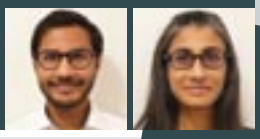

COMPETENCIES COVERED

DISPENSING OPTICIANS
Communication, Standards of Practice, Ocular Abnormalities

OPTOMETRISTS
Standards of Practice, Ocular Disease

This CET has been approved for one point by the GOC. It is open to all FBDO members, and associate member optometrists. The multiple-choice questions (MCQs) for this month's CET are available **online only**, to comply with the GOC's Good Practice Guidance for this type of CET. Insert your answers to the six MCQs online at www.abdo.org.uk. After member login, go into the secure membership portal and CET Online will be found on the L menu.
Questions will be presented in random order. Please ensure that your email address and GOC number are up-to-date. The pass mark is 60 per cent. The answers will appear in the June 2021 issue of *Dispensing Optics*. The closing date is 7 May 2021.

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C-76826 Approved for one CET Point

Getting to grips with OCT Part 2

By Prashant Shah BSc(Hons) MCOptom PGDipOphth DipClinOptom and Yashita Shah BSc(Hons) PGDipOphth

In Part 1 of this article (*Dispensing Optics* January 2021), we discussed the history of optical coherence tomography (OCT), its benefits, limitations and application in practice. Normal features of the retinal anatomy and scans were also described.

As the use of OCT increases in optometry, it will become imperative that all staff members – from optometrists to dispensing opticians (DOs) – understand its use and application in practice to recognise eye conditions and manage patients accordingly. As DOs are well placed in the optical setting to take scans, their role is vital in bringing any abnormal scans to the attention of the optometrist during the initial screening.

In Part 2, we will focus on some common conditions seen in practice, where OCT is useful in diagnosis and management. Each eye condition will not be discussed in extensive detail, though the reader can refer to the references and reading list if they wish for more information.

VITREOMACULAR TRACTION AND POSTERIOR VITREOUS DETACHMENT

A posterior vitreous detachment (PVD) occurs when the vitreous gel shrinks and liquifies. It can then detach from the retina at points of vitreoretinal adhesion, which are the peripheral retina, macula and optic nerve. The symptoms of PVD can be flashing lights, floaters, shadows, cobwebs or a 'veil' in the vision.

While the majority of PVDs occur without complications, potential complications include retinal tears, retinal detachments, macular holes and epiretinal membranes. The incidence of PVD is known to increase with age[1], and risk factors associated with earlier onset of PVD include high myopia, ocular trauma, ocular surgery, aphakia, intraocular inflammation, diabetes and postmenopausal women[2].

Vitreomacular traction (VMT) occurs when there is persistent vitreous attachment at the central macula following an incomplete PVD, causing traction and macular distortion. Vitreomacular adherence remains at the centre of the macula while there is adjacent vitreoretinal separation.

The clinical picture in VMT is variable, with symptoms ranging from mild blurring and distortion to severe decrease in visual acuity and distortion[3]. If the patient is relatively asymptomatic and VMT is a chance finding, they should be advised to self-monitor with an Amsler grid and be reviewed sooner to

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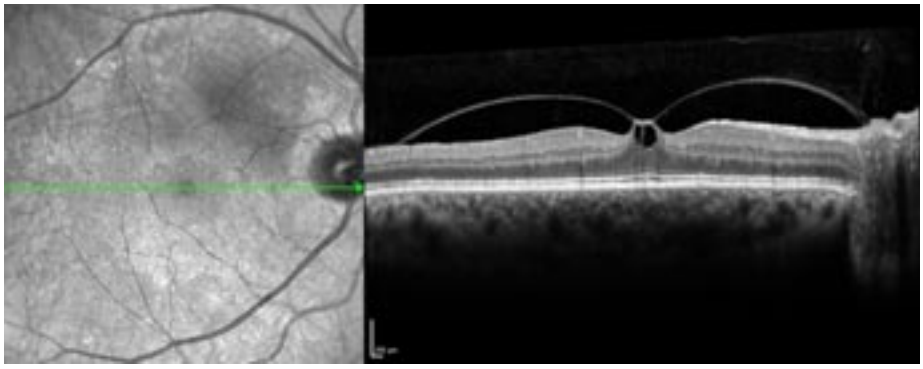

Figure 1. OCT of a right vitreomacular traction (note: on infrared fundus images: infrared light is absorbed by intraretinal, sub-retinal and sub RPE fluid. Fluid appears as a darker region on the infrared image. OCTs taken along green arrow)

see if spontaneous resolution or progression occurs. Spontaneous resolution of VMT occurs in approximately 50 per cent of cases[4].

Figure 1 represents the OCT scan of a 70-year-old female attending with symptoms of intermittent visual disturbance in the right eye for one week. Right visual acuity was 6/7.5 with slight central distortion reported on the Amsler chart.

As seen on **Figure 1**, the infrared fundus image appears normal. The OCT B-scan shows the posterior vitreous face attached to the inner retina at the fovea. The vitreous face is a thin hyper-reflective band, which has separated from the retina adjacent to the fovea but is gripping on the fovea centrally. Due to this traction, two cystic spaces are visible. Since the outer retina and ellipsoid zone at the fovea have remained intact, the patient has maintained relatively good visual acuity.

VMT can be subtle and difficult to detect on ophthalmoscopy, especially in the early stages. Therefore, without an OCT in practice, VMT would not have been identified as the cause of the patient's symptoms. The patient would probably have been referred as she had unexplained symptoms; instead, having an OCT allowed the optometrist to effectively manage this in practice by explaining the condition to the patient, giving reassurance and monitoring on a regular basis.

An Amsler chart was issued to the patient for self-monitoring, with advice to seek urgent attention if symptoms worsened. The VMT resolved within six months into a complete PVD without any residual traction on the fovea, and the patient's visual acuity returned to 6/6.

EPIRETINAL MEMBRANE

An epiretinal membrane (ERM) is a sheet of fibrous tissue that forms in between the vitreous face and the internal limiting membrane of the retina. The clinical appearance of ERM can vary depending on its thickness and associated retinal traction. A thin translucent membrane is often referred to as cellophane maculopathy, and is seen on fundus examination as an irregular light reflex or sheen over the macula. As the membrane thickens and contracts, it creates retinal folds which is known as macular pucker[5].

Unlike cellophane maculopathy, where vision remains relatively unaffected, macular pucker typically causes reduction in vision to 6/12 or worse, with associated metamorphopsia[5,6]. In severe cases, ERM is associated with retinal thickening and oedema.

PVD has a critical role in the pathogenesis of an ERM[5]. ERM can be a precursor to VMT and subsequent pathologies such as macular and lamellar holes. Other causes for ERM development include retinal surgery, retinal vascular disease, intraocular inflammation and ocular trauma.

Asymptomatic patients do not require referral and can be managed by regular monitoring with OCT and an Amsler chart. Patients with reduced acuity can be routinely referred to an ophthalmologist for vitrectomy and an ERM peel. The visual outcome following surgery varies depending on pre-operative visual acuity, with improved visual outcome achieved in patients who had a better initial acuity[6]. In some cases, it can take up to one year for the visual acuity to settle down following surgery.

Figure 2 shows the right fundus and OCT of a 68-year-old female who attended with symptoms of mild distortion when reading. Visual acuity measured as RE 6/7.5, N6 and LE 6/6, N5. Amsler showed slight paracentral distortion and no scotomas.

The infrared fundus image displays mild retinal wrinkling. Overall, the macula still has a concave profile as shown on the OCT B-scan, however, there is a hyper-reflective line on the inner retinal surface which is the ERM (depicted by a red arrow on **Figure 2**).

The ILM/RNFL are not smooth and straight; this is the retinal wrinkling. As the inner and outer retinal layers are still intact, relatively good visual acuity has been maintained. The patient was reassured and given an Amsler chart to use for self-monitoring regularly with advice to seek urgent attention if symptoms worsened.

When a patient presents with distorted central vision, OCT can be used to differentiate between conditions such as age-related macular degeneration (AMD), VMT and ERM.

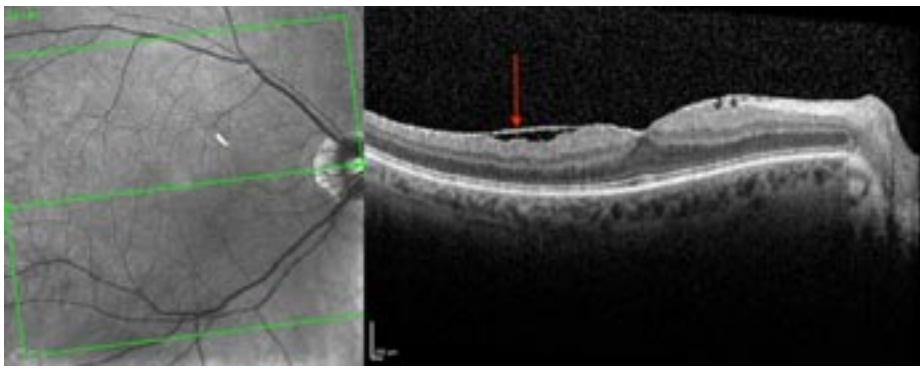

Figure 2. OCT of a right epiretinal membrane

CYSTOID MACULA OEDEMA

Cystoid macula oedema (CMO) is a painless central loss of vision occurring in

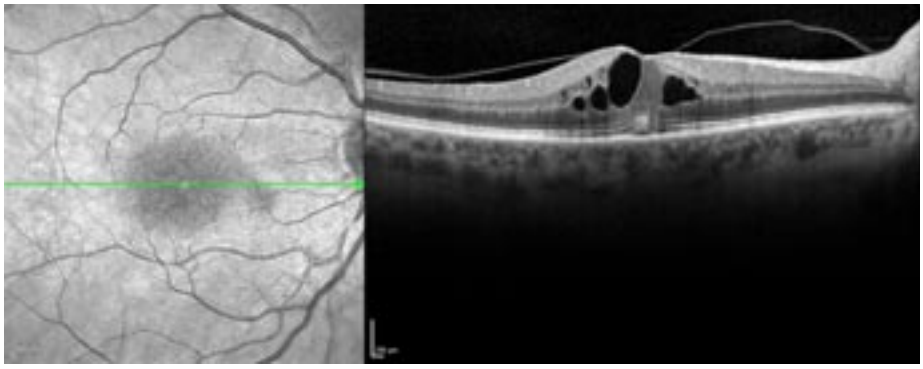

Figure 3. OCT of a right cystoid macula oedema

a variety of conditions such as uveitis, retinal vein occlusions, diabetic retinopathy and most commonly following cataract extraction[7-9].

Post-cataract CMO typically occurs four to six weeks after surgery, although it can occur months or years later[7]. Patients usually present with a drop in vision after initially having had a good post-operative visual outcome. Occasionally, the patient may report mild ocular discomfort or photophobia. There may be a slight red eye, mild anterior chamber activity and an absent or irregular macular reflex on ophthalmoscopy depending on the severity of the oedema.

Very mild cases tend to resolve without intervention within a few weeks, although persistent or severe cases can take up to several months to resolve even with treatment[8]. Common treatments are topical steroids in combination with non-steroidal anti-inflammatory drugs to manage or prevent CMO[10].

It is important to recognise that the cause of CMO in post-cataract patients may not be due to surgery alone, especially if there are co-existing conditions such as diabetes or hypertension. Therefore, it is essential to examine the fundus for other clinical signs and consider differential diagnosis, as the management for pseudophakic CMO will be different from that for macular oedema due to diabetes or vein occlusion[7,8].

Figure 3 shows the right OCT scan of a 70-year-old patient who presented for a routine eye examination following recent uneventful bilateral cataract extractions. He did not have any relevant general health issues and was not taking any regular medications. He was essentially asymptomatic, reporting only difficulty reading at near (a typical symptom post-op), which was thought to be likely due to the fact that he needed new prescription spectacles.

Good distance vision was reported, and he had begun driving again since having the cataract operation. Visual acuities were RE 6/18, N10. LE 6/7.5, N5. Binocularly 6/7.5, N5. He reported distortion on the Amsler chart with the right eye but not with the left eye.

The infrared fundus image shows irregular macula surface with ridges. Loss of the normal concave foveal profile is demonstrated on the OCT B-scan as well as an increased macular thickness. The vitreous face is still attached at the fovea, but there are medium to large intraretinal cysts, which are hypo-reflective and the fluid within the cysts are not casting shadows.

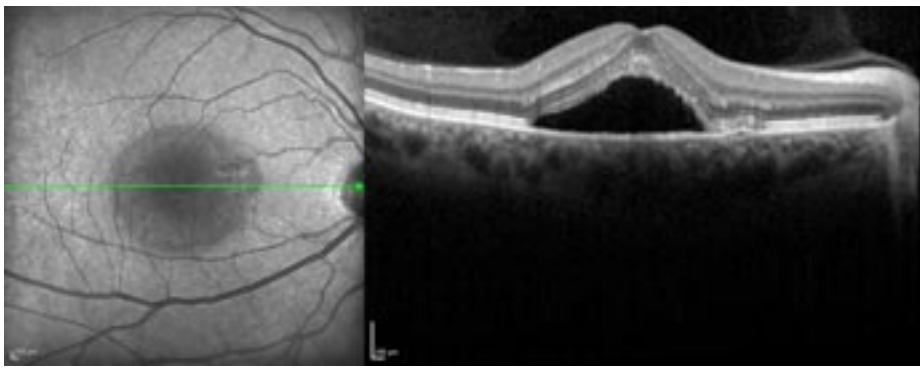

Figure 4. Right central serous retinopathy at presentation

The cysts are primarily within the outer nuclear and plexiform layers – this wouldn't have been detected by ophthalmoscopy. The ellipsoid zone at the fovea has been disrupted leading to reduced visual acuity.

The patient was consequently referred to the hospital eye service (HES) for treatment. Previously, CMO was diagnosed when vision loss was accompanied by cystoid spaces identified on ophthalmoscopy or detected with fluorescein angiography. In cases where the macula looks normal, but the vision is reduced, OCT allows the cystoid spaces in the neurosensory layer to be visualised effortlessly and CMO to be detected more frequently.

CENTRAL SEROUS RETINOPATHY

Central serous retinopathy (CSR) is characterised by detachment of the neurosensory retina from the retinal pigment epithelium (RPE), classically affecting young to middle-aged men with a Type A personality[11]. CSR can be aggravated by stress, untreated hypertension, high alcohol intake and corticosteroid use[12]. The exact cause of CSR is unknown – although it has been linked with increased choroidal vascular and RPE permeability[13]. Differential diagnosis of CSR includes choroidal neovascularisation/exudative AMD and serous RPE detachment (PED).

Typical symptoms of CSR include unilateral blurred vision/distortion and the patient may describe a dark spot in the middle of their vision. A hyperopic shift in prescription may be found due to the retinal elevation from the oedema.

Spontaneous resolution and absorption of fluid occurs in most cases of CSR within three to six months, with vision returning to normal or near-normal. However, recurrence can occur in up to 50 per cent of cases[1,12]. Treatment of prolonged or chronic CSR is commonly with laser or photodynamic therapy (PDT)[13].

Figure 4 shows a 32-year-old emmetropic male who presented with complaints of blurred vision for the past three months. His symptoms appeared to be worse when looking at a VDU screen. He was a trader working for a top investment bank in the UK. His unaided vision was 6/12 in the right eye and 6/6 in the left. A +1.00DS refraction was found

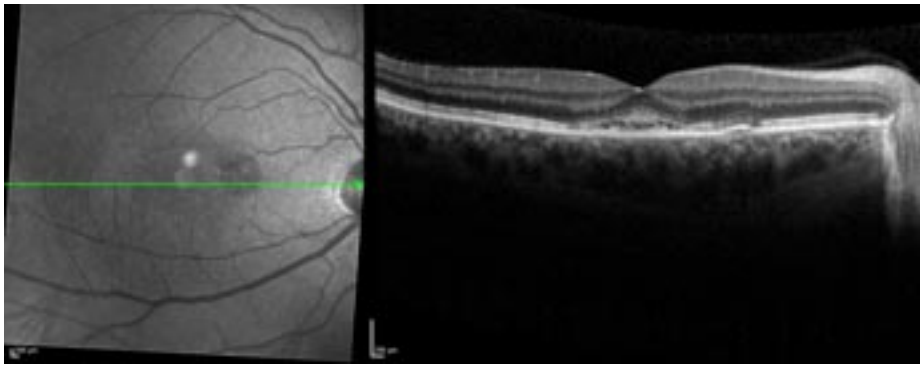

Figure 5. The same patient as Figure 4 attended three months later with resolution of central serous retinopathy

in the right eye, improving the visual acuity to 6/7.5 – and a plano result was found in the left eye. Amsler testing revealed central distortion in the right eye only; the left eye appeared normal.

The infrared fundus image shows a round grey lesion at the macula and the OCT shows a well-defined serous detachment of the neuroretina from RPE. The space between the neuroretina and RPE is hypo-reflective, indicating the presence of serous fluid as it does not cast a shadow on the retina below. There is no intraretinal or sub RPE-fluid.

The condition was explained to the patient and reassurance given. Despite being aware that the hyperopic refractive shift was due to the subretinal fluid, which would improve as the fluid resolved, the patient insisted on having a spectacle prescription. Against recommendation, a spectacle prescription was dispensed – in this case as a temporary measure to allow the patient to carry on working more comfortably. An Amsler was also issued for self-monitoring with advice to return sooner if symptoms worsened.

A further review was arranged three months later to monitor his progress (see **Figure 5**) at which point the vision in the right eye had improved to 6/7.5. The OCT foveal profile in **Figure 5** appears more normal. There is considerably less sub-retinal fluid, and the neuroretina and RPE have nearly re-joined corresponding to the improved vision. The infrared fundus image shows a smaller grey lesion than **Figure 4**.

OCT allowed the CSR to be easily visualised, and excluded other potential causes.

FULL THICKNESS MACULAR HOLE

A true macular hole is a well-defined oval or circular lesion with loss of all inner and outer retinal layers, visible as a red punched out hole on ophthalmoscopy. It is typically seen in females in the sixth or seventh decade of life and most likely to develop in response to persistent VMT, although it can also occur in high myopes or following blunt ocular trauma[14].

Symptoms typically are gradual and unilateral, causing central blurred vision or distortion. Patients may complain of difficulty with close work or watching TV. They may also describe a dark spot in the centre of their vision. These symptoms will vary depending on the size and depth of the macular hole. Often patients are asymptomatic as the fellow 'good' eye compensates, and so they may only notice it as a chance finding when the fellow eye is closed.

Full thickness lesions can be treated surgically with vitrectomy, ILM peel and insertion of a gas bubble to seal the macular hole. Following this procedure, patients are often instructed to posture face down for a number of days. Success rates for this procedure are high with closure achieved in around 90 per cent of cases[15] and an improvement in visual acuity in 79-95 per cent of patients[16]. Prompt referral to a vitreoretinal surgeon is thus essential as earlier intervention will have a better prognosis.

A lamellar hole differs from a full thickness macular hole in that the outer retinal layers are unaffected; interestingly on ophthalmoscopy it also appears as a well-defined round circular lesion[17-19]. It characteristically has an irregular shape, with an intraretinal split but an intact photoreceptor layer on OCT. While visual acuity can be good, patients often report distortion, however, referral is not usually indicated, as surgery is unlikely to improve vision[18].

Figure 6 illustrates a 78-year-old asymptomatic female who attended for a routine eye examination. She had previously had bilateral cataract surgery and was currently taking medications for rheumatoid arthritis. Her visual acuities on presentation were 6/60, N48 in the right eye (with no further improvement using a pinhole) and 6/7.5, N5 in the left eye. A red, well-defined round hole was visible on ophthalmoscopy in the right eye, however, the patient reported a negative response to the Watzke-Allen test as well as on Amsler.

The infrared fundus image demonstrates a clear, round punched out hole at the macula. The OCT B-scan confirmed a full thickness macular hole with no inner and outer retina at the fovea. The foveal profile was completely lost and there were large intraretinal cystic spaces, which were hypo-reflective. The RPE was intact but the ellipsoid zone and ELM were absent. There was also hyper-reflectivity of the choroid centrally, known as a window defect.

This patient was referred urgently to the HES (to be seen within two weeks) to increase surgical success of macula closure. She returned three months later following surgery and her visual acuity had improved to 6/9.5.

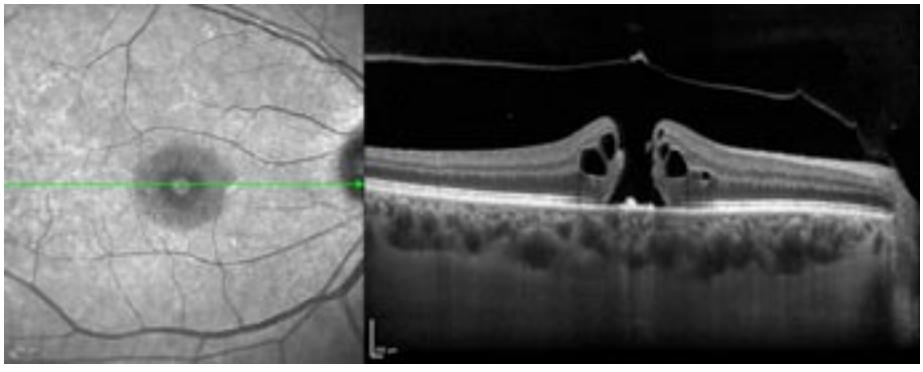

Figure 6. OCT of a right macula hole

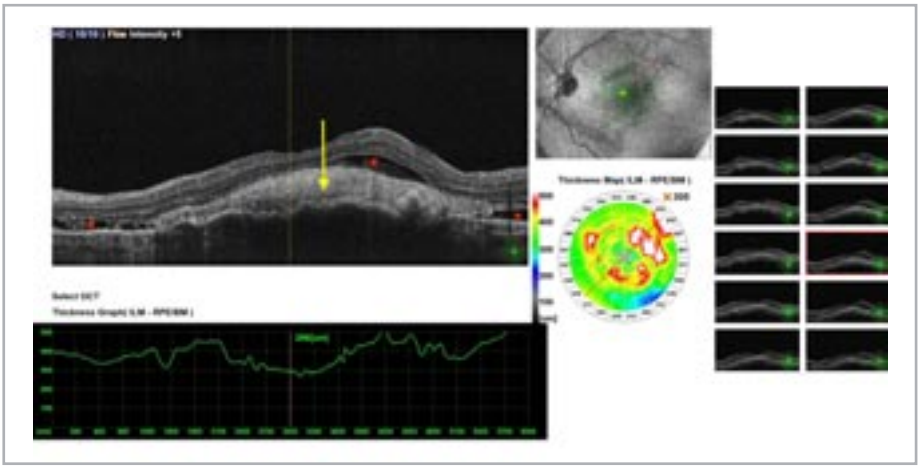

Figure 7. OCT scan showing wet AMD. The red stars indicate sub-retinal fluid which is hypo-reflective. The yellow arrow points to the CNVM which appears hyper-reflective

Unusually, this case did not present with any symptoms or positive reports on clinical tests that we would have expected the patient to respond to. Having an OCT in practice allowed the confirmation of a full thickness macula hole and prompt referral.

AGE-RELATED MACULAR DEGENERATION

Age-related macular degeneration (AMD) is the leading cause of irreversible blindness in the western world in patients over 65 years of age[20]. Half of all sight impaired or severely sight impaired registrations in the UK are due to AMD[21]. Optometrists are seeing this condition more regularly in practice due to an ageing population. Patients are also more conscious of the disease as there is a greater public health awareness of AMD. AMD can be classified into two categories: dry and wet.

Dry AMD

The 'dry' form is the most common type, accounting for approximately 90 per cent of AMD cases in the UK[1,22]. The key features of dry AMD condition are drusen and RPE changes.

Drusen are discrete yellow deposits of lipid and protein located between Bruch's membrane and the RPE that are a result of degenerative change[22]. Hard drusen are small, have sharply defined edges and appear as hyper-reflective RPE elevations on an OCT scan, whereas soft drusen have less defined edges and appear as large, rounded RPE elevations on OCT[23]. A large number of drusen is an increased risk factor for later stages of dry AMD[23].

Symptoms of dry AMD tend to be gradual, ranging from no symptoms to mild vision change and slight distortion to severe central vision loss.

Atrophic AMD is a term given to a more severe form of central vision loss due to RPE and photoreceptor degeneration. This is visible on OCT as retinal and RPE thinning, which can be localised or confluent. Choroidal hyper-reflectivity is also present in the corresponding area of retinal thinning[22].

Currently, there is no treatment available for dry AMD[1,22] and, therefore, optometrists are crucial in monitoring the condition and disease progression in the patient. Where vision appears to be deteriorating, OCT can help to differentiate between those who need fast-track referral, and those who do not require any intervention.

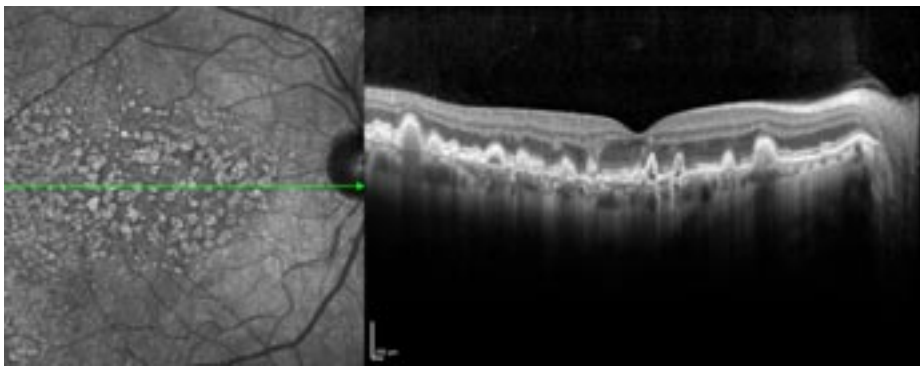

Figure 8. Drusen as seen in dry AMD on an OCT scan

Wet AMD

Wet AMD (also known as exudative or neovascular) accounts for approximately 10 per cent of AMD cases. It is characterised by the development of a choroidal neovascular membrane (CNVM)[22] which is a collection of new blood vessels from the choroid that can remain under the RPE or break through Bruch's into the subretinal space (see **Figure 7**).

These blood vessels are thin, fragile and more prone to rupture, leaking blood and fluid into the sub-RPE, sub-retinal or intra-retinal spaces. CNVM appears on OCT as hyper-reflective areas in front of, or beneath, the RPE.

Symptoms of wet AMD are usually unilateral, sudden and profound vision loss and distortion. Patients may report a dark patch or spot in their central vision. Fortunately, treatment is available for wet AMD and patients can be managed through fast-track referral pathways to minimise the risk of visual loss. The treatment is aimed at clearing the fluid and shrinking the CNVM using a course of anti-VEGF intravitreal injections[24].

Figure 8 shows the right OCT of an 80-year-old female who presented for an eye examination complaining of difficulty reading at near. Her last eye examination was 10 years ago, at which point she had just had bilateral cataract surgery. Her symptoms were worse in poorer lighting. The new spectacle refraction corrected her visual acuities to 6/15, N10 in the right eye and 6/9.5, N6 in the left. Binocularly, she achieved 6/9.5, N6.

In the infrared image, multiple round discrete lesions at the macula (i.e. drusen) can be seen. The OCT B-scan highlights the drusen as focal hyper-reflective RPE elevations, giving it a 'lumpy bumpy' appearance in contrast to a normally smooth straight line. The foveal contour is still concave but there is a possible interruption of the ellipsoid zone accounting for the reduced vision. The scan verified dry AMD and ruled out wet AMD. The patient was subsequently

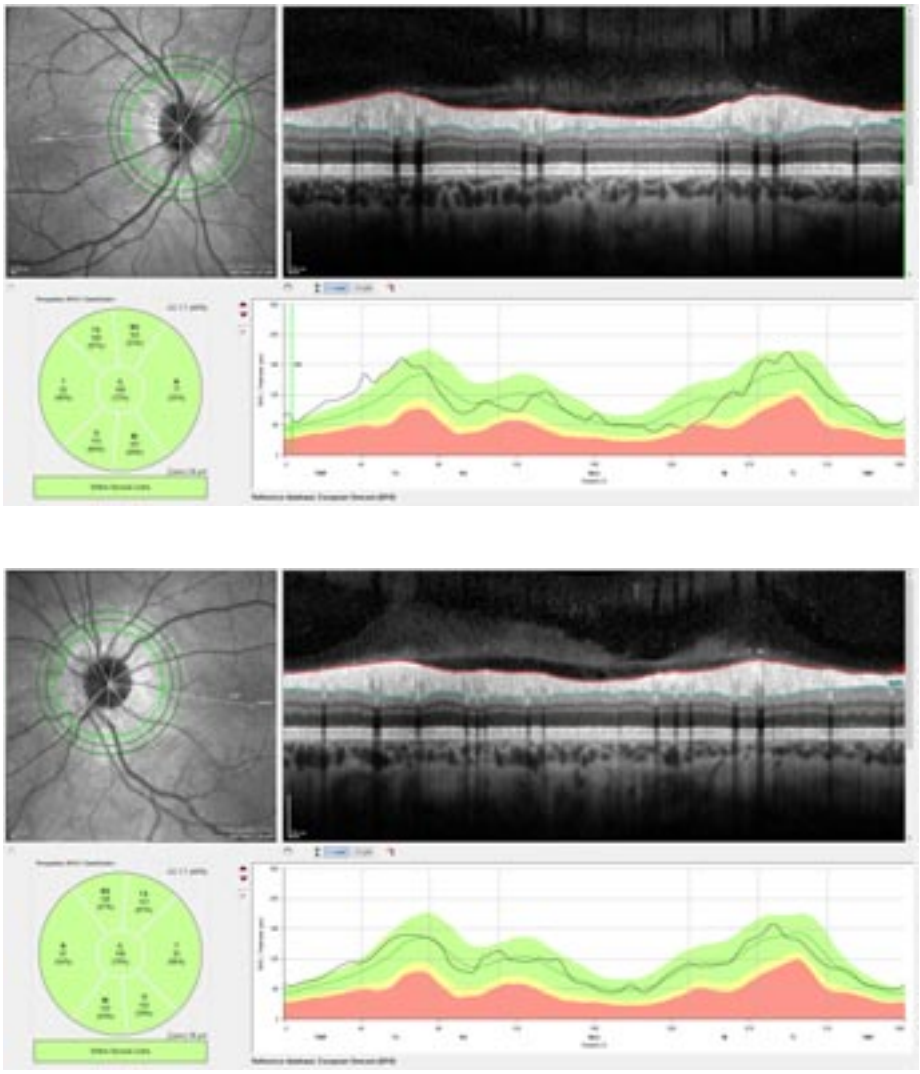

Figure 9. Optic nerve scans right and left unfolded in panoramic view has a double hump feature/twin peaks. The peaks are the thicker superior and inferior nerve fibre bundles. If these become damaged in glaucoma, an asymmetry develops between the superior and inferior nerve bundles

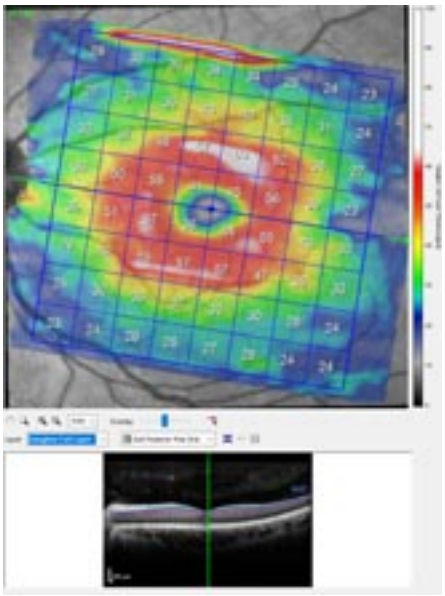

Figure 10. Ganglion cell thickness maps for the right and left eye

counselled, issued with a new spectacle prescription as well as an Amsler chart, and advised to return as soon as possible if she experienced any new or changed symptoms. She was also advised of the importance of having regular annual eye examinations.

In older patients, where the fundus can be harder to examine due to miotic pupils or media opacities, OCT allows the macula to be assessed more accurately, aiding in the early detection of wet AMD and helping to maximise the patient's chance of retaining good functional vision for longer.

Another benefit is that OCT can help differentiate between the dry and wet forms, allowing more accurate and appropriate referrals, speeding referral urgency and avoiding unnecessary referrals to the HES.

GLAUCOMA

Glaucoma is a group of optic neuropathies resulting in damage to the optic nerve and causing vision loss. Primary open angle glaucoma (POAG) is the most common form of glaucoma, characterised by raised intra-ocular pressure (IOP), leading to progressive death of the retinal ganglion cells and consequently irreversible blindness[25]. Risk factors include increasing age, positive family history of glaucoma and Afro-Caribbean ethnicity[26].

Optometrists are important in glaucoma detection, as in the early stages the condition is asymptomatic. Until recently, the diagnosis of glaucoma was based on the appearance of the optic disc, IOP measurement, visual fields and slit lamp exam.

Glaucoma detection and progression over time can be monitored by OCT objective analysis of the optic nerve head, retinal nerve fibre layer (NFL) and ganglion cell layer at the macula[25,27]. Damage or loss of the ganglion cell layer at the macula can precede visual field loss, and can be an early sign of glaucoma. OCT analysis should always be used in conjunction with other test results in glaucoma screening.

Figures 9 and 10 show the OCT maps of the retinal NFL at the optic nerves, and ganglion cell layer at the maculae of a 60-year-old male who presented for his routine annual examination. He was asymptomatic and had a mother with glaucoma. His best corrected visual acuities were 6/6 right and left. IOP measurements were 21mmHg in both eyes. The anterior chamber angle was open and visual fields were full in each eye.

The data in both maps is compared to age-matched normal. The NFL and rim of tissue at the optic nerve follows the ISNT rule (thickest inferiorly, then superiorly, then nasally and thinnest temporally). If this rule is broken, this could suggest NFL loss and possibly glaucoma. In this case, both OCT scans appeared within normal limits. The patient was reassured and recommended to continue his annual eye examinations with OCT imaging due to the positive family history of glaucoma to monitor any changes over time.

Angle-closure glaucoma is a less common form of glaucoma. It is caused by narrowing and blockage of the anterior chamber angle, resulting in raised IOP.

This can be chronic or acute. The acute form develops very quickly and requires immediate medical attention. The symptoms of angle closure glaucoma include a red painful eye, headache, blurred vision, nausea and haloes around lights. OCT imaging of the anterior chamber angle can be useful in assessing the risk of angle closure and detecting change in chronic cases.

CONCLUSION

OCT has become an indispensable diagnostic and management tool in optometry and, as such, dispensing opticians will eventually grow to be an integral part of its use.

Using OCT will enhance discovery of ocular abnormalities that may not have otherwise been visible on ophthalmoscopy. Some of these abnormalities may be clinically significant and appropriate management can therefore be undertaken promptly.

PRASHANT SHAH is an optometrist with more than 15 years of clinical experience. He holds postgraduate diplomas in ophthalmology and clinical optometry. Prashant is a regular contributor of CET articles and has had work published in the journals of both the College of Optometrists and Association of Optometrists.

YASHITA SHAH is an experienced optometrist working in independent practice where OCT is routinely used. She holds a postgraduate diploma in ophthalmology and has a keen interest in orthokeratology and dry eye.

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