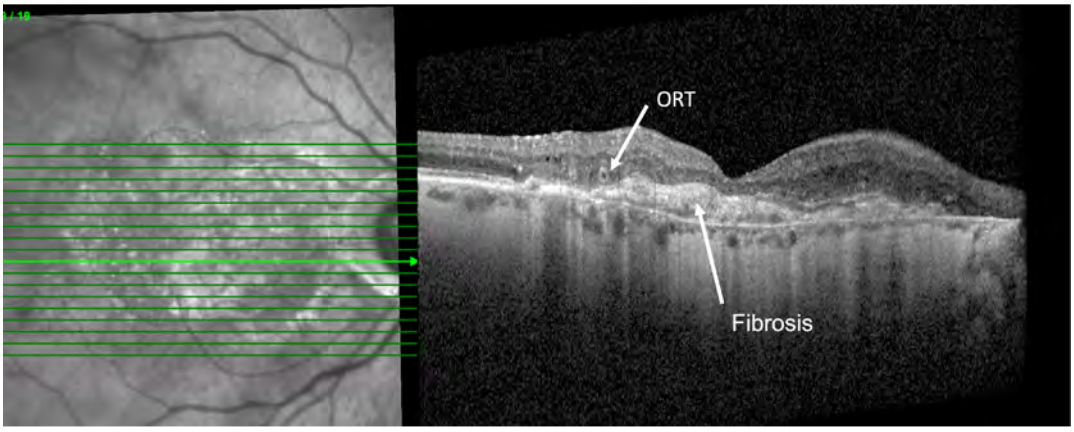

Figure 28b: Disciform scar with fibrosis

Classifying age-related macular degeneration

According to the Royal College of Ophthalmologists AMD guidelines for management there are a range of retinal conditions mimicking AMD.

Exudative Macular lesions mimicking AMD

A number of disorders can result in macular lesions which have to be accurately distinguished from AMD. These will be discussed in greater detail in subsequent articles in this series.

Diabetic maculopathy - This is the most common exudative central macular disorder in older adults (see part 2). Other signs of diabetic retinopathy will be evident on fundus examination e.g. microaneurysms, flame shaped, dot and blot haemorrhages venous beading and exudates. The visual function is often less severely affected in eyes with diabetic maculopathy compared with sub-foveal CNV. Fluorescein angiography is sometimes still needed to confirm the absence of choroidal neovascularisation and sub RPE pathology. Exudative AMD and diabetic maculopathy can often present in the same eye.

High myopia - This can be associated with choroidal neovascularisation. These neovascular complexes are believed to occur as a consequence of the development of minute cracks in thinned Bruch's membrane allowing choroidal vessels to access the sub-retinal space (**Figure 29**).

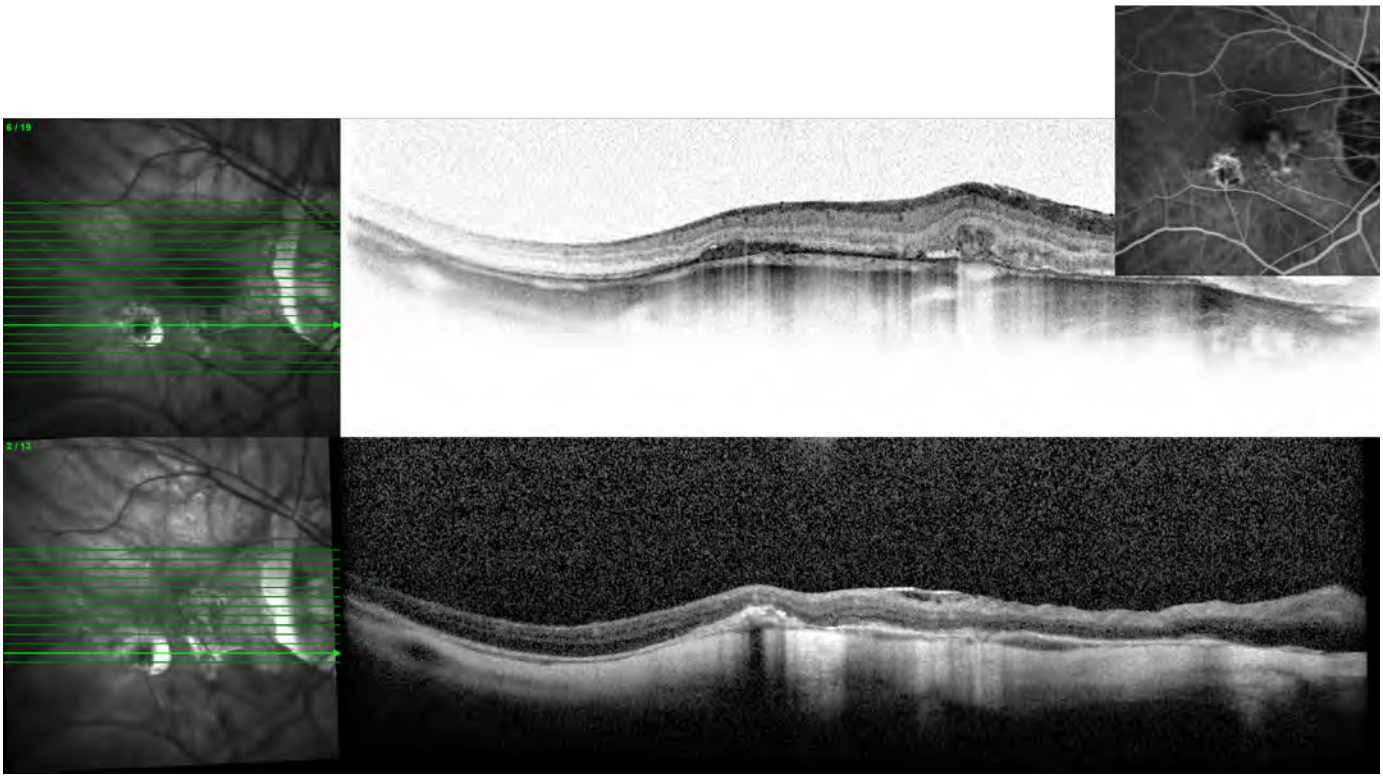

Figure 29: High myopia and choroidal neovascularisation with "classic" or MNV type 2 leakage on IVFA – pre and post anti-VEGF treatment.

Inflammatory CNV - A number of the choroidal inflammatory white dot syndromes. These are a group of inflammatory chorio-retinopathies in which the common clinical feature is the presence of multiple, discrete, white lesions located at the deeper levels of the retina and choroid. Examples include presumed ocular histoplasmosis (POH), punctate inner choroidopathy (PIC) and multifocal inner choroidopathy (MIC) can be associated with inflammatory choroidal neovascularisation.

Central Serous Chorio-retinopathy (CSCR) - This is characterised by a collection of serous fluid in the sub-neurosensory retina without any evidence of neovascularisation (see part 4). Chronic CSCR can sometimes be confused with AMD, again

the history, patient's age, symptoms and a combination of retinal imaging usually helps distinguish between the two.

Macular telangiectasia - Idiopathic macular telangiectasia (MACTEL) also sometimes referred to as peri-foveal or juxta-foveal telangiectasia and may be difficult to distinguish from nAMD, particularly with the RAP form of nAMD. Two types of telangiectasia have been described

- Type 1 MACTEL occurs in middle age, individuals and is usually unilateral. Exudative features are evident owing to leaking capillaries and resultant intra-retinal fluid accumulation giving rise to a cystic maculopathy with surrounding exudates (**Figure 30**).

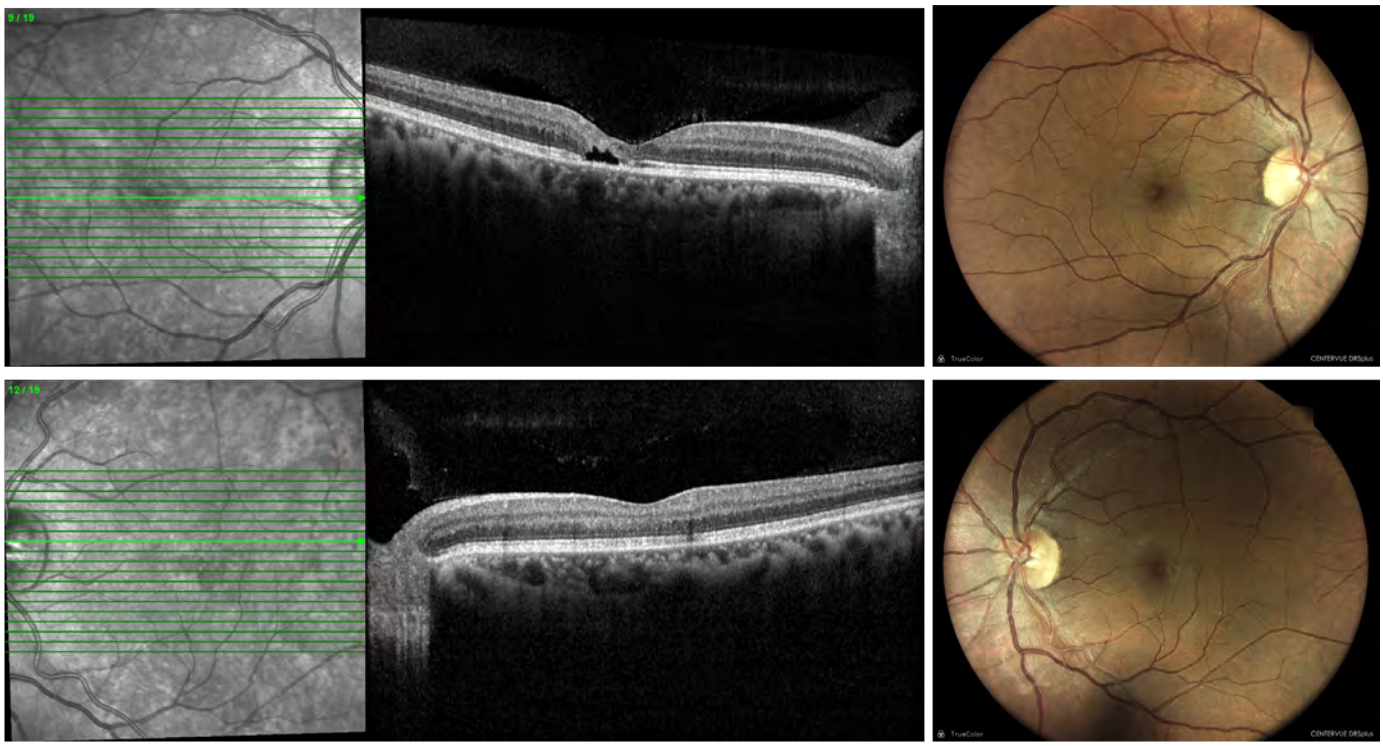

Figure 30: Type 1 Macular telangiectasia (MAC-TEL)

- Type 2 MACTEL occurs in older people and is usually bilateral with evidence of crystalline deposits, pigmentary changes, and right angled capillary changes evident temporal to the fovea then later in the entire perifoveal region. Cystic spaces are evident within the retina on OCT. Occasionally, sub-retinal neovascularization develops and arises from the retinal circulation.

Non exudative macular lesions mimicking AMD

Pattern dystrophy (PD) - PD can be easily confused with non-exudative AMD. The most common types of PD seen are: Adult vitelliform macular dystrophy (AVMD) and Butterfly shaped pattern dystrophy – less commonly.

PD is a condition which has a genetic basis; although a family history is often not present. PD is usually associated with a better visual outcome than AMD, unless complicated by choroidal neovascularisation or atrophic changes.

Diagnosing AVMD instead of AMD can be challenging. Symptoms may be similar, particularly if CNV or atrophy complicates PD, but often AVMD is identified in an asymptomatic individual at a routine fundoscopic review. Fundus auto-fluorescence imaging especially when combined with OCT is helpful in differentiating PD from AMD. Fluorescein angiography can show a typical 'corona sign' in AVMD, and the branching lines seen in butterfly shaped PD are associated with a hyper-fluorescence distributed in the area of the deposits, which does not show leakage throughout the phases of the angiogram. Occasionally, fluorescein angiographic staining of the vitelliform lesion can be mistaken for active leakage from CNV. Generally these conditions will not require treatment and monitoring is all that is required. This could be legitimately undertaken in community high street practice.

Diagnosis and referral of AMD according to NICE Guideline 82

A detailed fundus examination should always be undertaken for patients presenting with changes in vision, in particular, micropsia and metamorphopsia or other visual disturbances.

Early AMD

- Diagnosis of early AMD can be made by using slit-lamp biomicroscopic fundus examination with a Volk lens alone (66D or 78D recommended).
- Asymptomatic patients presenting with signs of early AMD do not need to be referred to hospital eye services for further diagnostic tests.

Late AMD (dry)

A diagnosis of late AMD (dry) can be based primarily on slit-lamp biomicroscopic fundus examination with Volk lens.

Individuals with signs of late AMD (dry) should only be referred to the HES if:

- Certificate of Vision Impairment (CVI) is required i.e. Severely sight impaired (SSI) or Sight Impaired (SI) registration.
- If this is how people access low-vision services in the local pathways
- If new visual symptoms are reported that may suggest late AMD (wet active)
- If the individual may benefit from participation in research into new treatments for late AMD (dry).

Late AMD (wet active)

- An urgent referral for people with suspected late AMD (wet active) to the HES, even if they do not report any visual impairment. The referral should normally be made within 1 working day, but does not need emergency (same day) referral.
- Optical coherence tomography (OCT) should be undertaken in all those individuals with suspected late AMD (wet active).
- Once referred, diagnosis in the HES depends primarily on fundus photos appearance, OCT and OCT angiography findings (see below).
- Fundus fluorescein angiography (FFA) is not usually required with suspected late AMD (wet active) if clinical examination, OCT and OCTA exclude neovascularisation.
- FFA is generally only needed nowadays with suspected late AMD (wet active) to confirm the diagnosis where OCT findings have not excluded neovascular disease.
- Where there is a confirmed diagnosis of late AMD (wet active) for which anti-angiogenic treatment is recommended this should be instigated as soon as possible (within 14 days of referral to the macular service).

Other investigations in addition to OCT that may be necessary following referral to the HES[41]

In a review by Freund et al (2019) imaging technologies that may be useful include optical coherence tomography, fundus photography, fundus autofluorescence imaging, fundus fluorescein angiography (FFA), indocyanine green angiography (ICGA), and optical coherence tomography angiography (OCTA).[42]

Optical coherence tomography angiography (OCTA)

This allows for high resolution imaging of the retinal vasculature (and to a lesser extent the choroidal vasculature) without the intravenous injection of fluorescein dye as in fundus fluorescein angiography (FFA – see below). It actually measures blood flow in the retina and choroid. To do this, multiple OCT scans are obtained in the same fundus locations over short periods of time. The flow of blood through these sections can then be detected through its effect on the reflected light (each system uses a different method, ranging from changes in phase to those of speckle noise or amplitude). Currently, OCTA systems are commercially available from Optovue (AngioVue), Zeiss (AngioPlex and PLEX Elite), Topcon (Triton), Nidek (RS-3000 Advance) and Heidelberg OCT2.

Although very useful in many situations, it cannot be relied upon to detect CNVM in all cases. OCTA is limited by the potential presence of significant image artefacts. These image artefacts occur due to eye movements in patients with poor fixation, or due to limitations inherent in the technology (e.g. so-called “projection” artefacts).

OCTA is routinely used nowadays in the HES to help confirm the presence of CNVN in new cases of wet AMD. FFA, being far more invasive and time consuming, is only undertaken for eyes where OCTA failed to identify any signs of a membrane but where OCT shows other evidence leading to a high degree of suspicion that neovascular AMD is present.

As will be seen in later parts to this series, OCTA is also useful to identify areas of non-perfusion in e.g. ischaemic diabetic maculopathy and retinal vein occlusions

Fundus Fluorescein Angiography (FFA)

This involves recording a rapid series of fundus images following intravenous injection of a sodium fluorescein dye. FFA progress through five phases:

- Pre-arterial (choroidal flush)
- Arterial
- Arteriovenous
- Venous
- Late circulation

Fluorescein is stimulated by blue light (490nm) and emits green light (530 nm). FFA images are acquired using a fundus camera or SLO devices incorporating blue excitation and yellow–green barrier filters. This aids visualisation of the retinal and choroidal vasculature. When requesting FFA it is essential to indicate which eye is of primary interest, so that early and late phase images can be obtained in order to make a definitive diagnosis.

OCT imaging has greatly reduced the need for FFA in recent years. There are times however when FFA is still helpful in the assessment of choroidal neovascularisation in wet AMD, where OCT and OCT angiography have proved inconclusive as mentioned above. FFA is also still very useful in certain cases involving diabetic retinopathy (identifying active new vessels), retinal venous occlusions, other macular diseases (e.g. central serous chorioretinopathy and posterior uveitis), as well as in the planning of laser procedures (see part 2).

Indocyanine Green Angiography (ICG)

ICG is usually performed at the same time as FFA, and is used to study the choroidal circulation. As ICG is 98% bound to serum proteins that do not pass through choriocapillaris vessel fenestrations, the larger choroidal vessels are not obscured by early leakage of dye from this layer. With an excitation peak at 810nm and emission of 830nm, the dye is excited by infrared radiation. The use of this long wavelength light enhances depth penetration, especially in cases of retinal haemorrhage.

ICG is most commonly used for:

- Assessment of patients with wet AMD where the presence of polypoidal choroidal vasculopathy (PCV) is suspected
- Investigation of patients with complex posterior uveitis and white dot syndromes
- The assessment of choroidal hyper-permeability in patients with central serous chorio-retinopathy

Auto-fluorescence

This refers to light-emitting properties of certain tissues in their natural state. The main fluorophore of RPE lipofuscin is A2E, a pyridinium bisretinoid conjugate containing two vitamin A aldehyde molecules and one ethanolamine molecule. Lipofuscin can respond to excitation by wavelengths 300–500 nm, with emission of 620–630 nm. Autofluorescence can be captured with a fundus camera equipped with the appropriate filters or more commonly now with a scanning laser ophthalmoscope or OCT.

Conclusions

OCT (including spectral domain and swept source) is an indispensable examination tool in ophthalmologic practice, as it provides microscopic, anatomically accurate cross- sections through the retina. Access to OCT imaging is an essential requirement for hospital eye services in the UK, and increasingly so for community high street optometric practice. OCT imaging plays a central role in the detection, diagnosis, and long-term monitoring of nearly all posterior segment diseases, in particular AMD as it is so commonly encountered. It is estimated that approximately 30% of adults older than 75 years have some sign of AMD.

OCT is now an essential adjunct to other clinical examination methods in the diagnosis of a wide range of ophthalmic conditions. Clinical management of a condition such as AMD, once it has been detected and diagnosed, does not always require treatment and sometimes monitoring is all that is required. This is provided clinicians are confident in their abilities to accurately differentiate the various presenting clinical signs and in particular the need to appropriately act upon features identified on with OCT.

Even after referral, OCT findings alone may well not be conclusive for a definitive diagnosis and other clinical investigations are relied upon to confirm a diagnosis, particularly where wet AMD is concerned. These include OCT angiography, fluorescein angiography and indocyanine green angiography (ICGA).

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How to explain that a piece of CPD benefits your practice and patients

Below are examples of situations where linking CPD to your practice may be less obvious. We've shown how you might link a learning objective to your own practice and patient care where the GOC might feel it is slightly out of core scope of practice:

For a DO completing a contact lens session

This CPD has the learning objective *Practitioners will have a greater understanding of the features, benefits and contraindications of toric contact lenses*. As a dispensing optician I am frequently asked by patients about their options for vision correction, and a common question is how to manage the problem of spectacles steaming up during mask wear which may be required throughout the working day. In order to help them I need to explain the options not only for spectacles but also for dual wear of spectacles and contact lenses, and know what products may suit their prescription. It is therefore important I am up to date with toric contact lenses and the patients who are and are not suitable for this form of vision correction so I can answer patient queries accurately and provide appropriate advice and care.

For an optom completing a therapeutics session

This CPD has the learning objective *Practitioners will have a greater understanding of the therapeutic management of red eye conditions*. As an optometrist, the more understanding of therapeutic options I have the better to enable me to give patients I decide to refer rather than to manage in practice information and reassurance about what may be considered in the next steps of their care pathway, to enhance their satisfaction with their care. Also as part of my CPD plan I am working towards taking on part-time work in a hospital clinic and therefore enhancing my understanding of therapeutic treatments of ocular conditions will support my development in preparation for this new role.

For a CLO competing an optometry session

This CPD has the learning objective *Practitioners will have a greater understanding of good record keeping and referral decision-making*. The cases involved investigation and diagnosis of anterior eye conditions and best practice in documentation of the results. I am part of a multidisciplinary team which is involved in a MECS eyecare scheme so I carry out under supervision diagnostic tests and complete records relating to collecting baseline data and investigation/ management of MECS patients. This session broadened my understanding of best practice in care and documentation relating to anterior eye conditions which I could come across in my MECS work.

How to log your CPD points with the GOC

In the CPD scheme the provider does NOT notify the GOC of your points.

To claim your CPD points you must enter the details on the GOC site MyGOC before the end of each calendar year.

You will be asked to provide the C-reference (see the front of this article), which will populate some of the details of this CPD unit. You will also be asked for evidence of completion. The evidence of completion you will need is the certificate which is automatically saved to your iLearn account when you complete the CPD.

More information on how to plan, access and record the CPD and download the certificate is available by clicking on the following link: CPD Information

Please send us your feedback

We would be very pleased for feedback on this or any other CPD we provide. Please give us feedback by clicking on the link provided in the confirmation email after you have submitted the quiz.