Country	CET/CPD information	Audience	Competencies	MCQs
UK	This article offers 1 non-interactive CET point (C-73779)	General Optical Council Approved CET For Optometrists General Optical Council Approved CET For Therapeutic Optometrists	STANDARDS OF PRACTICE OCULAR EXAMINATION OCULAR DISEASE OPTIONS	6
ROI	All articles are CPD accredited in the Republic of Ireland			6

OCT and Medical Retina: Case Studies

by **Jason Higginbotham** BSc(Hons) MCOptom FBDO
and **Mike Horler** BSc(Hons) MCOptom Cert Oc Pharm Oc Therapeutics

Outline

This series of online case studies presents details of the interpretation of OCT images in the diagnosis of a number of retinal conditions, highlighting the distinctive features of each condition and providing pointers on how to avoid misinterpretation.

About the authors

Jason Higginbotham is an Optometrist and Dispensing Optician with over 27 years' experience in the field. Jason achieved a high level of academic success before working in independent and multiple practices, owning his own practices, working in refractive laser, working in IOL clinics, working in hospital low vision clinics and in domiciliary optometry. Jason is currently undertaking an MSc in Optometry and is then proceeding to complete a taught PhD in Ophthalmology. After joining Birmingham Optical, Jason has been promoted to the position of Director of Medical and Education and has been providing training and CET lectures on a range of instruments and diagnostic techniques, including refractive laser, corneal surgery, OCT, dry eye assessment and other subjects. Jason has undergone extensive training with several manufacturers on numerous complex devices and is widely respected for his comprehensive training and CET sessions.

Mike is the Ophthalmic Director and Clinical Lead at Specsavers Brighton. Alongside this varied role, Mike also works as a specialist optometrist in the macula clinic at Sussex eye hospital and is an AMD Community Optometrist with Special Interest in Brighton & Hove. He offers support for other practitioners in his role as Head of Enhanced Optical Services in the South East. This role involves furthering clinical scope, development and professional standards. Mike is also a WOPEC lead assessor and sits on Specsavers Professional Leadership Council. Mike has lived and practiced overseas in New Zealand, where he gained full therapeutics endorsement and worked with ophthalmologists in co-management. He has delivered and examined on various CPD courses for students and optometrists and acted as an OCANZ examiner there. He has delivered a number of CET-accredited lectures and workshops including the Optometry Tomorrow Conference and Specsavers Professional Advancement Conferences.

GOC's Enhanced CET Scheme

CET and CPD regulators require practitioners to reflect on their learning. Additional activities are required to gain CET for distance learning.

Log into your CET dashboard via iLearn. On the menu you reach you will find non-interactive CET for this unit of learning.

For 'non-interactive' CET you have to pass (>60%) a six-question multiple-choice quiz.

The learning objectives for this article are:

2.7.2 Optometrists will have an enhanced understanding of the use of OCT for retinal assessment and diagnosis of a range of retinal abnormalities

3.1.3 Optometrists will have an evidence-based understanding of the principles of use of OCT to assess various structures within the retina

6.1.5 Optometrists will have an enhanced understanding of the identification of a range of retinal abnormalities and conditions using OCT

2.1.2 Therapeutic optometrists will have an enhanced understanding of the identification of a range of retinal abnormalities and conditions using OCT

Dry AMD

Typically, AMD patients will be over 50 years of age and the majority of subjects will be over 75. The classic presenting features affect the macular area.

Early signs of dry (atrophic/non-exudative) AMD include drusen, hyperpigmentation of the RPE and some signs of hypopigmentation. Drusen may be small and hard or soft. Later stages show confluent and calcific drusen, geographic atrophy, posterior epithelial detachment (PED), serous fluid, retinal neovascularisation (change to wet) and fibrotic scarring.

Case 1:

Drusen and hyperpigmentation

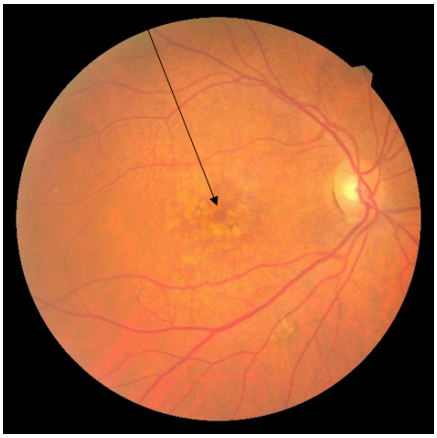
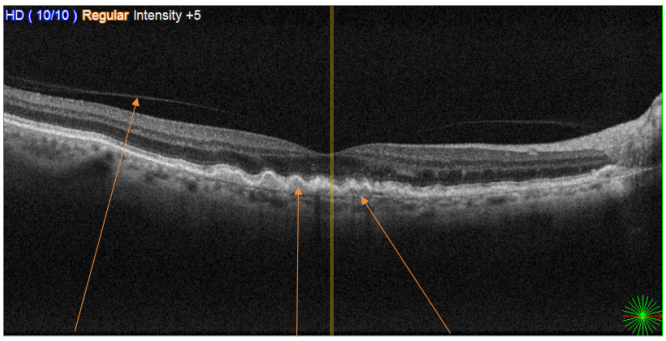


Figure 1: RE colour fundus

As can be seen in the colour fundus picture in Figure 1, there are central hard and soft drusen along with patches of hyperpigmentation.



Posterior hyaloid face **Drusen** **Bruch's membrane**

Figure 2: RE B-scan

There is nothing in the B-scan (Figure 2) that indicates any cause for concern — it would be clinically sound to continue to monitor such patients in practice. The AREDS and AREDS 2 publications would class this as a Category 1 AMD. There is validity in providing advice to these patients that they may benefit from retinal supplements (Nutrof, Macushield Gold, etc), but more advanced patients (Category 3) have been shown to gain the most benefit from reduced risk of progression to advanced AMD. However, the earlier supplements, smoking cessation and dietary changes take place, the more chance there is that AMD progression will be slowed.

Case 2:

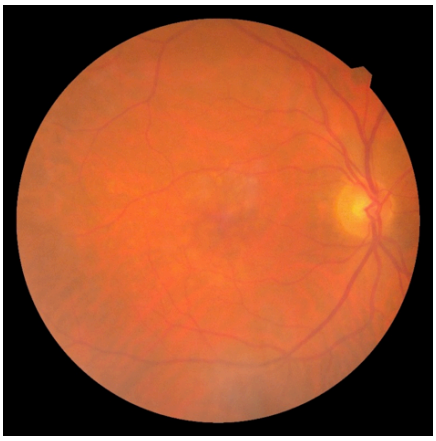


Figure 3: RE colour fundus (Patient has lens opacities)

In Figure 3, soft drusen are visible, although the view is slightly obscured by media opacities.

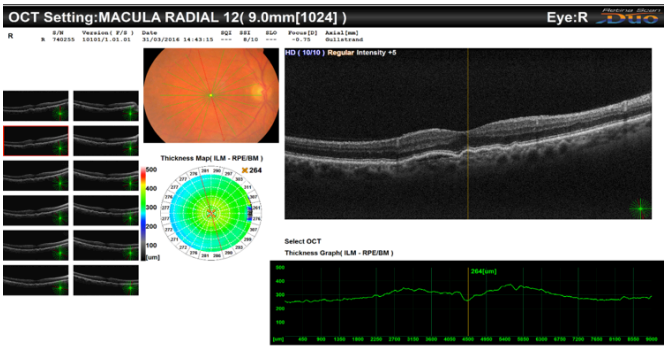


Figure 4: RE B-scans

In this case, the drusenoid PEDs look more like serous PEDs, but closer examination reveals that there is some hyperreflectivity present, indicating that these are large soft drusen (Figure 4). This is still early AMD as there are no signs of geographic atrophy, subretinal fluid (SRF) or intraretinal fluid (IRF).

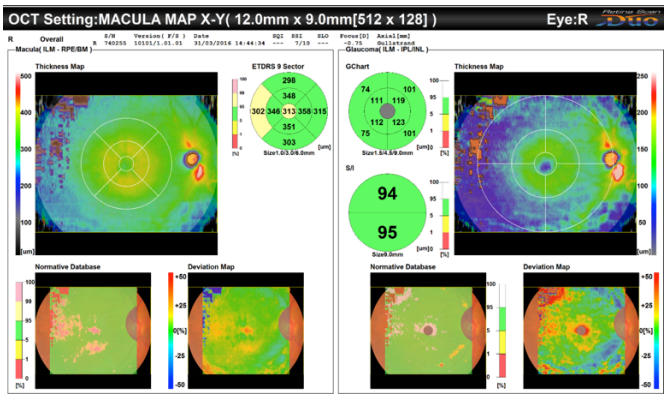


Figure 5: RE macula map

There is subtle thickening of the macula seen on the colour thickness map and on the ETDRS chart (Figure 5).

Three months later, the patient returned with increased distortion in the RE and a drop in vision.

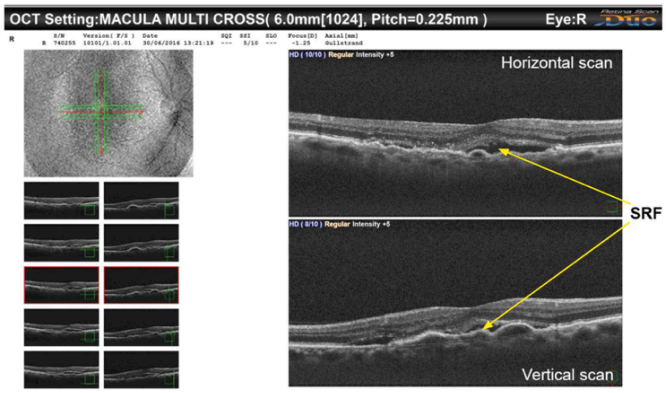


Figure 6: Macula multi scan showing that subretinal fluid (SRF) is now present

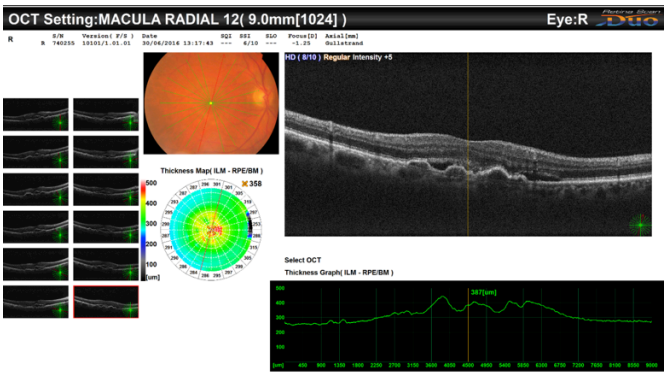


Figure 7: Macula radial scan also shows SRF

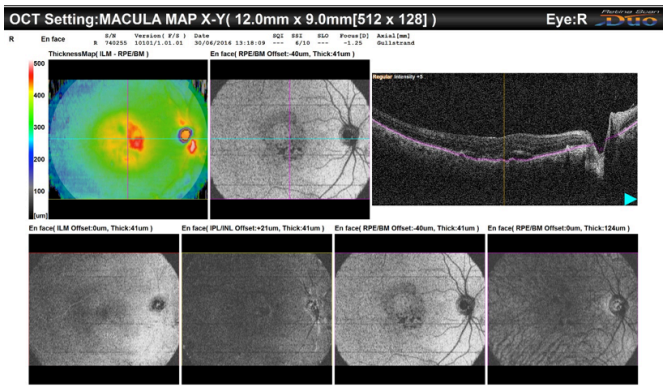


Figure 8: En Face RE showing oedema and leakage at RPE/BM as well as IPL/INL

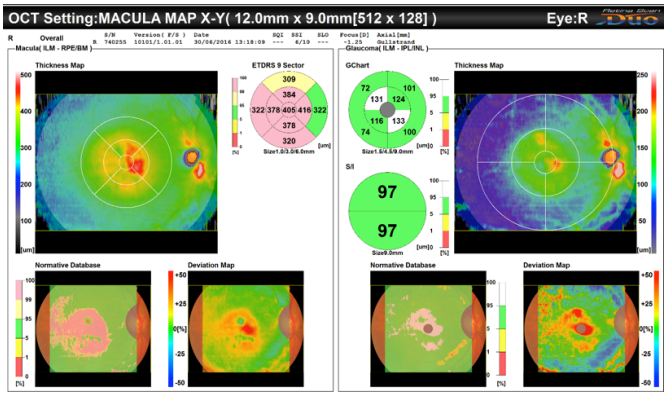


Figure 9: Increased retinal thickness on the macula map overview

It is evident from the thickness map that there is now a very raised central macula area, which is likely to be outside normal limits according to the ETDRS (Figure 9). The presence of SRF above the RPE (Figures 6–8) indicates this patient is highly likely to have wet AMD. Depending on the local CCG, hospital trust or other schemes, you will need to refer the patient to the macula clinic or into community ophthalmology at this stage. It is imperative that you are aware of your local area AMD pathway and follow it.

Case 3:

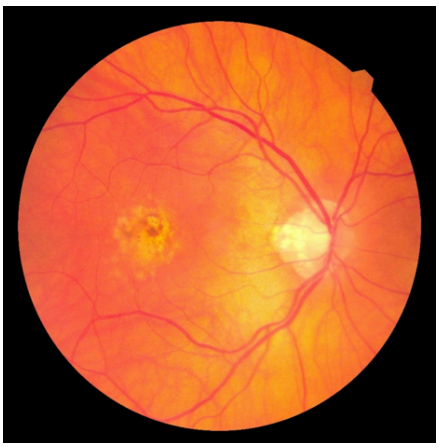


Figure 10: RE colour fundus

In Figure 10, there are confluent drusen present with what initially appears to be pigment clumps; however, looking more closely, there could be haemorrhages present.

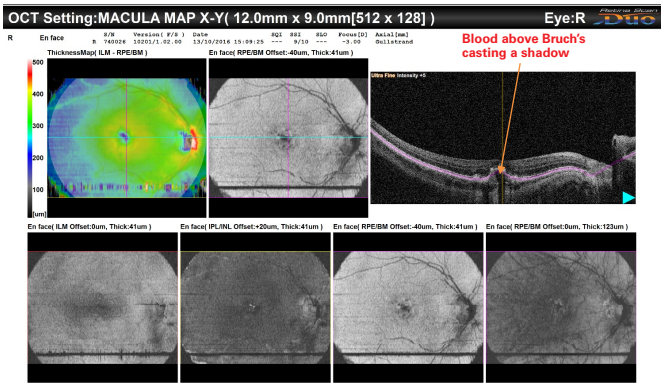


Figure 11: RE En Face showing blood 'above the line'

The shadow seen on the small B-scan on the En Face screen (Figure 11) shows that there is fresh blood (the top is hyporeflective).

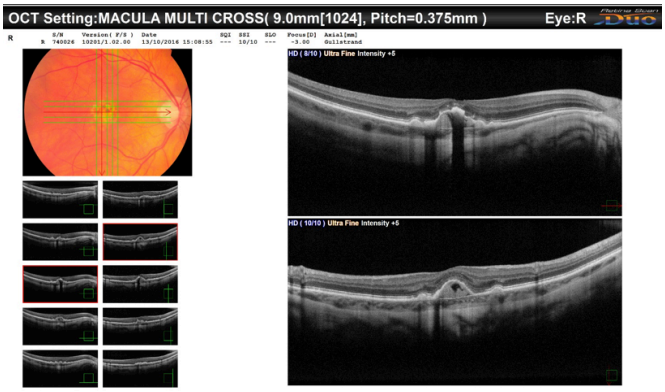


Figure 12: RE macula multi B-scan showing drusen, turbid fluid and haemorrhages

As there is no break in Bruch's membrane or obvious choroidal neovascularisation (CNV) (Figure 12), this is still classed as a dry AMD case, but it demonstrates a risk of becoming exudative/wet AMD. Urgent (though not emergency) referral to a macular clinic would be the best course of action.

Wet AMD

Wet AMD (exudative or neovascular) typically presents with some form of choroidal neovascularisation (CNV) and often a break in Bruch's membrane. Leakage can also be seen in the retina in many cases too.

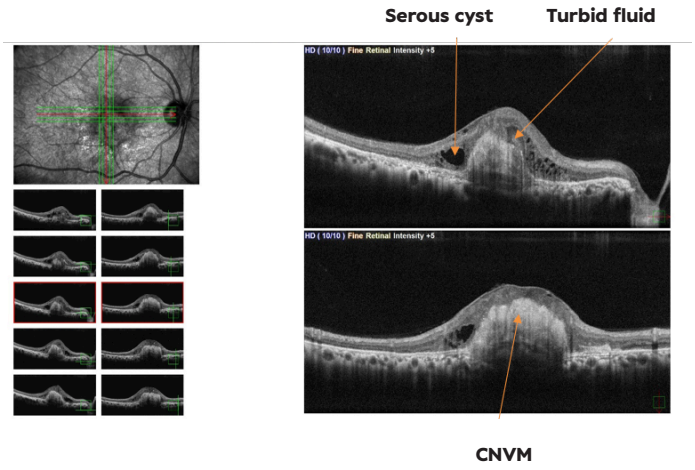


Figure 13

In Figure 13, you can see there is a very raised central lesion at the macula, showing a choroidal neovascular membrane (CNVM) with evident shadowing, indicating the presence of leaking blood and vascular fluid. There are intraretinal serous cysts as well as turbid fluid spaces showing a likely break in Bruch's membrane.

Case 1:

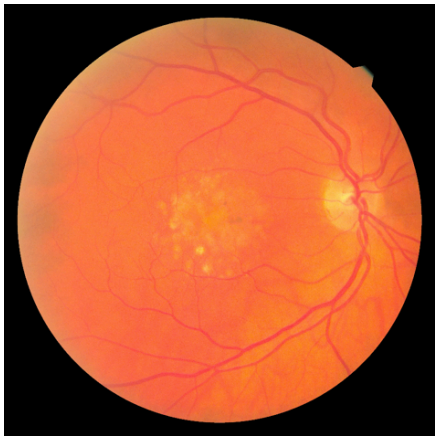


Figure 14: RE colour fundus

In Figure 14, we can see what appear to be hard and calcific drusen centrally in the patient's RE. It would be easy to consider this a fairly ordinary case of dry AMD from this image.

However, looking at Figure 15, it becomes evident that there is more happening at the macula than was apparent from the colour fundus image.

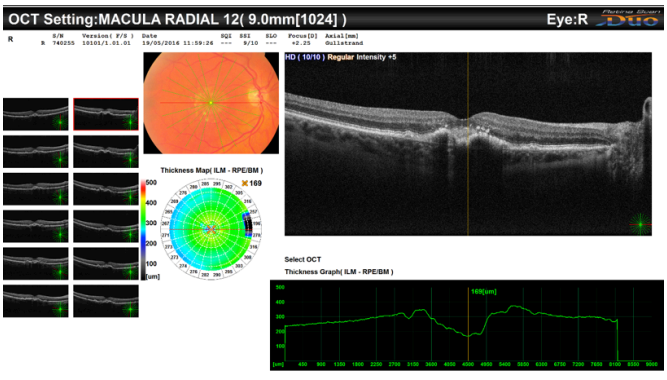


Figure 15: RE B-scans (notice this is the horizontal B-scan, i.e. 180/0 degrees)

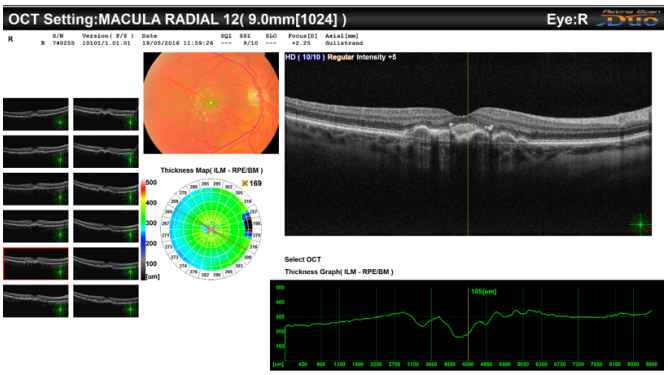


Figure 16: RE B-scans (this B-scan is a slice at 150 degrees)

From Figures 15 and 16, we can see that there is pigment migration with drusenoid PEDs and potentially some turbid fluid leakage. What looks like pigment migration may in fact be exudation and shows a case of subretinal hyperreflective material (SRHM). In such circumstances, it would be prudent to refer for an ophthalmologist's opinion. This is a classic case of 'do I refer or monitor?'. As clinicians, we need to avoid over-referral, but where there is doubt as to the presence of exudative changes, we have to consider this patient at risk of wet AMD and caution is really the best option.

Case 2:

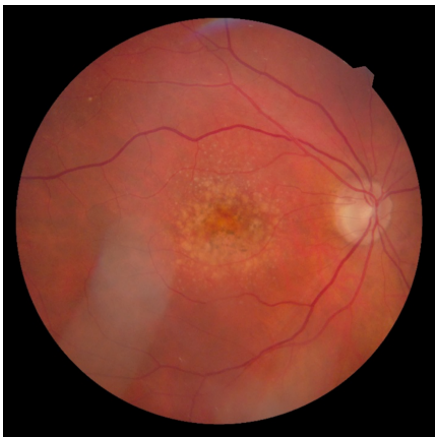


Figure 17: RE colour fundus (eyelash artefact)

Figure 17 shows central hard and soft drusen with hyperpigmentary changes. Again, this looks very much like a moderate dry AMD case from the photo.

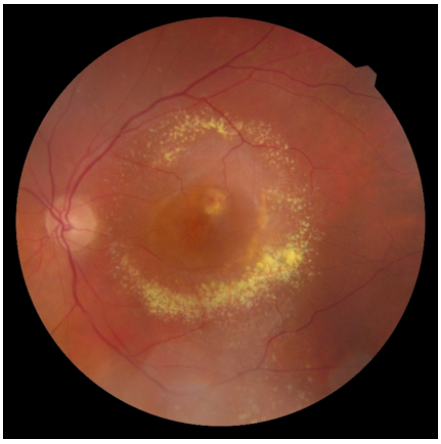


Figure 18: LE colour fundus

It is evident from Figure 18 that previous leakage has occurred in the LE and there is clearly lifting of layers and oedema present in the photo. There may be a break in Bruch's membrane visible too, with what might be some haemorrhages present. The circinate deposits show the extent of previous oedema.

Looking at Figure 19, we can see that the left macula is significantly thickened. However, on the thickness temperature map, the macula is so distorted that the computer has measured the neurosensory retinal thickness and not from the inner limiting membrane (ILM) to Bruch's membrane, hence why it shows thinned areas which, in fact, are not present. This is a useful reminder to us all that computer software is there to help us understand OCT scans, but we have to use our own judgement too.

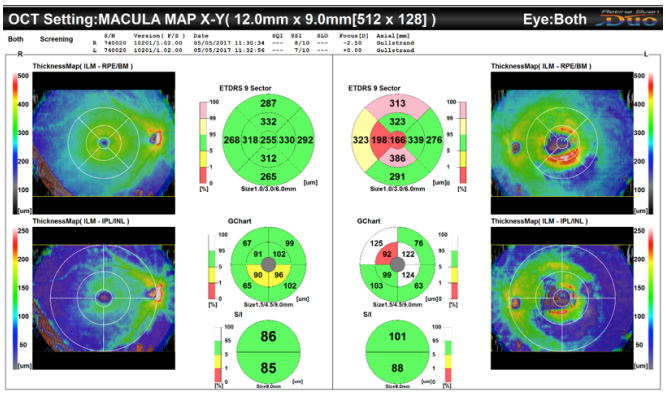


Figure 19: Bilateral macula maps

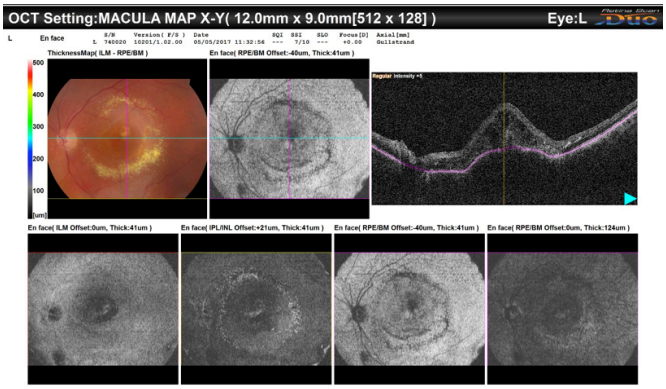


Figure 20: LE En Face (B-scan is from the map and is off-centre, hence lower resolution)

The En Face image in Figure 20 shows just how significant the oedema is, but always remember that OCT images are hugely vertically stretched and the 'mountainous' lesion is 0.7mm high – hardly Everest!

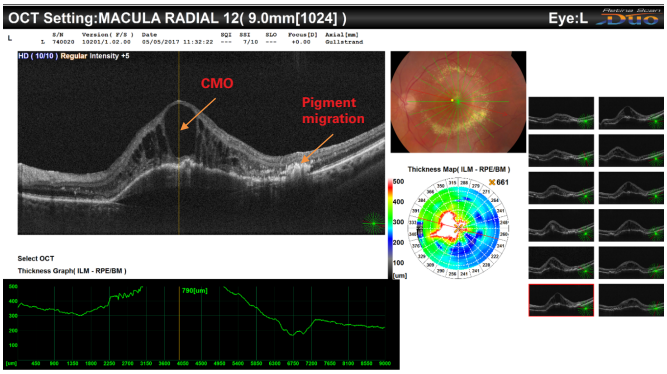


Figure 21: LE B-scans (better resolution than En Face off-centre image in Figure 20)

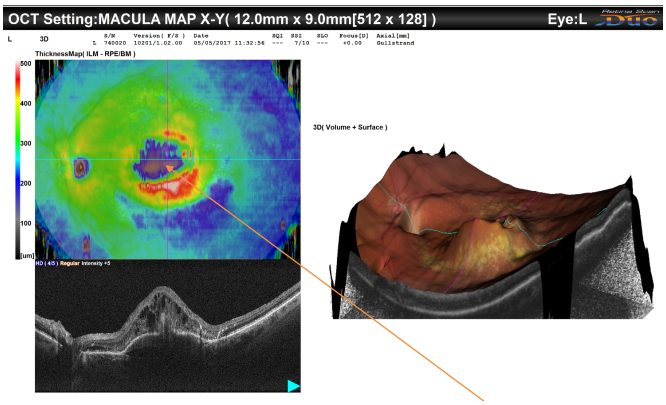


Figure 22: LE 3D colour fundus overlay **Software measurement error**

Epiretinal membranes (ERMs)

ERMs are quite common and can often be spotted using Volk, slit lamp BIO or colour fundus. However, with OCT, you will notice a much higher prevalence of ERMs in everyday practice. In most cases, you will come across patients who are achieving good visual acuities and are not experiencing any symptoms.

Where a patient is noticing distortion and/or there is a drop in vision, referral may be necessary, but it does depend on your local eye department as to when you should refer. From experience, across the UK, referral criteria for ERMs do vary. In some areas, where VA has dropped below 6/18 with significant distortion, this would be enough for them to consider pars plana vitrectomy and membrane peel. In other areas, VA would need to have dropped to 6/24 (or even 6/36) before they will consider such surgery due, in part, to the inherent risks and costs to the NHS. Another factor for consideration is the extent of any 'hole' formation by the ERM — this will be discussed further later on.

Case 1:

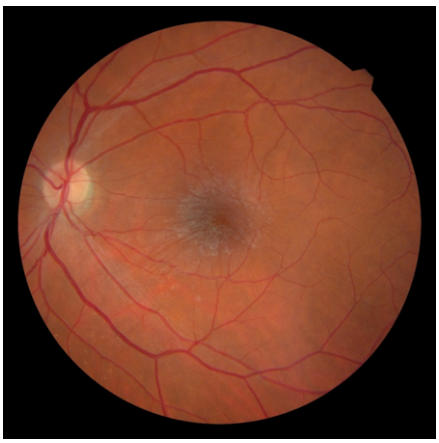


Figure 23: LE colour fundus

Looking at Figure 23, it is pretty clear that there is macula pucker and some form of ERM present. However, there will be many cases that are not obvious or even visible on the photo.

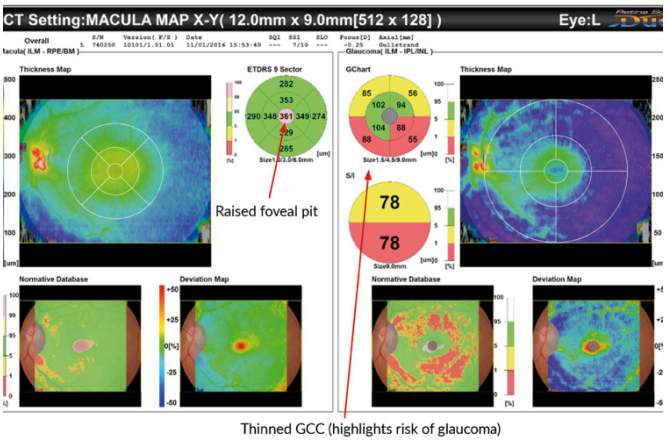


Figure 24: LE macula map

The macula map in Figure 24 shows a raised foveal pit. This may be normal, but it is unlikely. It should be noted that there is a clear sign there could be a risk of glaucoma — it would be worth checking the disc map and perhaps conducting progression analysis several times over 12 months.

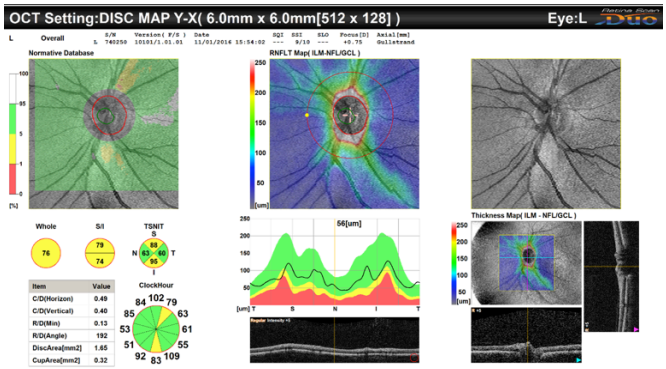


Figure 25: LE disc map

The disc map in Figure 25 confirms thinning of the retinal nerve fibre layer (RNFL) superiorly and inferiorly, meaning progression analysis over 12–24 months would be warranted to see if there is tissue loss. If there is loss over that period, then referral would be the next course of action.

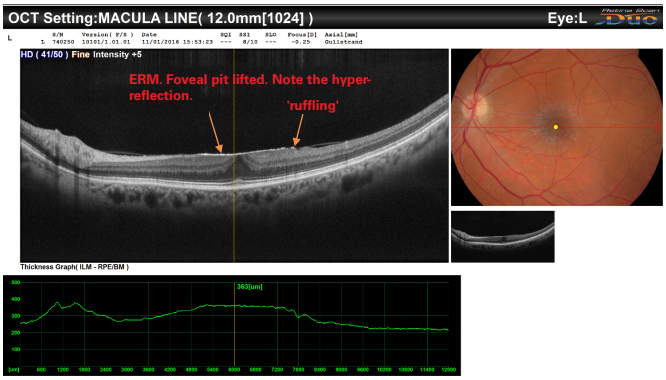


Figure 26: LE B-scan

In Figure 26, you can see the hyperreflective ERM and the subtle 'ruffling' of the RNFL often seen on B-scans just temporal to the fovea.

Some ERMs 'break off' and traction is released. This usually improves the VA and distortion, though initially, there can be glare and floaters experienced by the patient. ERMs can lead to pseudo-macular holes and lamellar holes; though rarely full thickness macular holes.

Case 2:

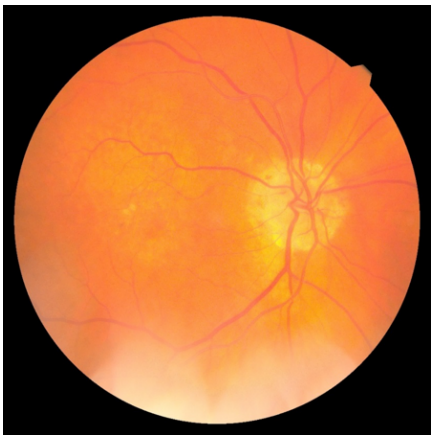


Figure 27: RE colour fundus

The fundus image in Figure 27 shows peripapillary atrophy (PPA) of the disc and what appears to be retinal thinning and some drusen. Apart from arteriovenous nipping, there is little else obvious from this photo.

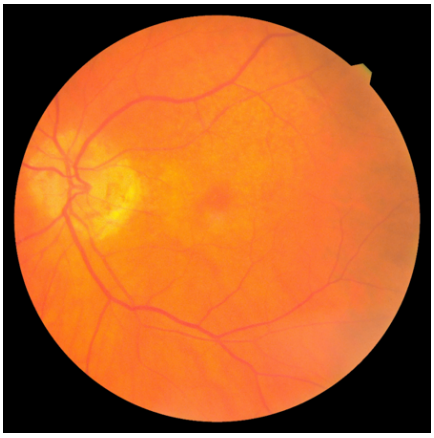


Figure 28: LE colour fundus

Again, PPA and some atrophic changes seem to be all that is present in Figure 28.

Figure 29: RE B-scans

Figure 29 shows an ERM that looks to be peeling off temporally and superiorly (probably due to gravity). It is likely that this will reduce traction over time. Detached ERMs are often slowly reabsorbed, though they may linger and cause floaters and glare. As mentioned, most ERMs occur after patients have had PVDs. ERMs tend to be overgrowth of glial tissue from the RNFL through the ILM. Other causes can be from vitreous haemorrhages and can be fibrovascular and far more destructive and troublesome.

Case 3:

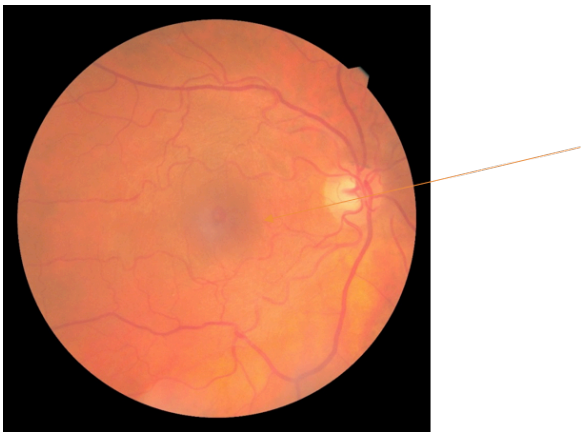


Figure 30: RE colour fundus

Looking at Figure 30, it is clear that there is an ERM, despite it being affected by lens opacities. Note the wispy, frosty appearance around the edge of the macula.

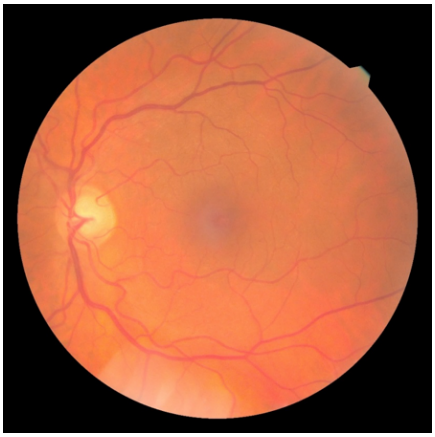


Figure 31: LE colour fundus

The colour fundus image for the LE, seen in Figure 31, does not show any obvious ERM. We may, however, be concerned by the asymmetric discs and bilateral cupping.

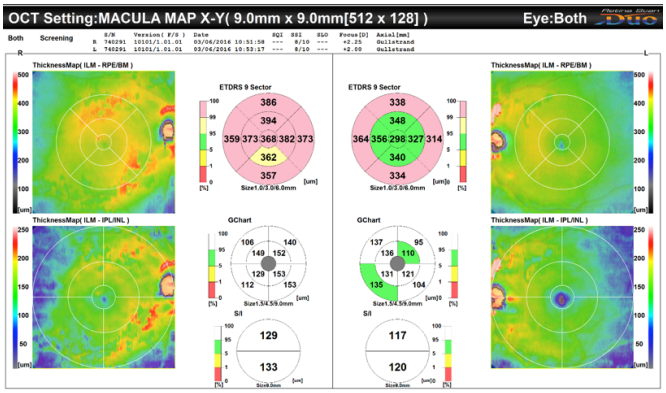


Figure 32: Bilateral macula maps

The macula maps (Figure 32) show increased retinal thickness in both eyes (RE more so) with irregularity of the thickening on the thickness map for the RE in particular.

Both the rough B-scans (Figures 33 and 34) show a pseudo-macular or lamellar hole in the RE, and what may be a lamellar hole in the LE.

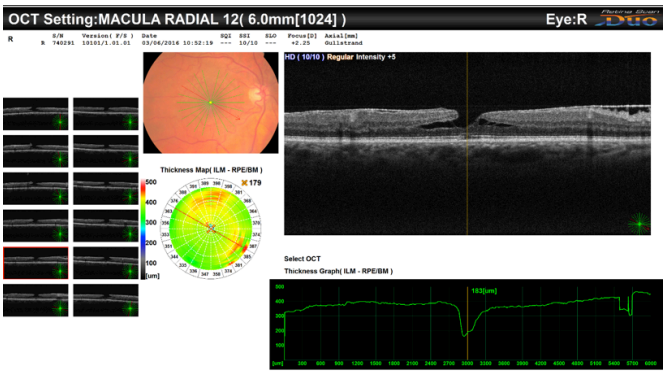


Figure 33: RE B-scans (lamellar hole)

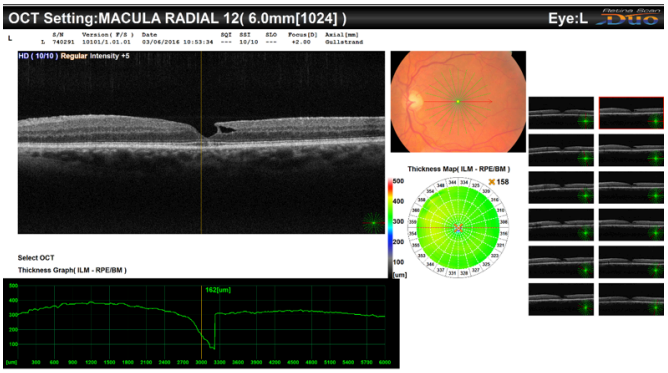


Figure 34: LE B-scans (lamellar hole)

Pseudo-macular hole

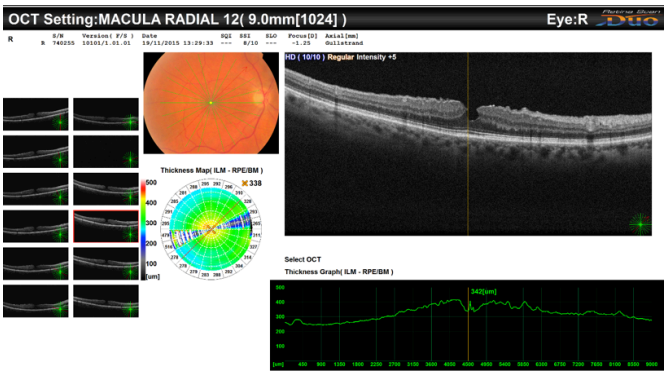


Figure 35: RE B-scan

The B-scan in Figure 35 shows an ERM causing a pseudo-macular hole. When viewing this with a Volk lens, it may sometimes be mistaken for a macular hole, though the VA will generally be too good in pseudo-hole cases. Again, unless the VA is dropped sufficiently, most eye departments will not want referrals for such cases as the treatment involves risks that are not worth taking unless the patient is suffering vision loss and significant distortion.

Pigment epithelial detachment

Another pathology you will encounter quite often is a pigment epithelial detachment (PED). With OCT, clinicians tend to see far more PEDs than without. Often they are completely asymptomatic and usually do not require treatment, but may need monitoring short-term.

OCT does make clinicians realise how many minor and generally innocuous lesions they miss on a regular basis. They must remember this is not a limitation of their ability, but merely a limit to their own visual resolution and the viewing technology they had prior to the OCT. Clinicians will normally have other signs and symptoms to help them locate and diagnose many lesions that

aren't necessarily visible. The OCT is just another very useful tool in assisting clinicians in finding lesions, but is not always the 'panacea' some professionals consider it to be.

Case 1:

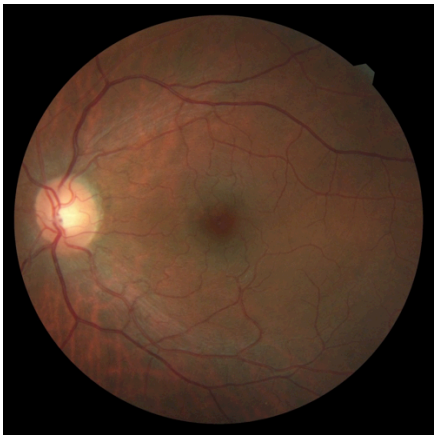


Figure 36: LE colour fundus

Looking at Figure 36, this looks to be a perfectly normal fundus.

However, when we view the B-scan in Figure 37, we can see a small, discrete PED. From experience, macula clinics tend to advise patients to avoid heavy lifting and extreme sports (particularly bungee jumping!). Anecdotally, an ophthalmologist suggested that if the PED increased in size over an eight week period, and was not close to or at the fovea, then laser treatment might be considered to avoid the PED spreading and becoming a larger detachment. If the PED is near the fovea, monitoring is the best option. The patient should be advised to use an Amsler (and return if distortion occurs or increases).

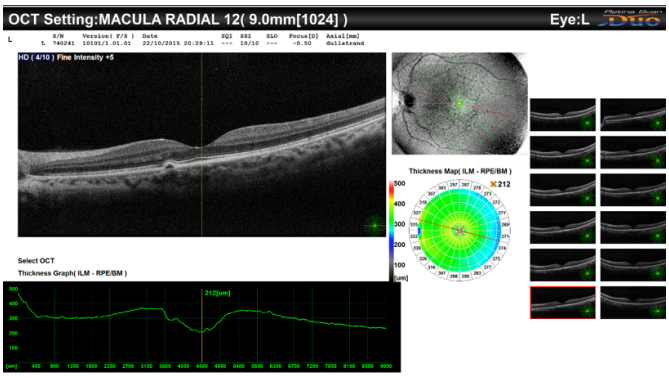


Figure 37: LE B-scans

Vitreomacular traction

One of the main causes of full thickness macular holes is vitreomacular traction (VMT). This is caused by an unusually strong adherence of the posterior vitreous face to the retina, especially at the fovea. VMT is more common in women (65%) and typically occurs between 60 and 80 years of age. Patients normally present with reduced vision and metamorphopsia. Examination often reveals an ERM and there may be separation of retinal layers with cystic spaces. Referral is advised due to risk of full thickness macular holes and vitrectomy surgery is the normal course of action.

Case 1:

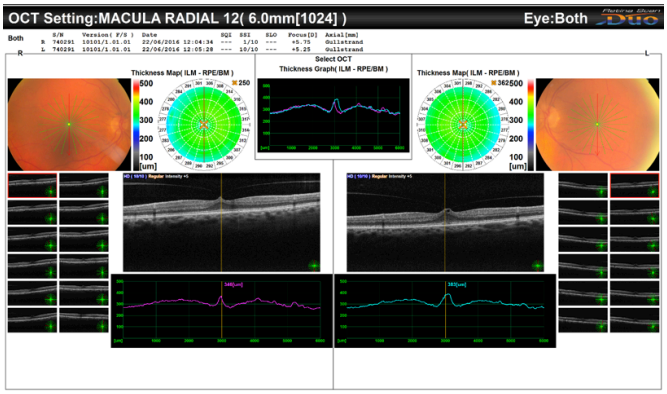


Figure 38: Bilateral radial B-scans

Often, as seen in Figure 38, you will find patients with bilateral VMT. Looking at these images, there is only a small area of adherence and the likelihood is that there will be bilateral PVDs with minimal, if any, damage to the fovea. An operculum may be left, where a small amount of ILM tissue is left on the posterior hyaloid face, and this can lead to visual disturbance lasting weeks or sometimes months.

Case 2:

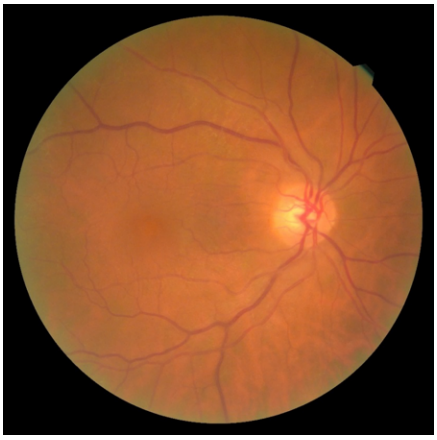


Figure 39: RE Colour fundus

It is not obvious from Figure 39 that there is any VMT present.

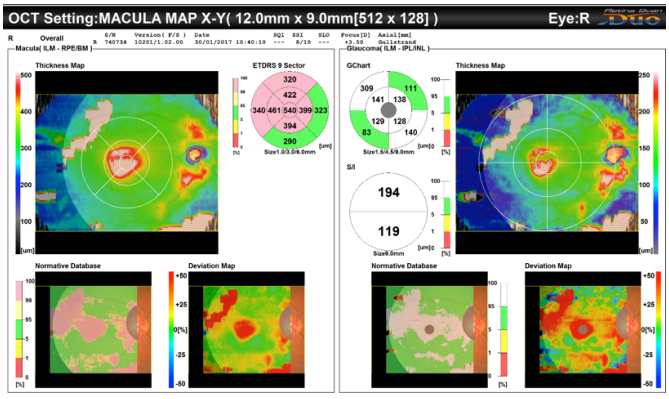


Figure 40: RE macula map

Figure 41: RE multi scans

The B-scans (Figures 40 and 41) show an alarming amount of traction with significant tractional retinoschisis. The spaces are all filled with serous fluid (no shadow). Judging by the large area of traction, there is considerable risk of this becoming a macular hole.

Case 3:

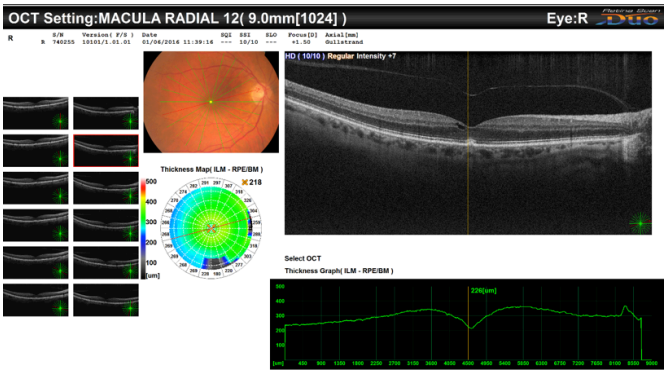


Figure 42: RE multi scans VMT present

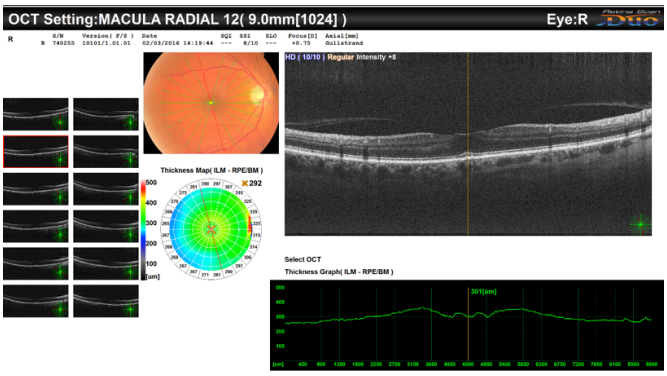


Figure 43: RE multi scans post-VMT

It is clear from this example (Figures 42 and 43) that PVD occurred with some lasting effect on the fovea. Ultimately, vision settled to normal with this patient.

Full thickness macular hole

As mentioned, the most common cause of a macular hole is VMT. There are circumstances where cystoid macular oedema (CMO) can 'burst' out of the foveal pit. Where traction has caused the hole, it may not be a complete full thickness hole or may be highly distorted. Where CMO has caused the hole, it tends to be central and is the classic 'punched out' hole with cystic spaces in the retina adjacent to the hole.

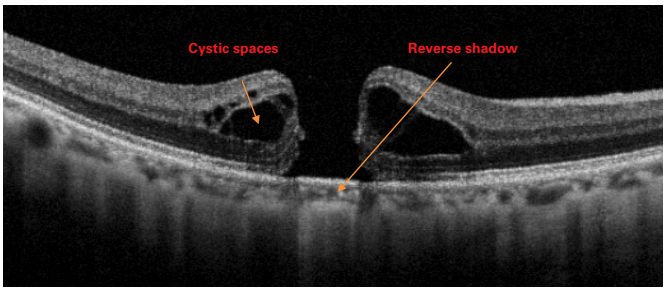


Figure 44: Full thickness macular hole

Central serous retinopathy

Primarily caused by excess levels of cortisol (a stress hormone), central serous retinopathy (CSR) is found most often in males aged between 20 and 40 years old. Sometimes, the term 'alpha male' is used to describe the individuals that often present with the condition. However, women and older people are also susceptible to the condition and in older patients, we must be cautious about both the cause and also the potential that CSR is caused by neovascular changes in the choroid.

Case 1:

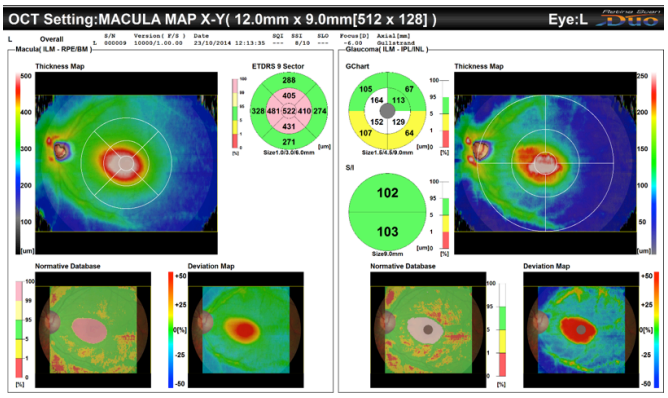


Figure 45: LE macula map showing round raised lesion centrally

In Figure 45 we can see a raised lesion on the thickness map that is round and regular, which implies that the cause of thickening is fluid. Being central, we can assume it may be CSR or some form of wet maculopathy. The patient has experienced a hyperopic shift in prescription and has mild metamorphopsia. The patient is male and is 41 years old. From the presenting information, we can safely assume CSR. He only had sypmtoms in his left eye.

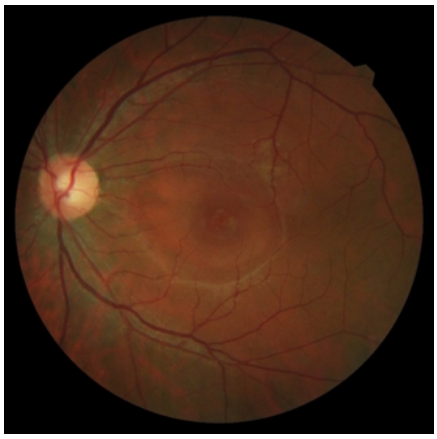


Figure 46: LE colour fundus

From the fundus photo (Figure 46), it is clear that there is notable central oedema present.

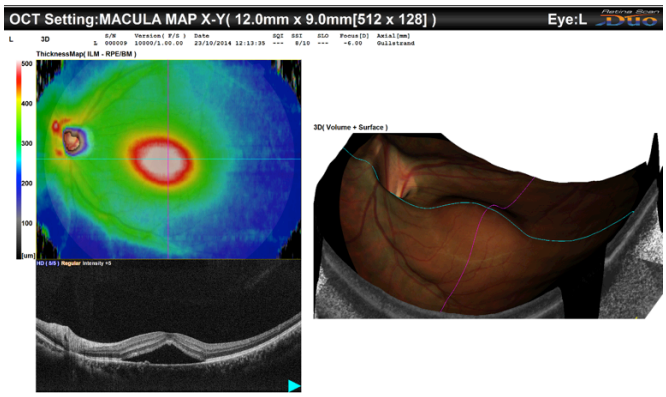


Figure 47: LE 3D colour fundus overlay

A 3D colour fundus overlay (Figure 47) is a useful way of describing CSR to the patient and can help explain why their vision has changed and what we hope will happen over time with or without treatment.

Case 2:

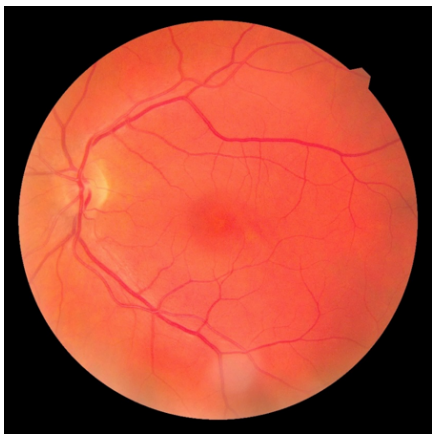


Figure 48: LE colour fundus

The colour fundus image in Figure 48 appears fairly normal, with subtle disturbance centrally (though this could be easily missed).

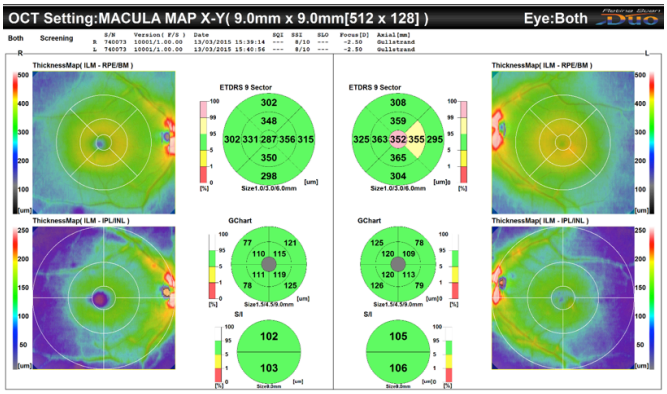


Figure 49: Bilateral macula maps

From the bilateral maps (Figure 49), we can see that the foveal area in the LE is thicker than in the RE. This is unlikely to be normal and thus prompts us to investigate further.

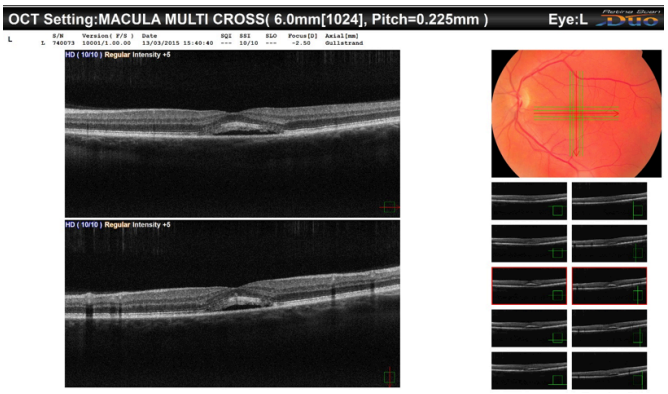


Figure 50: LE B-scans

Looking at the B-scans (Figure 50) in higher resolution, we can see this is a small CSR. If this is the first presentation from the patient, then monitoring may be an option to see if it changes. However, many local eye departments request referral on any CSR presentation. My advice would be to check with your local ophthalmologists about referral criteria for this, as many will only treat a chronic CSR of persistent duration over 3 months. Patients that are older may also have CSR, but may need fundus fluorescein angiography to differentiate this from AMD.

Cystoid macular oedema

Cystoid macular oedema CMO most commonly presents several weeks after intracapsular phacoemulsification cataract surgery and intraocular lens implant. There are many other cases of CMO which are not post-operative and, ultimately, any oedema which is cystic and occurring at the macula is essentially CMO! The fluid normally accumulates around the IPL/INL layers and is often innocuous when it presents with small cysts. If the cysts accumulate into large spaces however, then there is risk of lamellar or full thickness macular holes.

Apart from cataract surgery, other causes include BRVO, YAG laser treatment, intraocular inflammatory conditions, pharmacological reactions, ERMs, VMT and other retinal vascular disease.

Case 1:

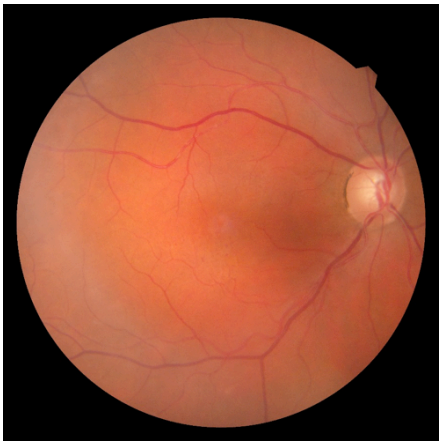


Figure 51: RE colour fundus

You need to look closely to see any obvious signs of CMO in Figure 51. There are some potential vascular changes which may be the cause of the CMO in this case.

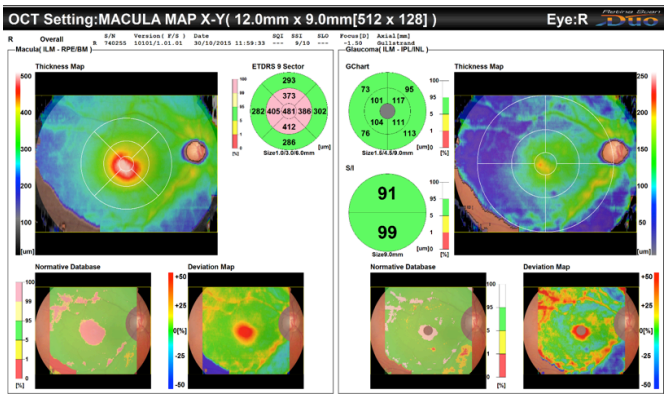


Figure 52: RE macula map

From the macula map in Figure 52, we can see a central round, regular raised area, implying that an oedema is the cause of the thickening.

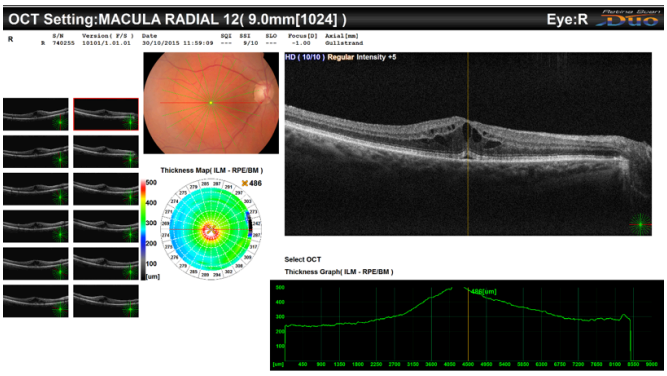


Figure 53: RE B-Scans

The B-scans of the RE (Figure 53) show there pockets of oedema centrally and within the inner retinal layers. There is also a small pocket of SRF present in this case. Again, it would be good if clinicians make themselves aware of local protocols regarding referral criteria for specific conditions. This adds strength to optometrists' status in primary care and assisting secondary care and it reduces over-referrals and strain on NHS outpatients.

Adult vitelliform dystrophy

Adult vitelliform dystrophy is a reasonably common condition that results in progressive central visual loss, typically manifesting in symptoms around the sixth decade of life. Not to be mistaken with Best's (juvenile vitelliform dystrophy), many patients don't suffer severe visual loss in many cases where onset is very late and progression is slow. The classic presentation is with the 'egg yolk' lesion on the colour fundus image. However, there are many occasions where OCT changes are visible long before this stage.

There is no known treatment for adult vitelliform dystrophy, but intravitreal injections of either ranibizumab or bevacizumab may be effective in the short-term.

Case 1:

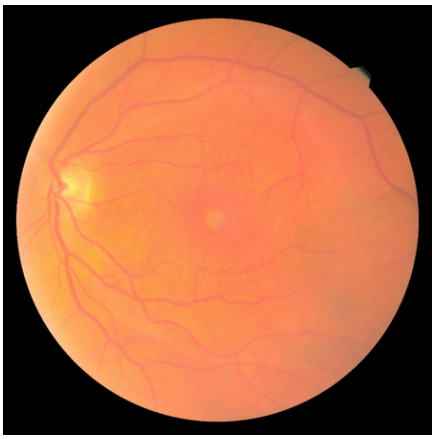


Figure 54: LE colour fundus

Note the central 'egg yolk' on the colour fundus image in Figure 54. In adult vitelliform dystrophy, the lesion is almost always directly central at the fovea.

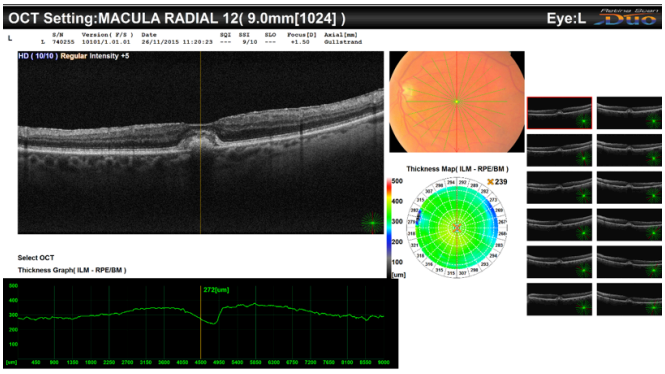


Figure 55: LE radial scans

Likewise, the central raised lesion at the fovea on the B-scan is very typical of adult vitelliform dystrophy and so it is generally easy to differentiate from other conditions (Figure 55). In this particular case, there is also an ERM present.

Case 2:

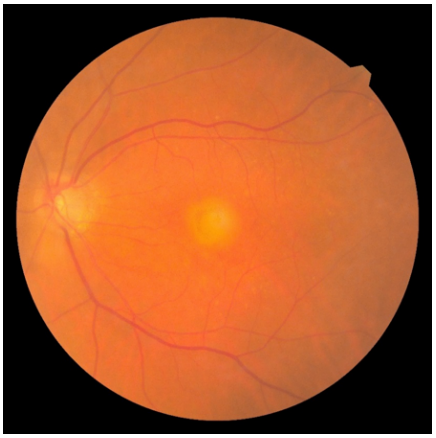


Figure 56: LE colour fundus

Once again, we can see the classic 'egg yolk' lesion in Figure 56.

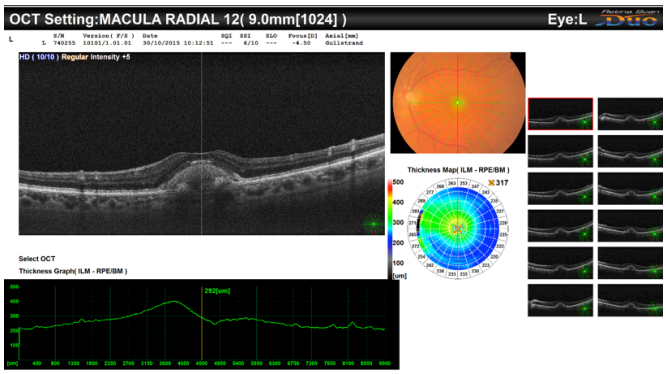


Figure 57: LE radial scans

Again, we can see how typical this lesion is from the B-scan in Figure 57.

Retinal detachments

Many retinal detachments and tears are too peripheral for OCTs to spot; this highlights the continued need for clinicians to use slit lamp BIO or other peripheral fundus viewing techniques. However, there are occasions where more central detachments occur and these are easy to spot with an OCT.

Case 1:

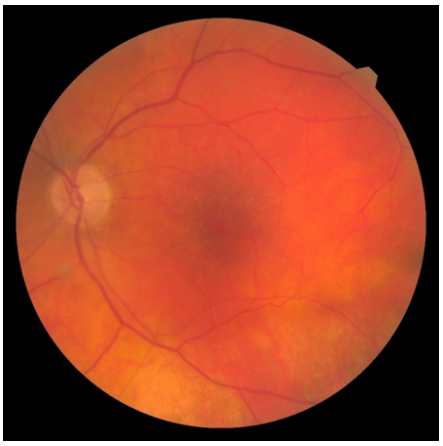


Figure 58: LE colour fundus

Apart from a subtle central ERM, there is no obvious sign of any detachment in Figure 58.

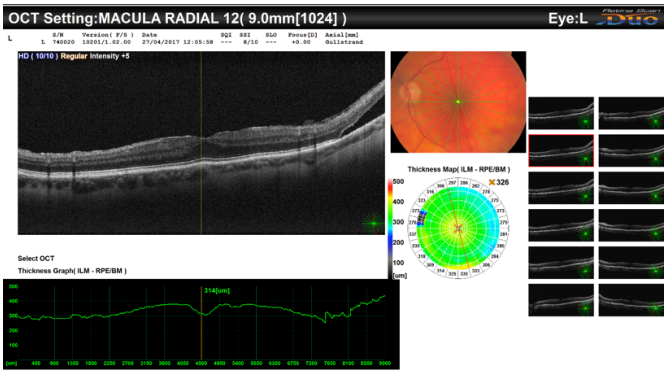


Figure 59: LE radial scans

Figure 59 clearly shows a detachment (neurosensory) temporal to the fovea in the LE. We do not know how far out this stretches, but if the patient were asymptomatic, we are immediately aware that we have to conduct dilated peripheral fundoscopy to examine further prior to referral.

Posterior vitreous detachments

Posterior vitreous detachments (PVDs) are extremely common and prevalence in the general population is around 25% — this lifts to over 50% in the over 75s. The two main risk factors for PVD are age and refractive error, with a higher prevalence in high myopes.

In most cases, apart from floaters and the initial symptoms of 'spider webs' and occasional flashing lights, PVDs are innocuous and do not pose any serious prognostic concerns. In many cases, post PVD, we eventually see the development of ERMs. However, where a PVD exists, it is always worth checking for retinal tears, microhaemorrhages and other damage to the retina and vitreous. The most common sign of a full PVD is a Weiss ring.

PVDs are generally easy to see on OCT — sometimes false colour scans can highlight vitreous lesions more clearly.

Case 1:

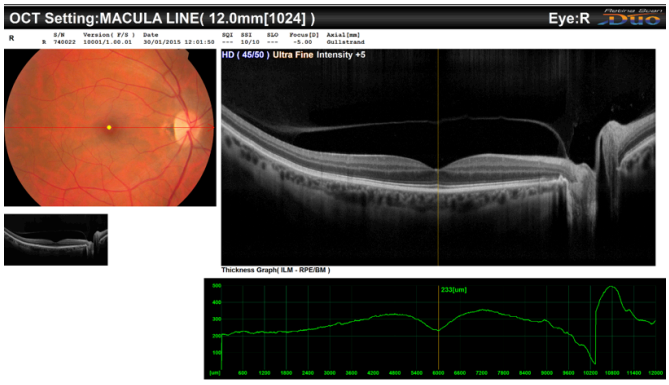


Figure 60: RE radial scans

Figure 60 shows very evident PVD with potential traction temporally, but still well attached at the disc.

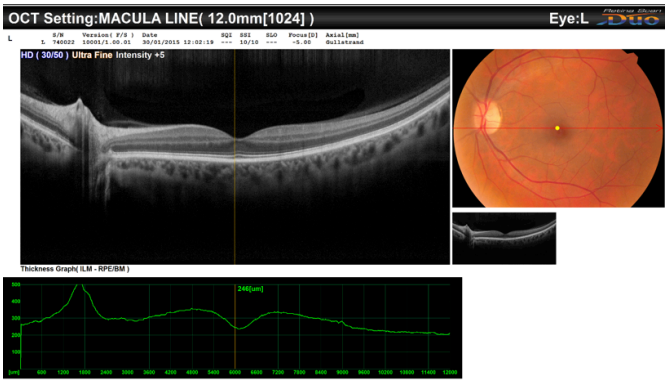


Figure 61: LE radial scans

In comparison, in Figure 61, it is clear the vitreous is still firmly attached at the fovea in the LE.

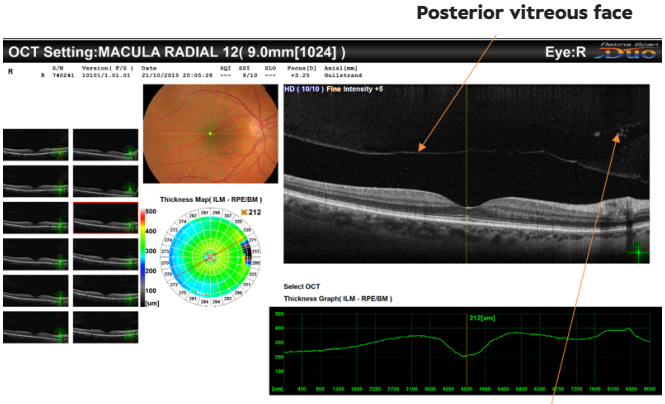


Figure 62: RE radial scans

Another example of a PVD is shown in Figure 62. In these scans, we can now easily see the posterior vitreous face fully lifted off the fovea and the disc. There is no operculum centrally, but you can see some vitreal floaters that are part of a subtle Weiss ring.

Pigment clumps

Large accumulations of pigment can cast shadows. Looking at the B-scan in Figure 63, we can see a large amount of pigment casting a shadow. The pigment is also very evident in the colour fundus picture (Figure 64).

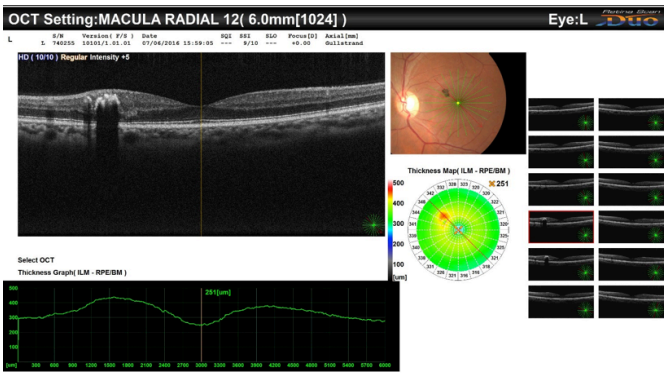


Figure 63: B-scan of pigment clump

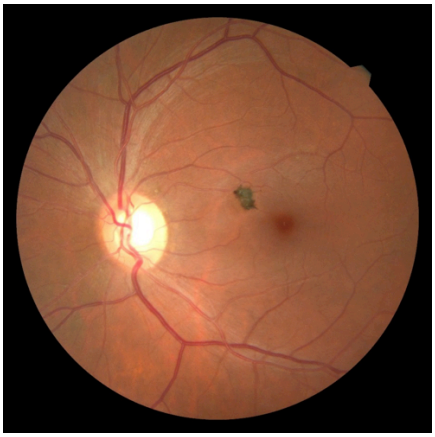


Figure 64: Pigment clump photograph

Summary of main learning points

- Understand referral criteria
- Drusen and geographic atrophy in dry AMD and SRF in more advanced cases
- Switch from dry to wet AMD (exudative) with neovascularisation and leakage
- Difference between oedematous and tractional lesions
- General ability to spot the main medical retina lesions and identifying risk