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Optical Coherence Tomography (OCT) in the diagnosis and management of retinal abnormalities

Part 3: Retinal vascular occlusions

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

Part three in this series of five articles describing the use of OCT in the detection and ongoing management of a range of different retinal conditions explores retinal vascular occlusions. The article will detail the current use of OCT, and more recently OCTA in the diagnosis of retinal artery and vein occlusions. We will also look at the treatment options available and the importance of OCT in referrals to secondary care from optometric practice.

Learning objectives

Domain: Clinical practice

Registrants will have up to date knowledge in the use of OCT and OCTA for the diagnosis retinal vascular occlusions and the ongoing management and treatment of retinal vein and artery occlusions (s.5).

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

Introduction

Retinal vein occlusion (RVO) including central RVO, branch RVO, and hemi-central and hemispheric RVO is the second most common vascular cause of visual loss, surpassed only by diabetic retinopathy (see part 2). Retinal vein occlusion (RVO) is a common cause of visual loss in the UK. It is caused by an obstruction of the retinal venous system by thrombus formation and may involve the central, hemi-central or branch retinal vein (see below).[1] RVO typically occurs in middle aged and older patients (> 50 years) with equal sex distribution for both BRVO and CRVO.[2, 3] In younger patients CRVO is often referred to as a papillophlebitis and it is usually associated with inflammatory and bleeding disorders.[4]

CRVO is thought to be caused by thrombotic occlusion of the central retinal vein at or just posterior to the lamina cribrosa[5] (**Figure 1**). The cause of CRVO involves a common mechanism:[6]

- Venous occlusion/ blockage often caused by compression from a sclerotic central retinal artery or occlusion by primary vessel wall disease (degenerative or inflammatory)
- Back pressure on capillaries
- Capillary wall endothelial cell junction dysfunction
- Haemodynamic disturbance leading to leakage of plasma fluid and blood leading to macular oedema
- Non-perfusion leading to ischaemia
- These changes trigger an increase in vascular endothelial growth factors (VEGFs), which increases vascular permeability and new vessel proliferation (see below).

In BRVO, arterial compression of the vein at arteriovenous crossings is thought to stimulate thrombus formation by causing turbulent blood flow in combination with pre-existing vascular endothelial damage secondary to systemic cardiovascular risk factors.

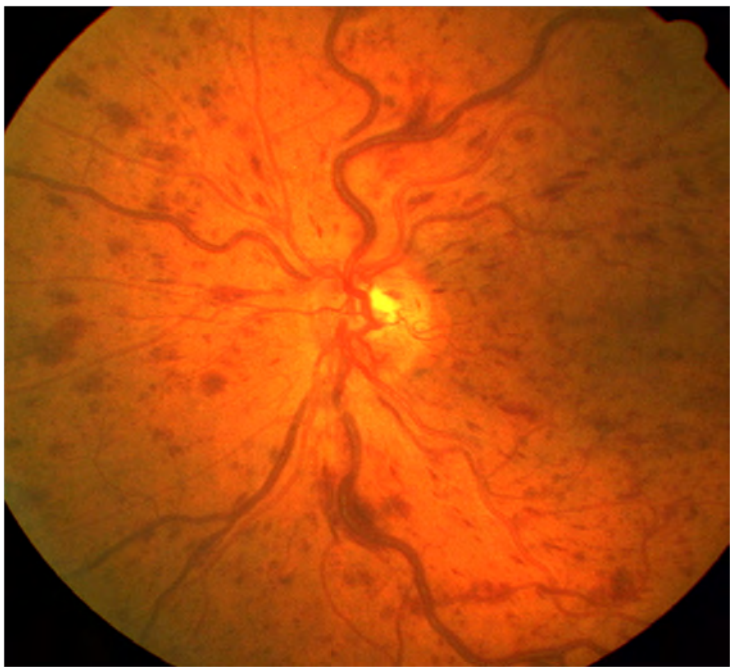

Figure 1: Non-ischaemic Central retinal vein occlusion (CRVO) - VA logMAR 0.30

Retinal vein occlusion (RVO) classification

The classification of RVO may be broadly summarised as follows:

- Central retinal vein occlusion (CRVO)
  - Non-ischaemic (**Figure 1**)
  - Ischaemic (iCRVO) (**Figure 2a - b**)

- Branch retinal vein occlusion (BRVO) may vary in size and extent considerably, depending on which localised area of the retina is drained by the occluded branch retinal vein (**Figure 3a - b**). BRVO can be sub-divided into:
  - Major BRVO (**Figure 4**) where a quarter or more of the retina is affected
  - Macular BRVO where only part of the macula is affected.

- Hemi-central retinal vein occlusion (HCRVO) is a more severe form of BRVO affecting the whole upper or lower hemisphere of the retina.[7] These can be sub-divided into:
  - Non-ischaemic HCRVO
  - Ischaemic HCRVO (**Figure 2c**)

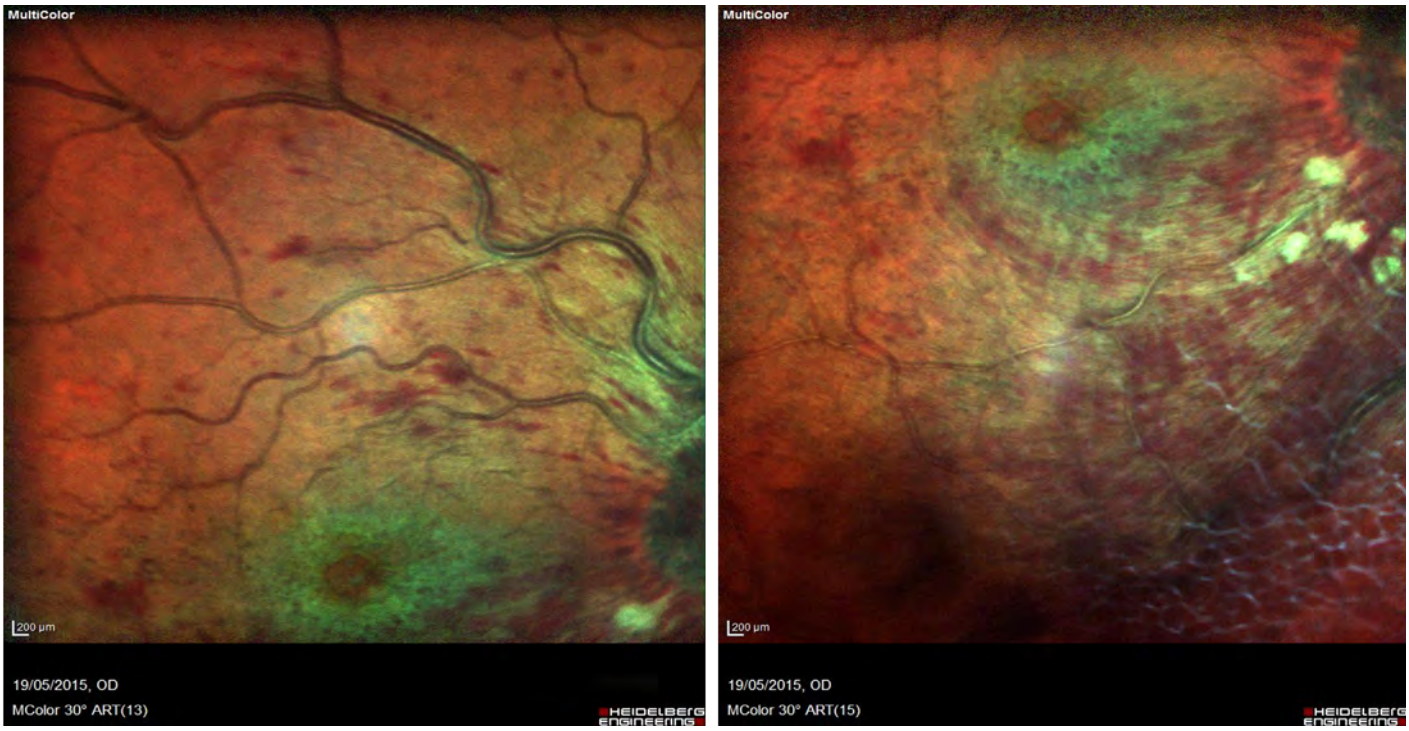

Figure 2a: MultiColor of an ischaemic central retinal vein occlusion (CRVO) - VA logMAR 1.0 (6/60)

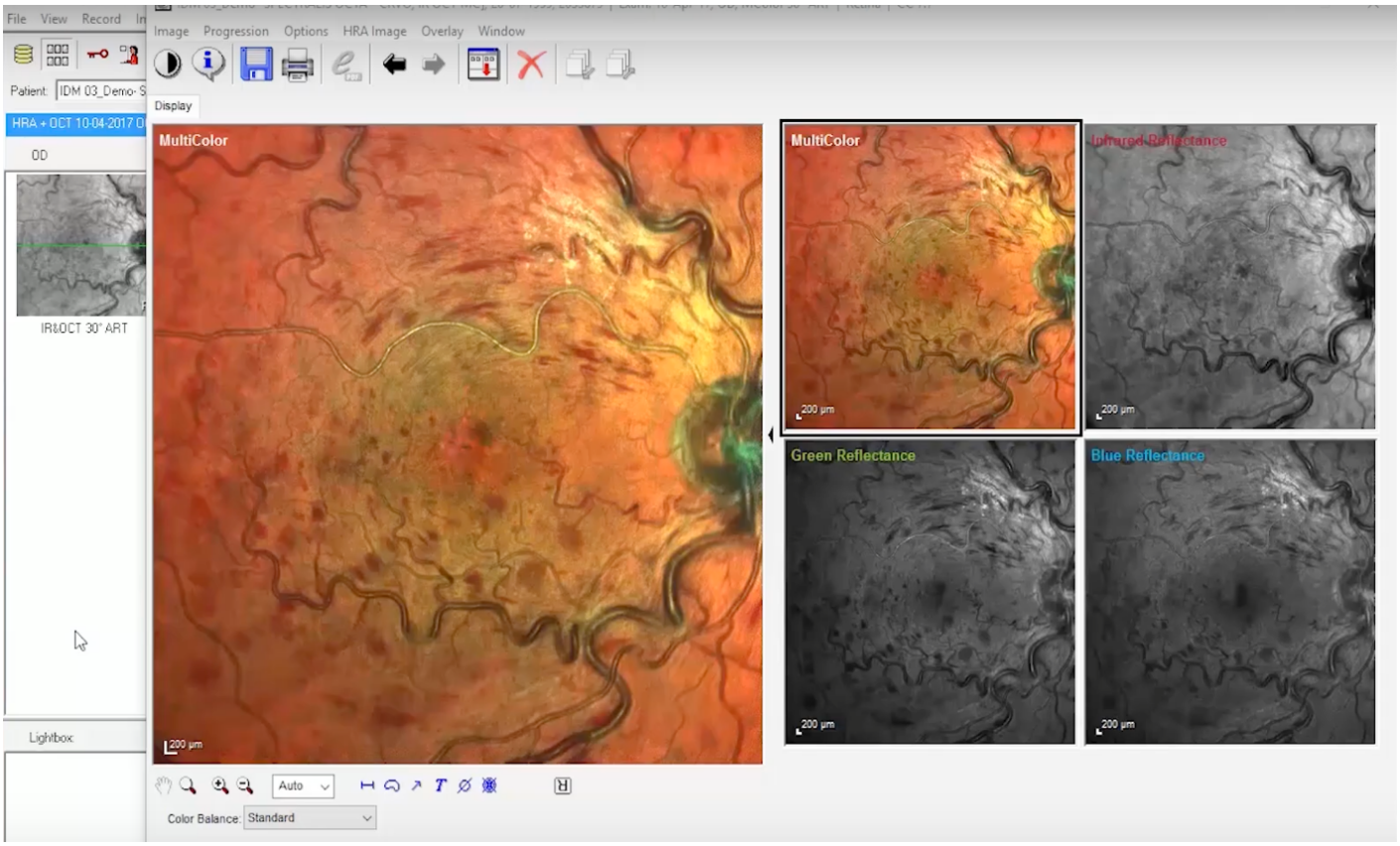

Figure 2b: Central retinal vein occlusion (CRVO) in MultiColor

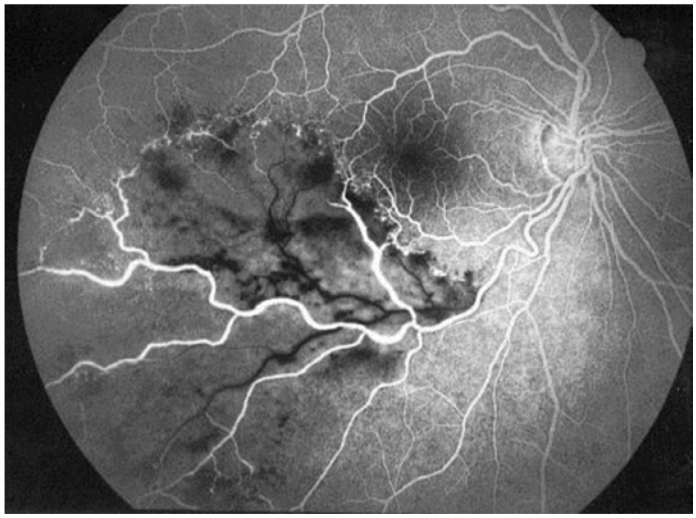

Figure 2c: Fundus fluorescein angiogram (FFA) showing an area of non perfusion following ischaemic hemi-central retinal vein occlusion (HCRVO)

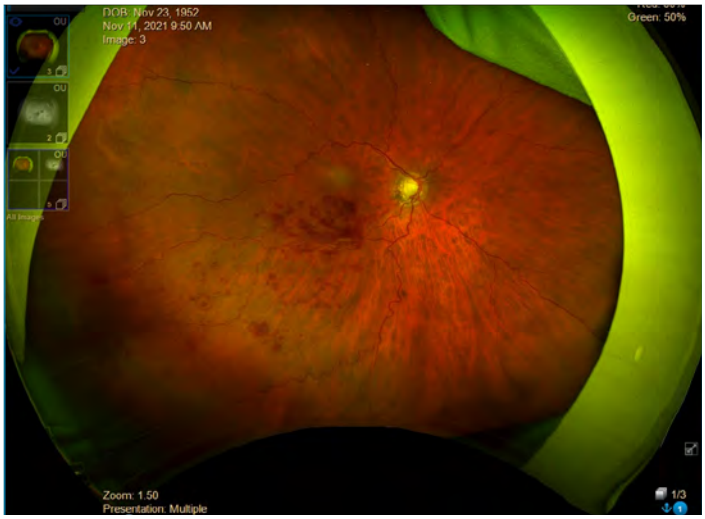

Figure 3a: Branch retinal vein occlusion (BRVO)

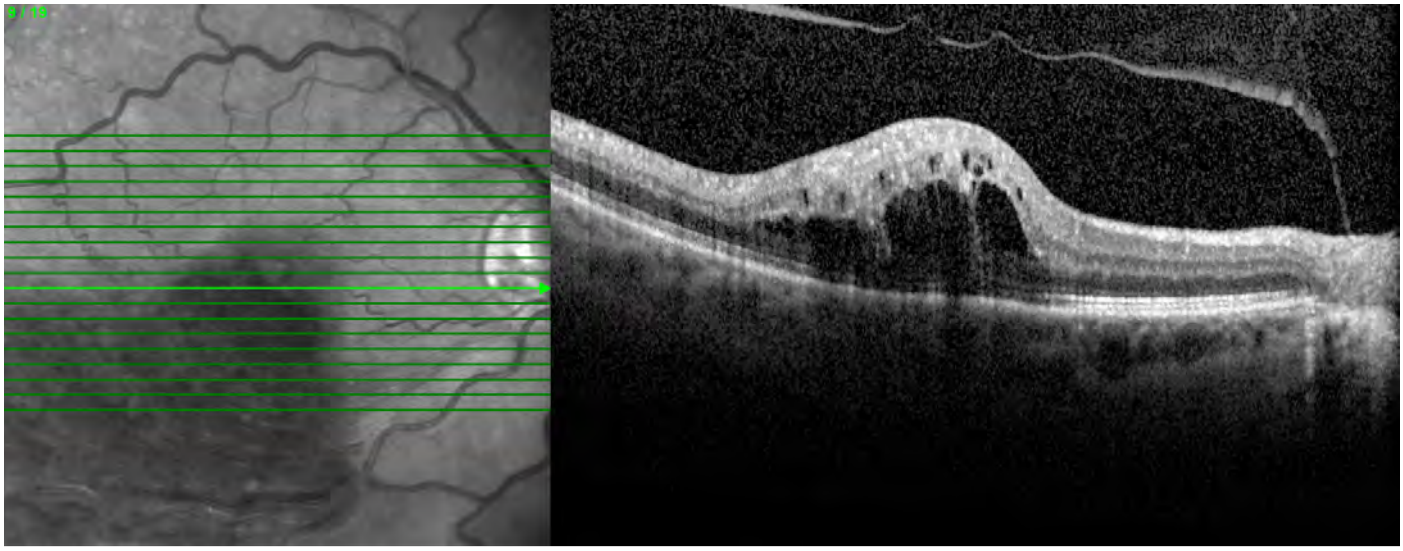

Figure 3b: Same BRVO on infra red image and OCT showing macular oedema

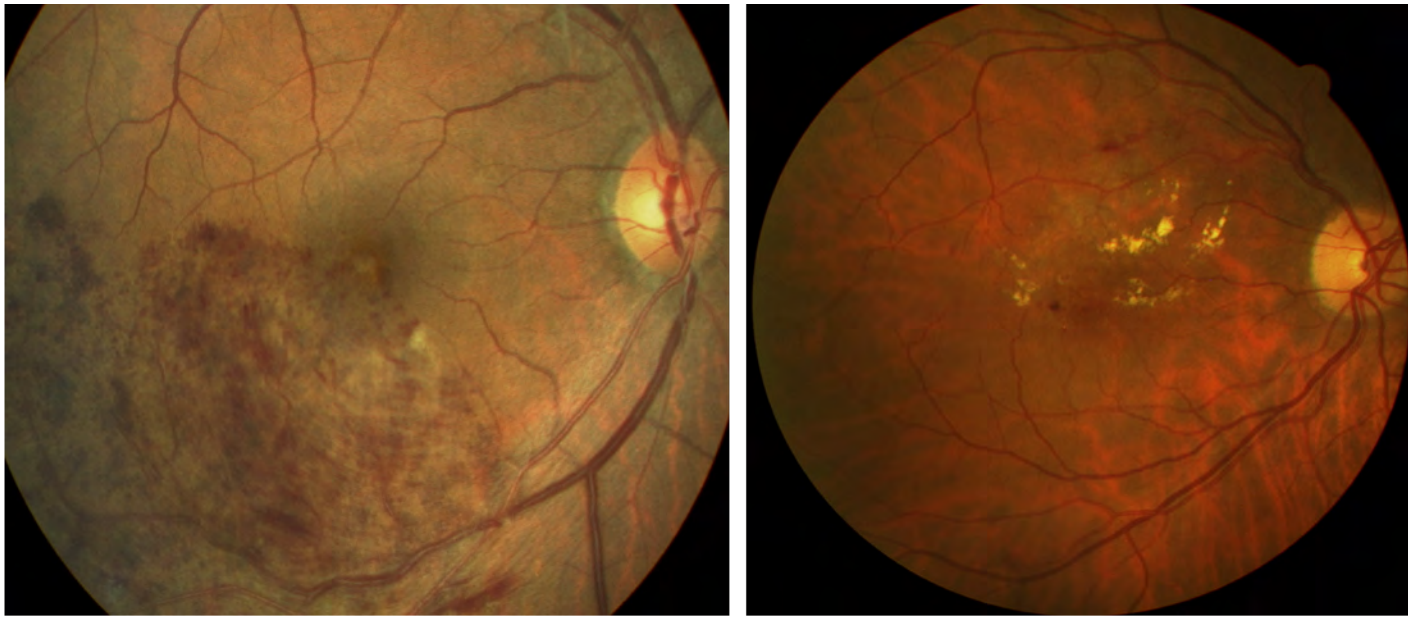

Figure 4: Branch retinal vein occlusion (BRVO) (b) BRVO with secondary macular oedema and exudates

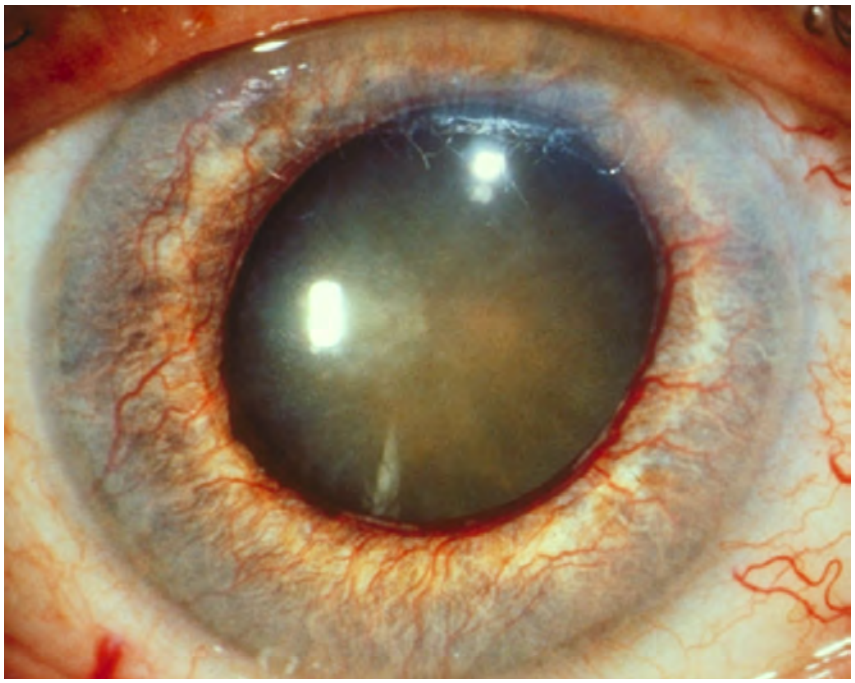

Figure 5: Neovascular glaucoma with extensive iris rubeosis

The risk of rubeosis in ischaemic HCRVO is greater than that of BRVO but less than that of CRVO (**Figure 5**).[8] The risk of disc neovascularisation appears greater for HCRVO than either ischaemic CRVO or BRVO.[9] The management of hemispheric vein occlusion is very similar to that described for branch retinal vein occlusion.

RVO clinical presentation

CRVO

CRVO presents with variable painless visual loss (<logMAR 1.0; Snellen 6/60) that often decreases further over time, retinal haemorrhages (both superficial flame shaped as well as deeper blot types) affecting all four quadrants of the retina, dilated tortuous retinal veins, cotton-wool spots, macular oedema, and optic disc oedema (**Figure 6a - c**).

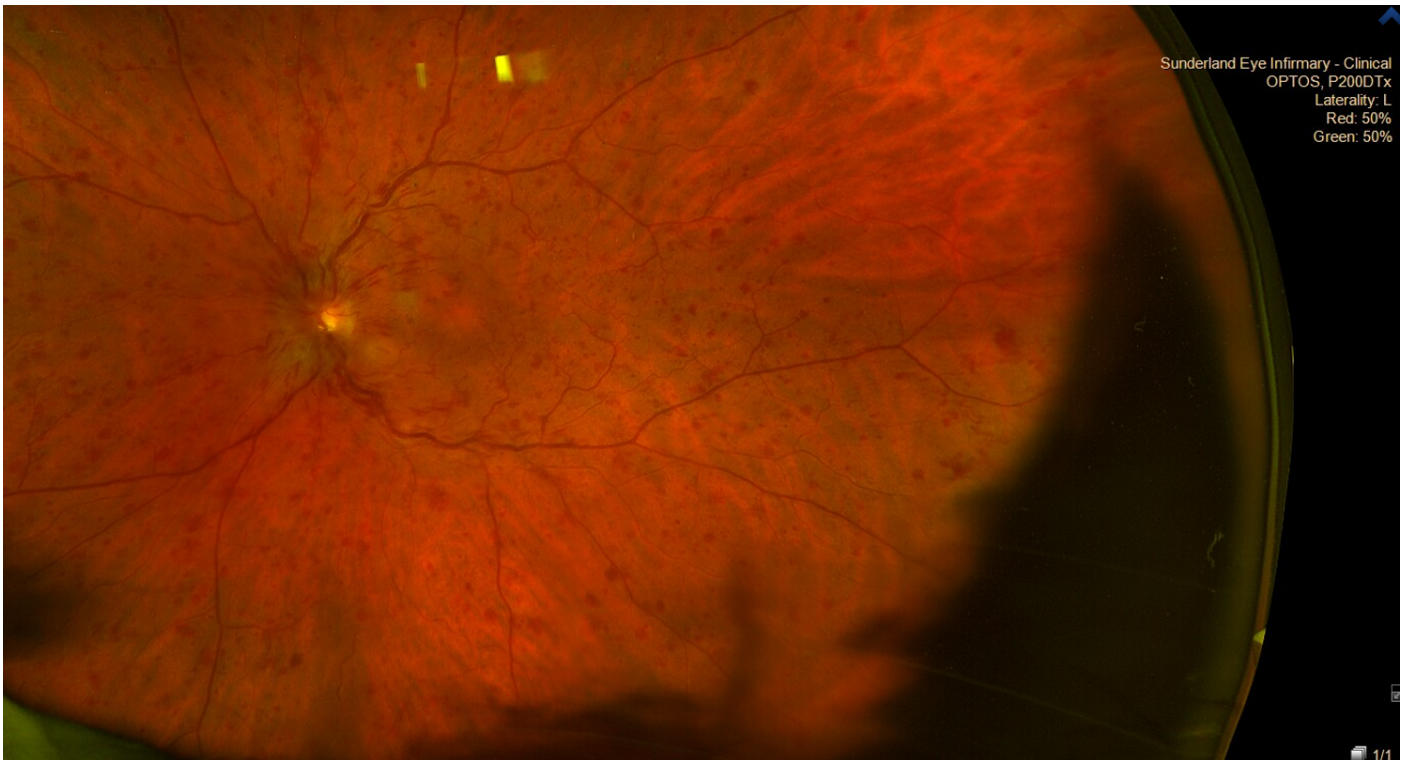

Figure 6a: Optos image of central retinal vein occlusion (CRVO)

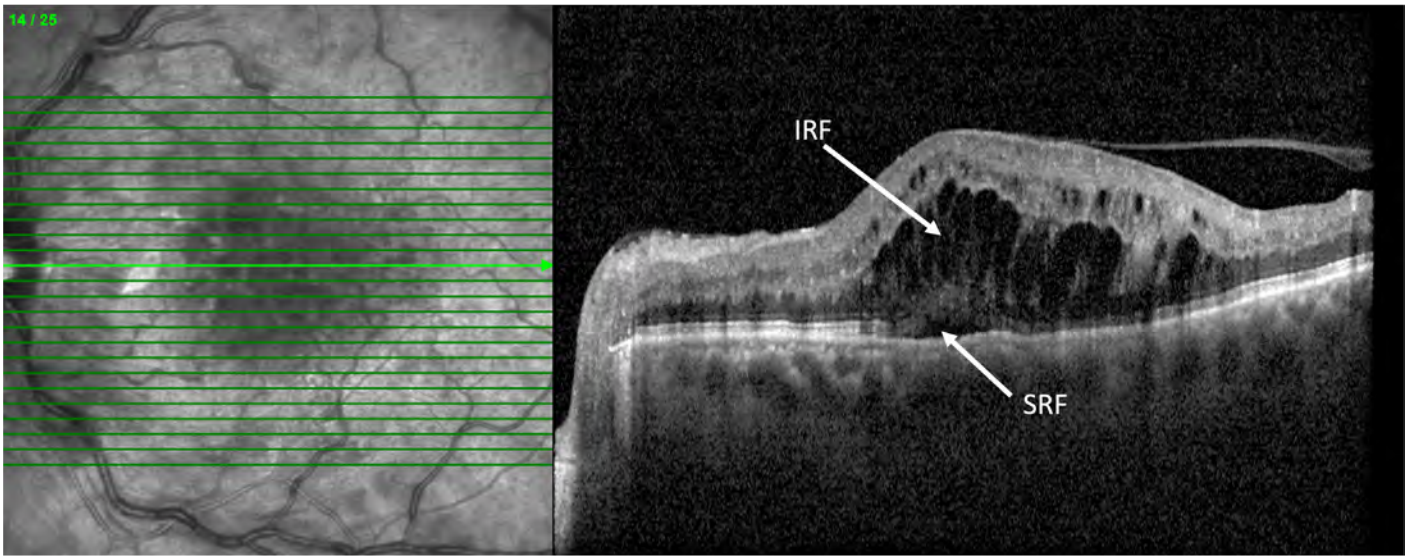

Figure 6b: Infrared image and OCT of same CRVO. Note extensive haemorrhages on IR image and macular oedema (intra-retinal fluid and sub-retinal fluid) on OCT

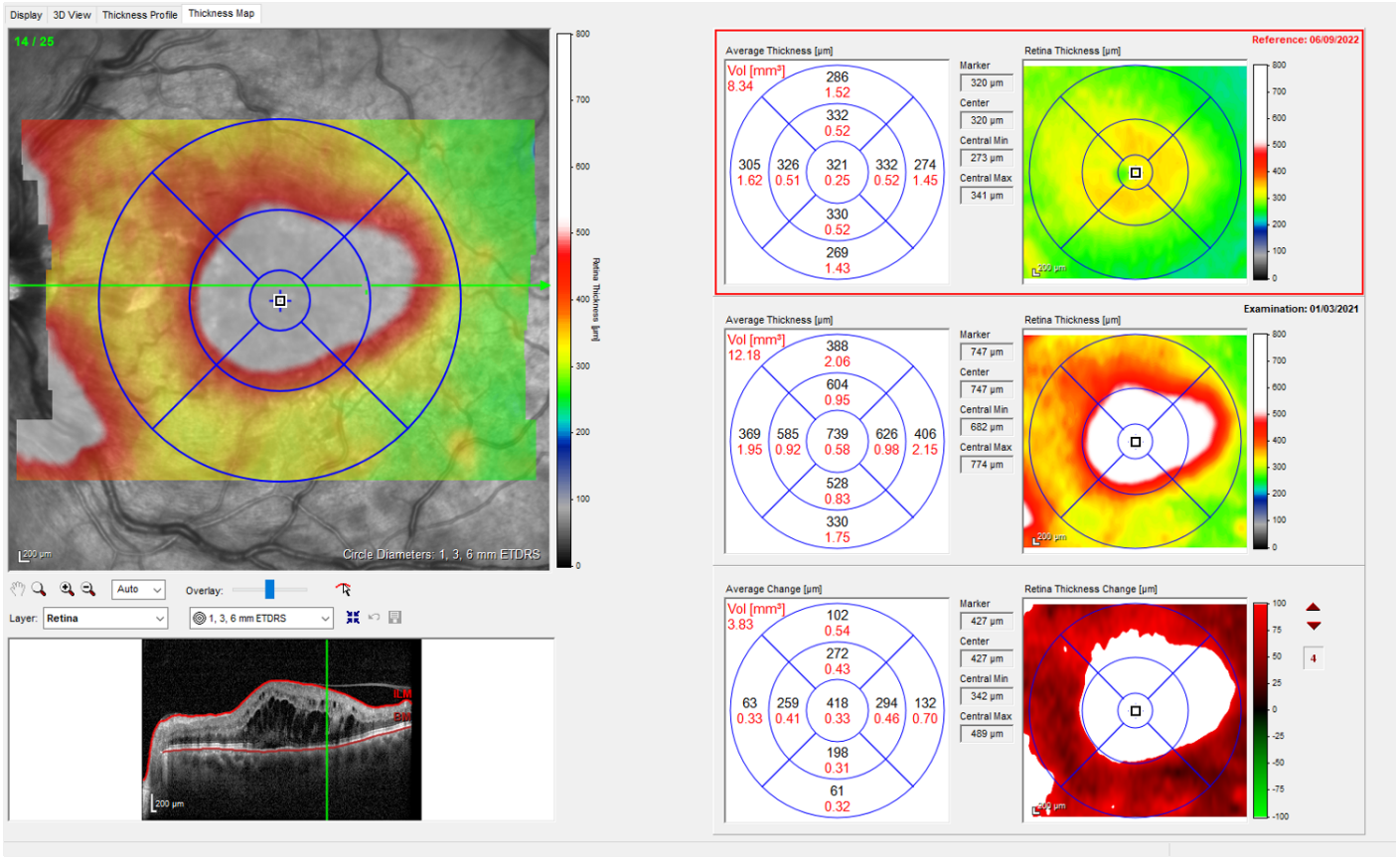

Figure 6c: Central retinal thickness map of same CRVO

The patient's history will normally reveal a number of associated risk factors including: age (90% greater than 50 years old), sex (similar risk for men and women), hypertension (~64% have hypertension), hyperlipidaemia (>6.5mmol/l) (especially in patients <50 years) diabetes mellitus (70% of type 2 diabetics are hypertensive), open angle glaucoma and less commonly bleeding disorders.[10]

Non-ischaemic CRVO (accounting for ~75% of CRVOs) is the milder form of the disease. It may present with relatively good vision, few retinal haemorrhages and cotton-wool spots, no relative afferent pupillary defect (RAPD), and good perfusion to the retina observed on fundus fluorescein angiography (FFA). Non-ischaemic CRVO may resolve fully with good visual outcome or may progress to the ischaemic type (see below) (**Figures 7a - c** and **8**).

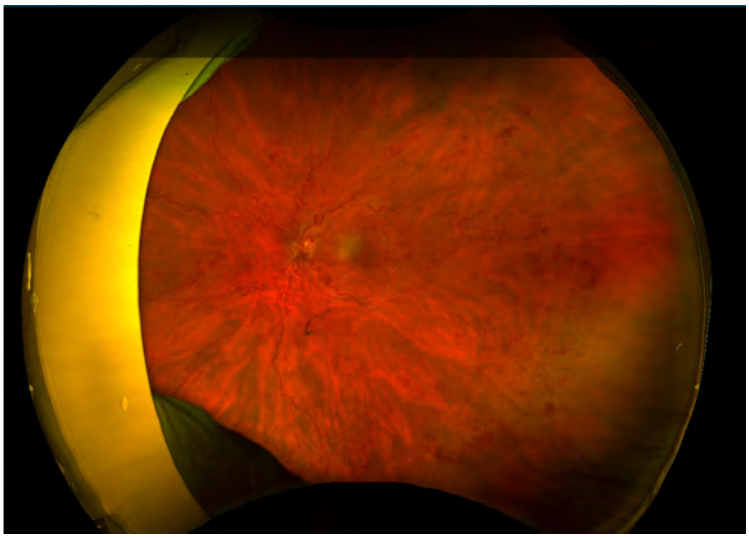

Figure 7a: Optos image of a non-ischaemic CRVO

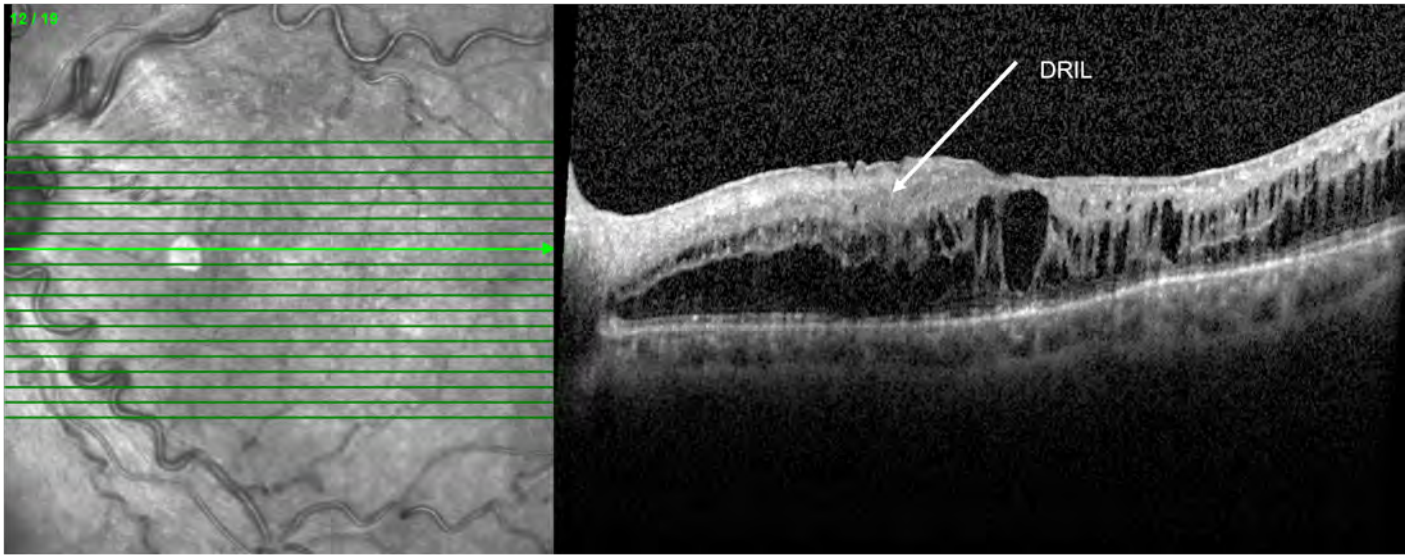

Figure 7b: Infra red image OCT of same eye showing very tortuous vessels and persistent macular oedema and disorganisation of retinal inner layers (DRIL)

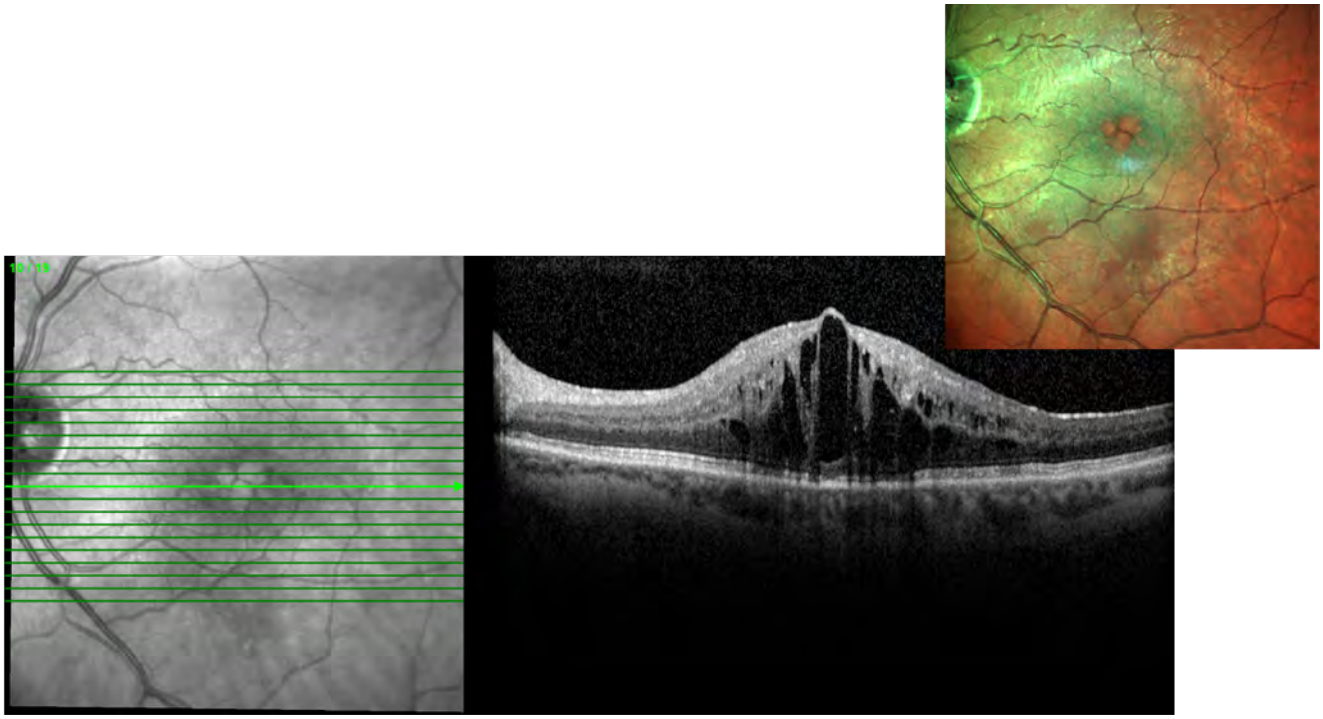

Figure 8: CRVO – Left persistent CMO (post Ozurdex 12) VA 6/24

Ischaemic CRVO (iCRVO) is the severe but less common form. Usually, iCRVO presents with severe visual loss (<logMAR 1.0; Snellen 6/60), extensive retinal haemorrhages and cotton-wool spots, presence of RAPD, poor retinal perfusion on FFA greater than 10 disc diameters and demonstrable severe electrodiagnostic changes (**Figures 9a - c** and **10**).

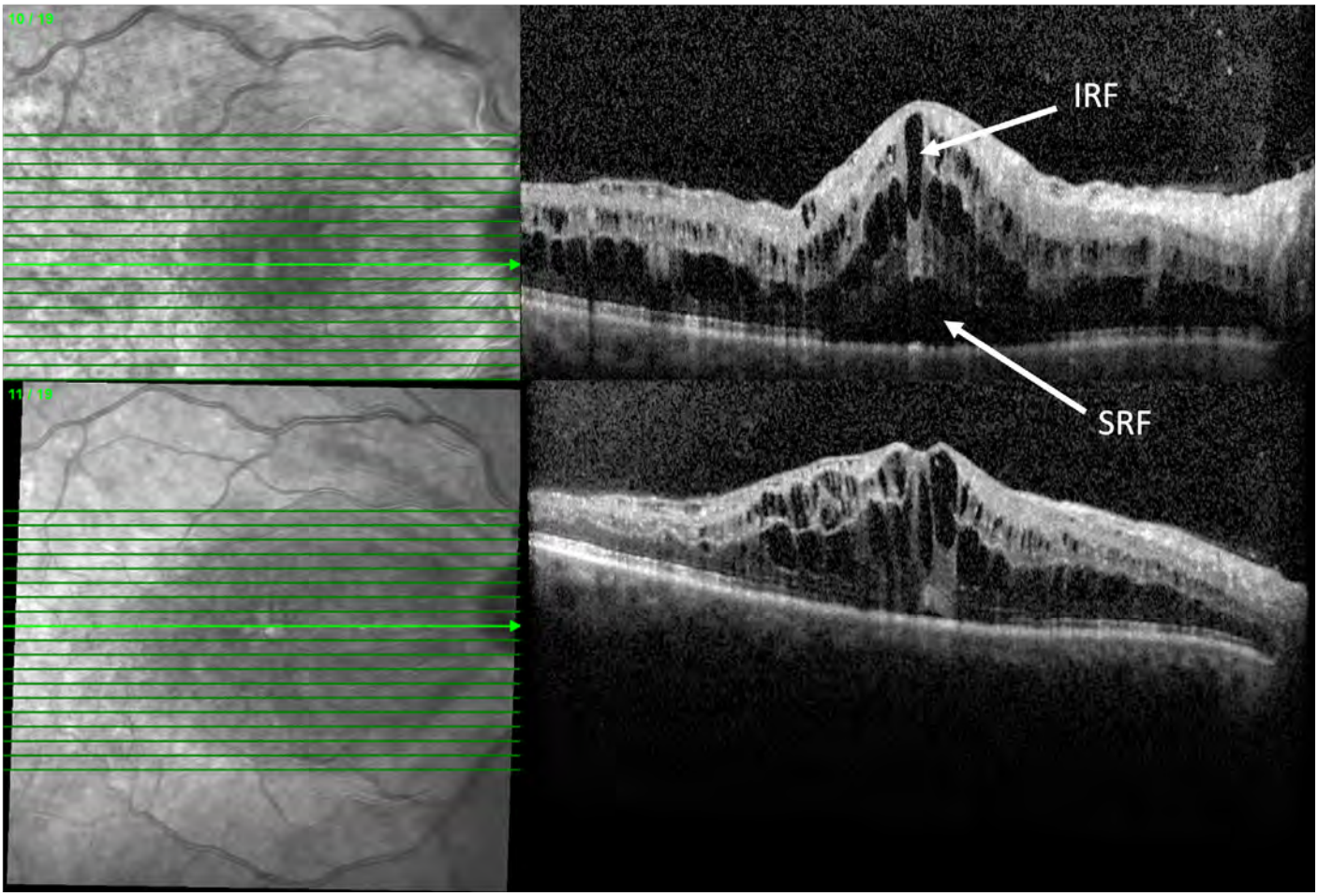

Figure 9: RE CRVO with extensive macular oedema and disorganisation of retinal inner layers (DRIL)

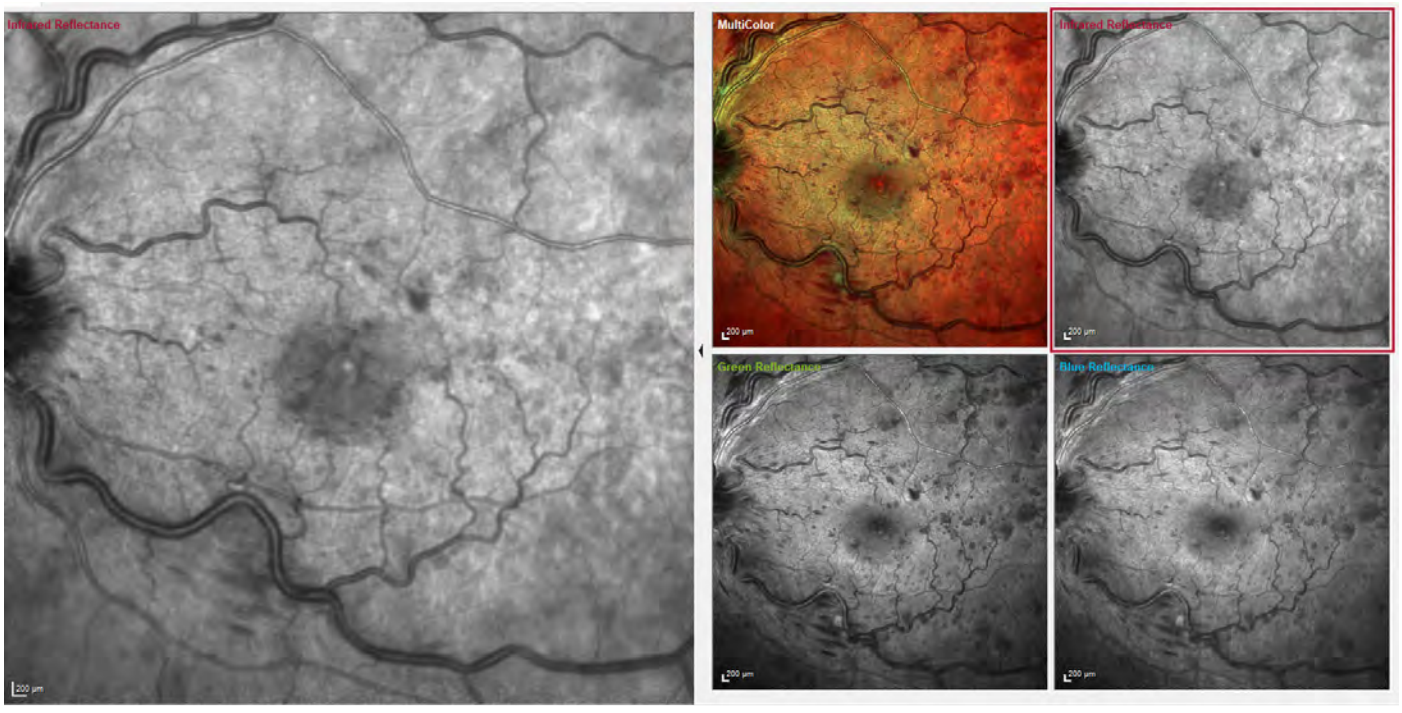

Figure 10a: Infra-red image and Multicolor of a left CRVO

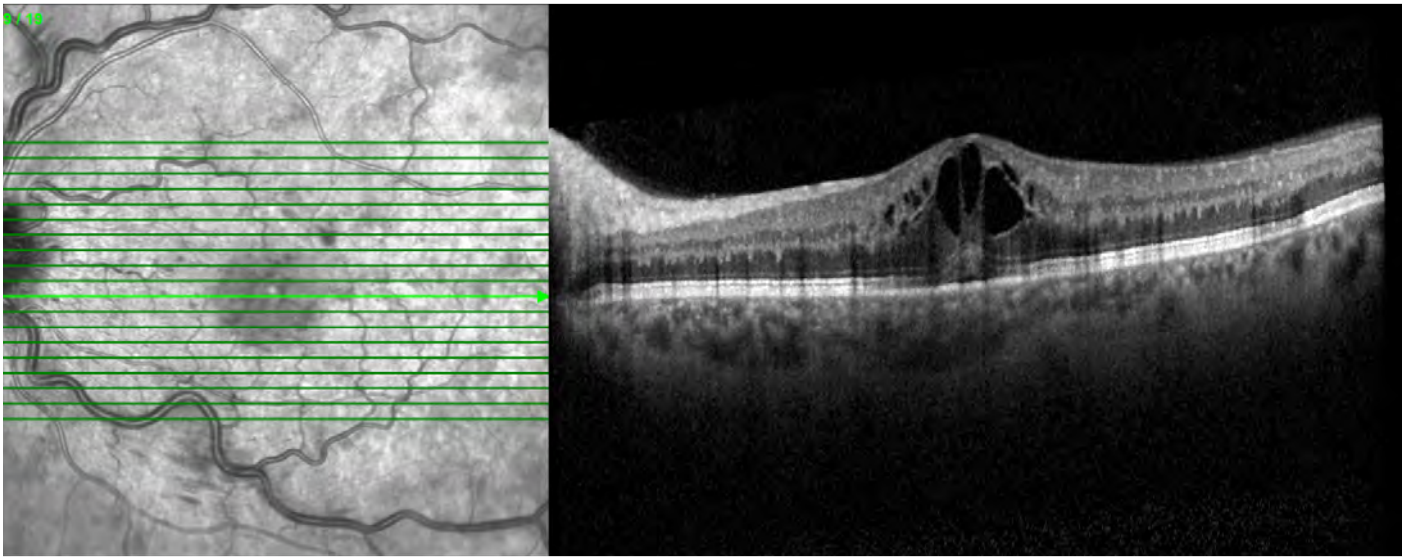

Figure 10b: Same CRVO showing significant macular oedema

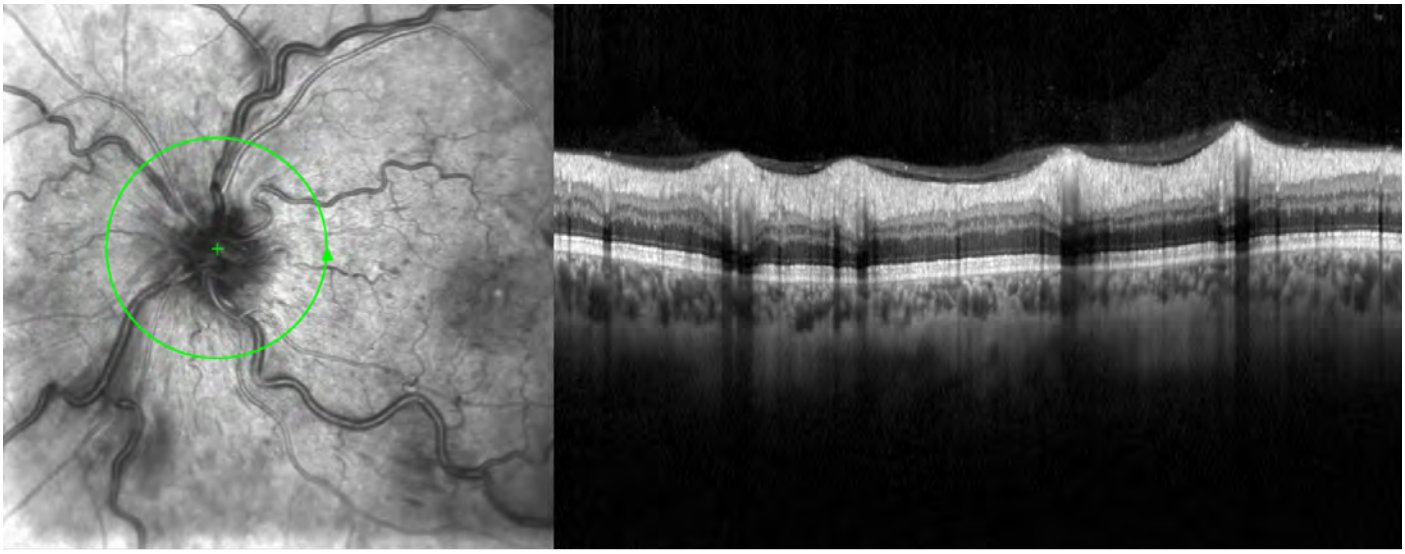

Figure 10c: Same eye optic nerve head scan demonstrating disc swelling on OCT

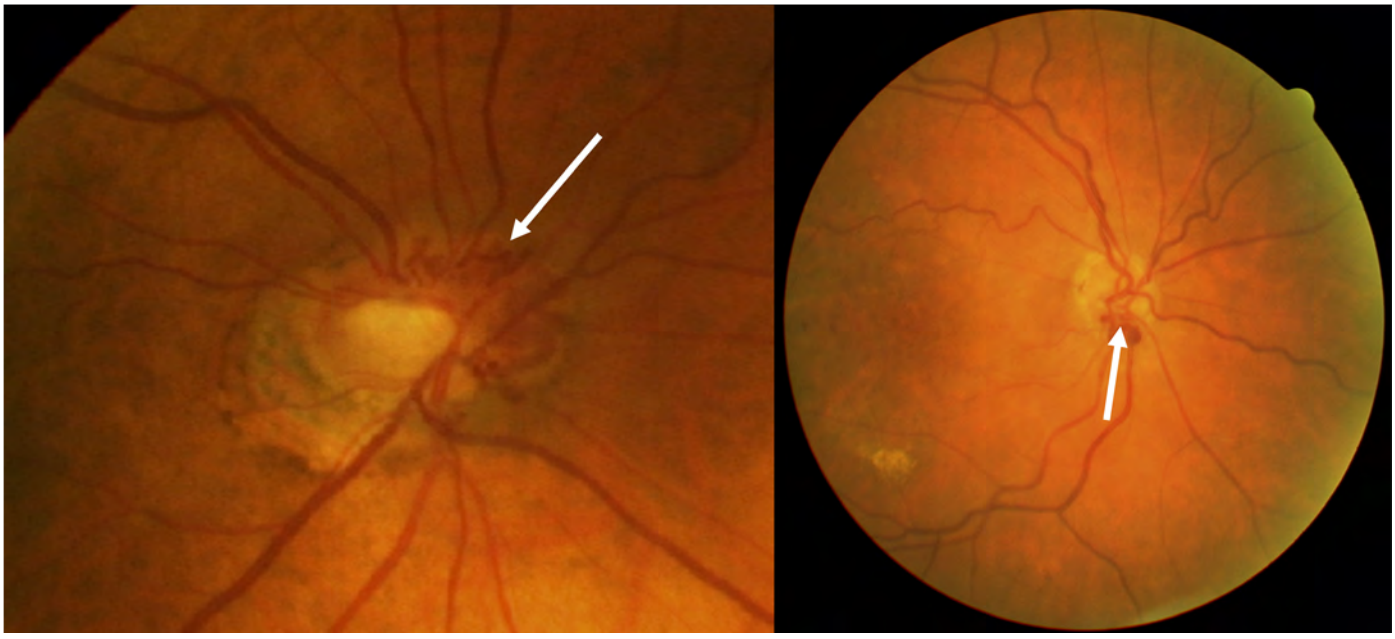

Figure 11a: Optic disc collaterals vessels

Development of neovascular (rubeotic) glaucoma is a major concern in ischaemic CRVO as a result of intra-ocular neovascularisation (**Figure 5**). The Central Vein Occlusion Study (CVOS) confirmed the direct correlation between severity of ischaemia and the risk of developing neovascularisation.[11] It is important to differentiate between optic nerve head neovascularisation (NVD) or new vessels elsewhere (NVE) and collateral vessels.

Collaterals are compensatory channels that develop to provide a blood supply from the choroidal or pial circulation to obstructed vessels on the optic nerve head or elsewhere. Typically, they appear as tortuous "chunky" loops that do not usually leak on FFA (**Figure 11a - b**) compared with the lacy fronds found with new vessels that do leak on FFA.

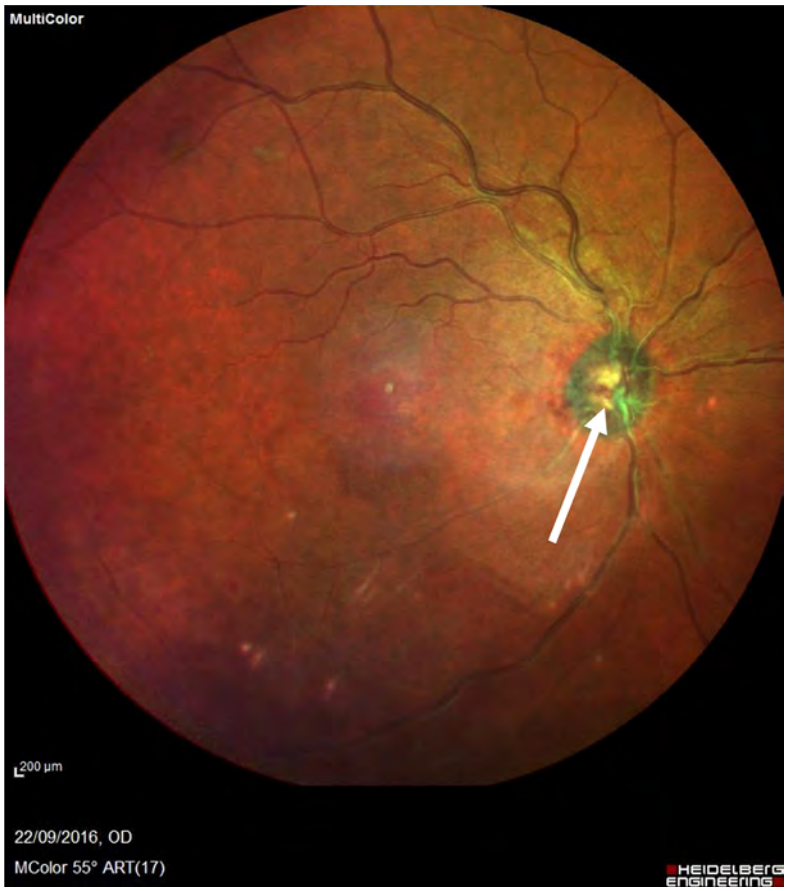

Figure 11b: Post CRVO Collaterals on a Multicolor image

Although CRVO can be broadly divided into these 2 main clinical types (non-ischaemic and ischaemic) as described above a number of patients may have an intermediate presentation with a variable clinical course, as some patients may change from an initially non-ischaemic to ischaemic presentation over time. In one reported systematic review up to around a third of eyes with non-ischaemic CRVO converted to ischaemic CRVO over a 3-year period. In ischaemic CRVO cases, neovascular glaucoma developed in at least 23% of eyes within 15 months. In non-ischaemic CRVO cases, macular oedema resolved in approximately 30% of eyes over time, and subsequent neovascular glaucoma was rare.[12]

BRVO

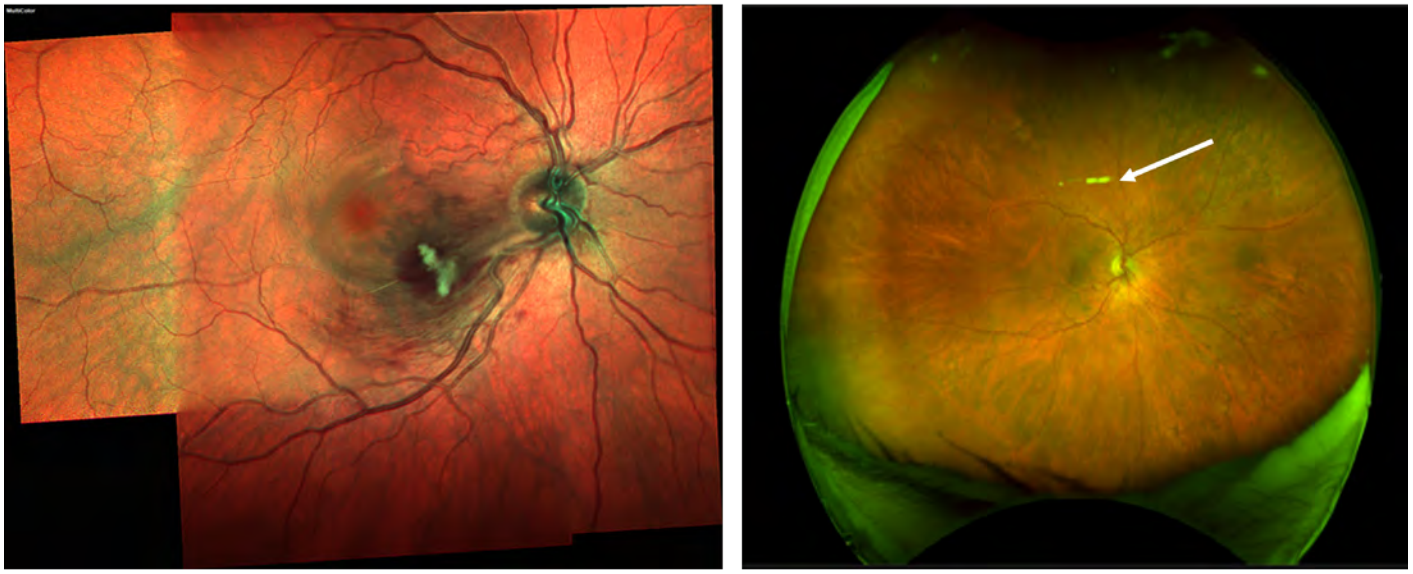

Figure 12: Branch retinal vein occlusion affecting one quadrant of the fundus. Note Ozurdex implant in the vitreous

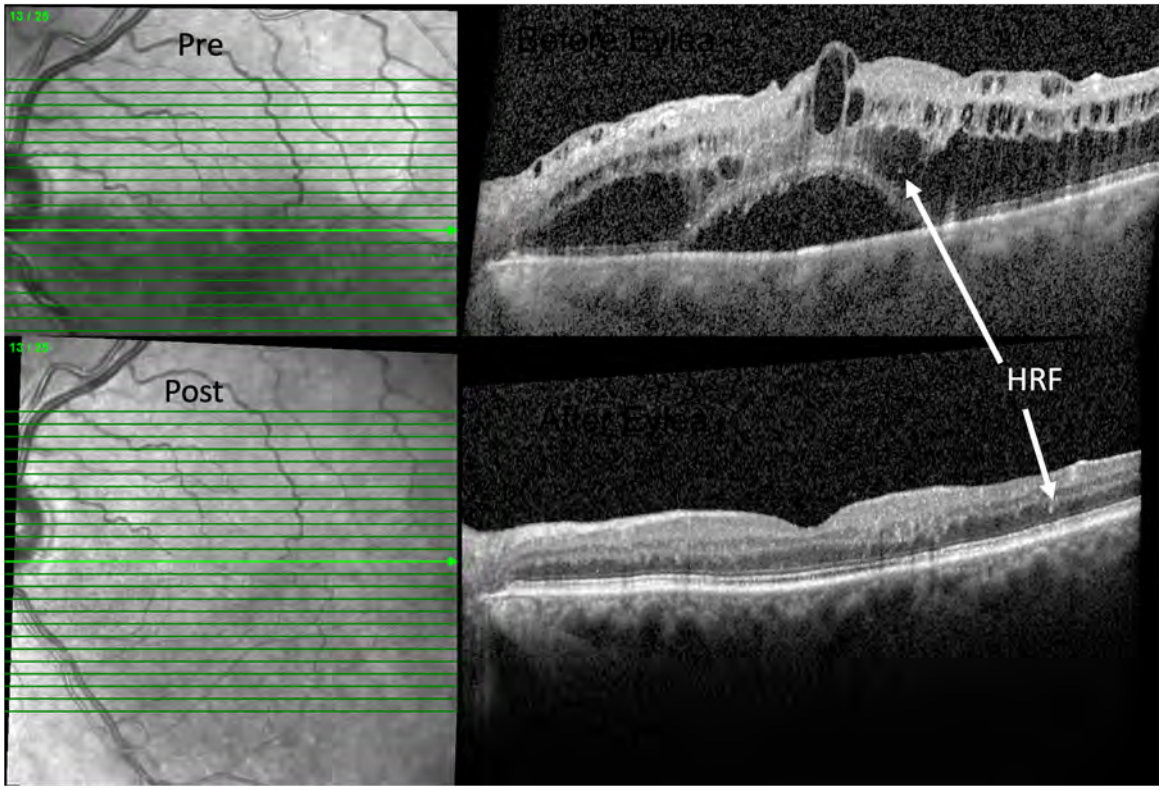

Figure 13a: Hemi-retinal vein occlusion (HRVO) with extensive macular oedema – pre and post Eylea x 5 intraviteal injections. Note multiple hyper-reflective foci (HRF) or spots

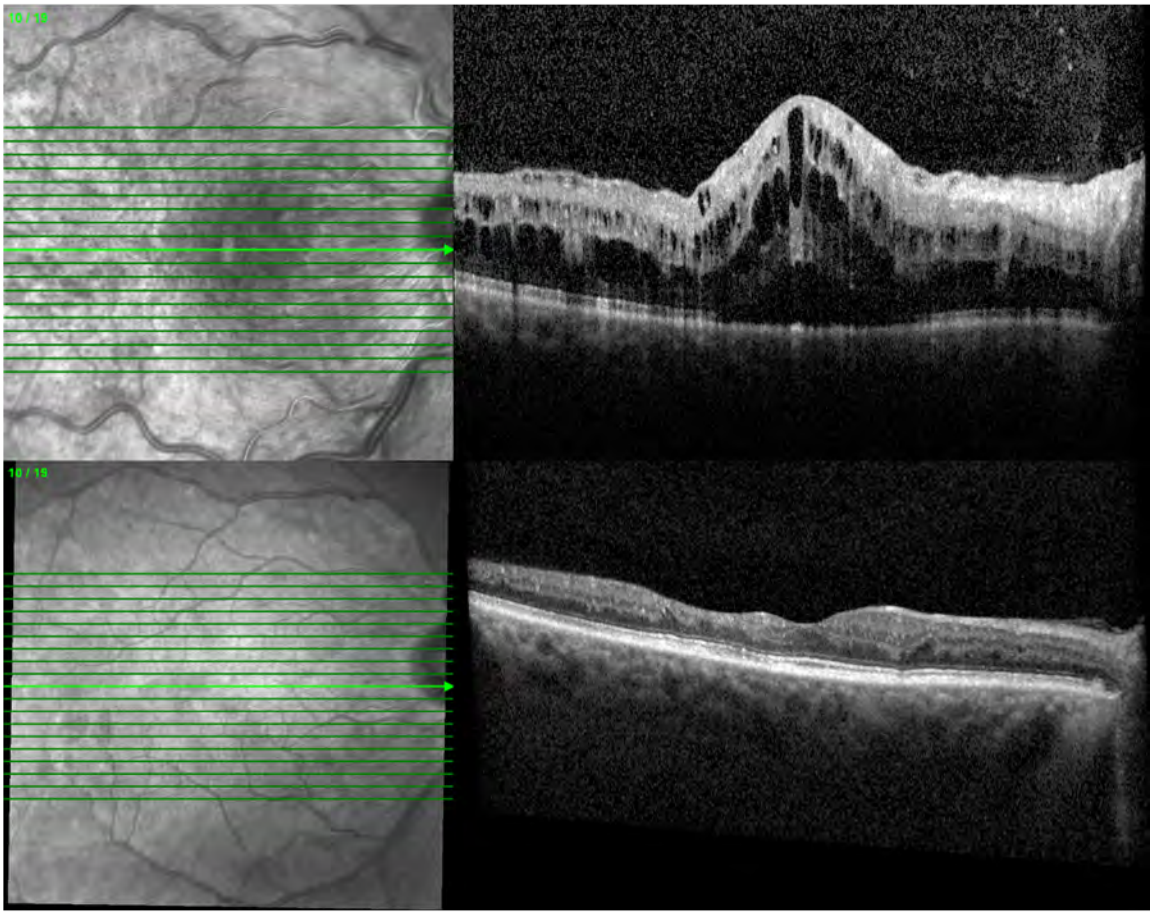

Figure 13b: Non-ischaemic CRVO pre and post Eylea x 26 intravitreal injections. (VA logMAR 1.0 (6/60) letters improved to logMAR 0.40 VA (6/15)

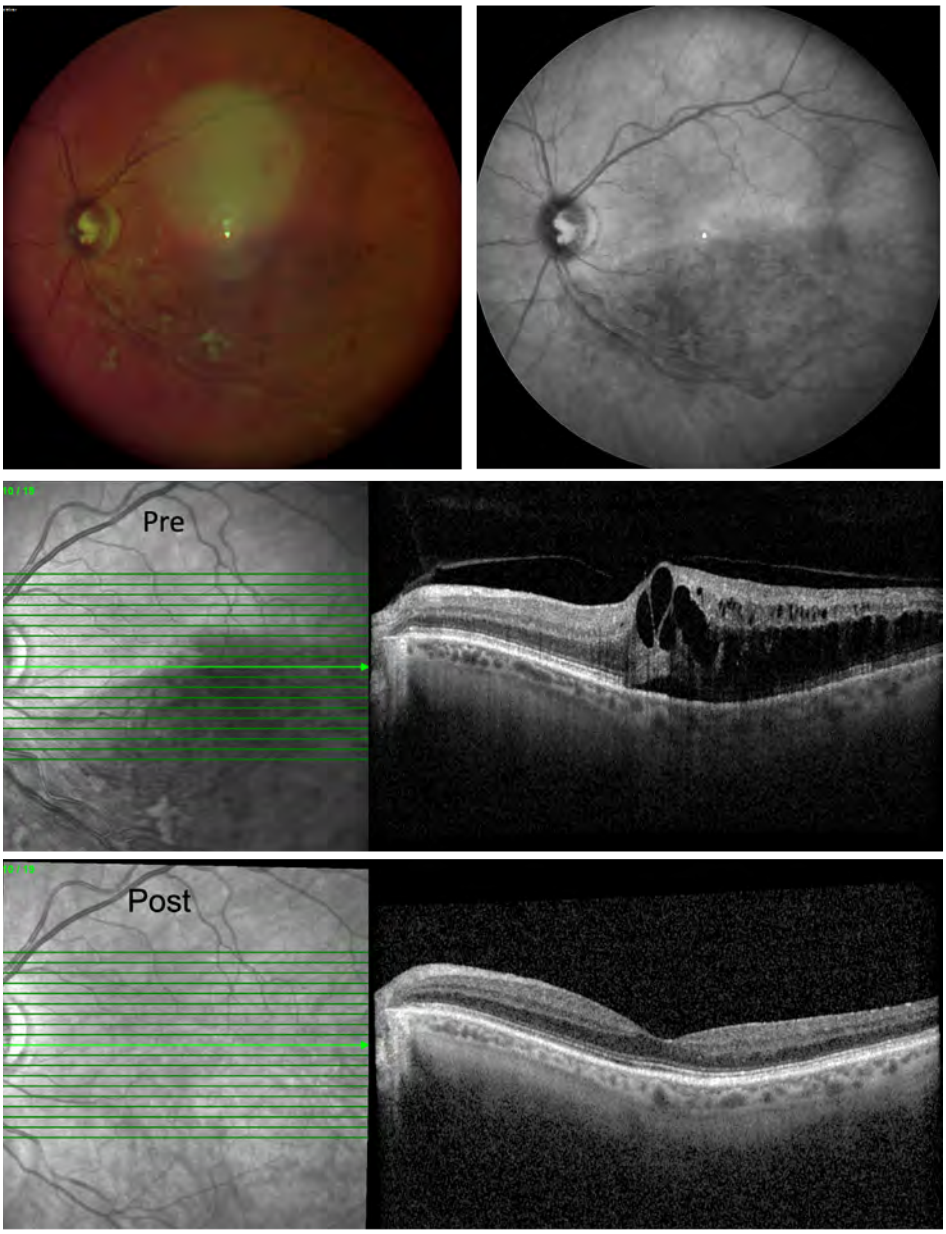

Figure 14: LE Branch retinal vein occlusion (BRVO) preand post Eylea x 5

On presentation VA is usually reduced (worse than logMAR 0.30; Snellen VA 6/12), with a clinical appearance similar to the above but with retinal haemorrhages and cotton wool spots, limited to one or two quadrants of the retina (**Figures 12** and **13a - b**). Although there may be some improvement in the follow-up period, this is often limited, and VA does not usually ever improve beyond Snellen 6/12. In BRVO, haemorrhages are largely localized to the area drained by the occluded branch retinal vein (**Figure 14**). Vision loss occurs secondary to macular oedema or ischaemia. Macular oedema may develop in 5% to 15% of eyes over a one year period. However, up to 40% of eyes that develop macular oedema at presentation may show some resolution over time.[14] In ~20% of untreated eyes vision will deteriorate over time. Fellow eye involvement by BRVO may occur in 10% of cases.[13]

RVO clinical management

For all RVO patients a full history is important including duration of vision loss and all pertinent risk factors mentioned above. A complete ophthalmic examination should include visual acuity measurement, pupil reactions (RAPD), intraocular pressure (IOP), anterior and posterior segment slit lamp biomicroscopy, un-dilated iris assessment (for rubeosis) and very importantly, gonioscopy to check for any fine new vessels in the anterior chamber angle and/ or pupil margin. Sometimes these may precede the appearance of new vessels elsewhere (NVE) in the retina. If left untreated neovascular glaucoma may well develop leading to a blind, painful eye.

Where new treatments are being considered OCT and OCTA should always be undertaken (see below) to assess the extent of associated macular oedema and other retinal changes. The longer the duration of macular oedema the more likely structural foveal damage is and therefore early initiation of treatment is recommended (see below).

In CRVO electrodiagnostic tests are not usually undertaken unless there is any doubt in differentiating the type of CRVO present. A fundus fluorescein angiogram (FFA) is very helpful in differentiating ischaemic from non-ischaemic CRVOs, although sometimes this should be delayed if there is substantial haemorrhage present, as little will be gleaned from the angiogram images. If necessary, it is best to wait until the haemorrhages have cleared to some extent. Further medical investigations should include:

- Full blood count (FBC)
- Erythrocyte sedimentation rate (ESR)
- Urea electrolytes
- Creatinine
- Cholesterol and HDL cholesterol
- Random blood glucose and/or glucose tolerance test
- Thyroid function tests (TFTs)
- Plasma protein electrophoresis

Depending on clinical indication more specialised tests (particularly in younger patients) may include (Royal College Ophthalmologists 2010):[14]

- Thrombophillia screen
- C-reactive protein
- Serum ACE
- Chest X ray
- Auto-antibodies
- Fasting homocysteine level

The presence and extent of retinal ischaemia in RVO is associated with a worse prognosis. As defined above, ischaemic retinal vein occlusion (iRVO) and non-iRVO are often considered as separate entities based on set thresholds of existing retinal ischemia as determined by FFA. However, there is still no fully accepted definition for iRVO which is associated with a higher risk of visual loss and neovascular complications. Therefore, iRVO should be identified as accurately as possible in patients with this disease and also be carefully considered in clinical and experimental studies. Most recently conducted clinical trials evaluating new treatments for macular oedema secondary to RVO included none or only few patients with iRVO based on previous definitions (i.e., few patients with sizeable areas of retinal ischaemia were recruited in these trials), and thus it is uncertain whether the results observed in recruited patients can be extended to those with retinal ischaemia.[15] The role of OCT and OCTA is now becoming increasingly important in differentiating the different types of RVO (see below).

If RVO has been considered as a diagnosis after fundus examination, three questions need to be asked:

- ***Is another diagnosis possible?***
- ***Is there any macular oedema?***
- ***What is the extent of the retinal ischaemia?***

The different diagnoses to consider depends on the retinal clinical presentation that could be related to any blood circulatory problems:[16, 17, 18]

- Increased blood viscosity or altered coagulation leading to retinal hemorrhages (usually in both eyes). The presence of retinal vasculopathy in the absence of typical pre-disposing factors should suggest a possible underlying haematologic abnormality. In such cases, a systemic investigation may reveal a potentially fatal hyper-coagulability or hyper-viscosity syndrome[19]
- Carotid occlusion that can simulate some aspects of vein occlusion
- Certain macular telangiectasias, which can initially be confused with small BRVO (signs are present on both, the superior and inferior temporal side of the fovea, which depends on two different venular branches)
- Downstream vein pressure such as in a cavernous sinus thrombosis

Optical coherence tomography (OCT) in retinal vascular occlusion (RVO) assessment

OCT is the most commonly used imaging modality in RVO. The fast-emerging new technologies, mainly swept-source OCT technology, are especially useful as they allow microstructural and near histologic imaging. Densely spaced volume scans create high-resolution images, where many different features can be distinguished. In RVO, the identified features look very much like those of chronic diseases such as diabetic macular oedema (DMO – see Part 2), but with the acute onset of CRVO and BRVO there may be other features, or the same features can be interpreted differently. Following RVO a range of specific identifiable pathological features (biomarkers) on OCT include:[20]

- **Central retinal thickness (CRT)**, defined as the mean thickness of the retina between its inner and outer boarders, on all A-scans taken in the central 1-mm area, is the most used OCT feature (**Figure 6c**). CRT has been the major end-point in most randomised clinical trials. Increased CRT closely correlates with functional loss, whereas its decrease correlates with functional gain.[21, 22] CRT measurements are used to evaluate disease activity and progression as well as the treatment response in each individual patient and can be imaged with any OCT device. Also, CRT is a robust variable because RVO is an acute-onset disease and may not show as many long-term changes as chronic retinal diseases. The photoreceptor status has no direct influence on VA at the time of the acute onset of the disease but plays an important role in VA prognosis after therapy. CRT is usually closely linked to the existence of intra-retinal fluid.
- **Intraretinal fluid (IRF)/cystoid fluid** usually presenting as minimally or non-reflective round or oval cystoid spaces within the neurosensory retina (**Figures 6-10**)
- **Subretinal fluid (SRF)**. This is the non-reflective space between the neurosensory retina and the retinal pigment epithelium (**Figure 9**)
- **Macular oedema** (**Figures 6-10**)
- **Changes in integrity of the photoreceptor bands** (external limiting membrane (ELM) and ellipsoid zone (EZ))
- **Disruption of the external limiting membrane (ELM), ellipsoid zone (EZ), and interdigitation zone (IZ)**
- **Hyper-reflective foci (HRF)** or spots (HRS) are dot like well-demarcated lesions, often scattered throughout all retinal layers in RVO on OCT images (see parts 1 and 2).[23] HRF are associated with poorer visual outcomes and a reduction of HRF can be achieved with anti-VEGF or steroid treatment (see below).[24] HRF are more common in patients with more retinal thickening (**Figure 13a**). HRF in the outer plexiform layer (OPL) have an especially negative impact on VA prognosis. The more HRF that are present leads to a greater likelihood of photoreceptor layer (inner and outer photoreceptor segment line and the external limiting membrane) disruption. This in turn explains the worse VA outcomes in these patients.[25, 26] The accumulation of HRF look different from that in DMO, where HRF usually accumulate around fluid departments. In RVO most HRF accumulate around the outer plexiform layer regardless of the fluid around them.[27]
- **Choroidal thickness changes**

- **Disorganisation of retinal outer layers (DROL)**
- **Disorganisation of retinal inner layers (DRIL).** This was previously characterised as the lack of distinguishable boundaries between the inner retinal layers. DRIL can in fact be caused by many different features, such as intraretinal cysts (IRC) that alter retinal boundaries, an increase or decrease of optical intensity, HRF or a generalised blurring of layers that presents as a homogenous mass.[28] The extent of disorganisation of the retinal inner layers is strongly correlated with any area of capillary non-perfusion. Therefore, the extent of DRIL can be used as an easily observable and important indicator of capillary ischaemia (**Figure 7b**). The greater the degree of DRIL at baseline correlates with worse baseline VA.[29] DRIL is therefore a negative predictor of visual outcome in retinal vein occlusion (RVO), as well as a range of other conditions such as diabetic macular oedema (DMO), central retinal artery occlusion (CRAO), uveitis, and epiretinal membrane.[30]
- Other signs of ischaemia seen in RVO, include:
    - **Prominent middle limiting membrane (p-MLM)**. This is a hyper-reflective line located at the inner aspect of outer plexiform layer and is suggestive of acute ischaemia with a poor visual prognosis.
    - **Paracentral acute middle maculopathy (PAMM)** which is associated with focal VA loss.[31]
    - **Hyper-reflectivity of inner retinal layers (HIRL)** (**Figure 15**).

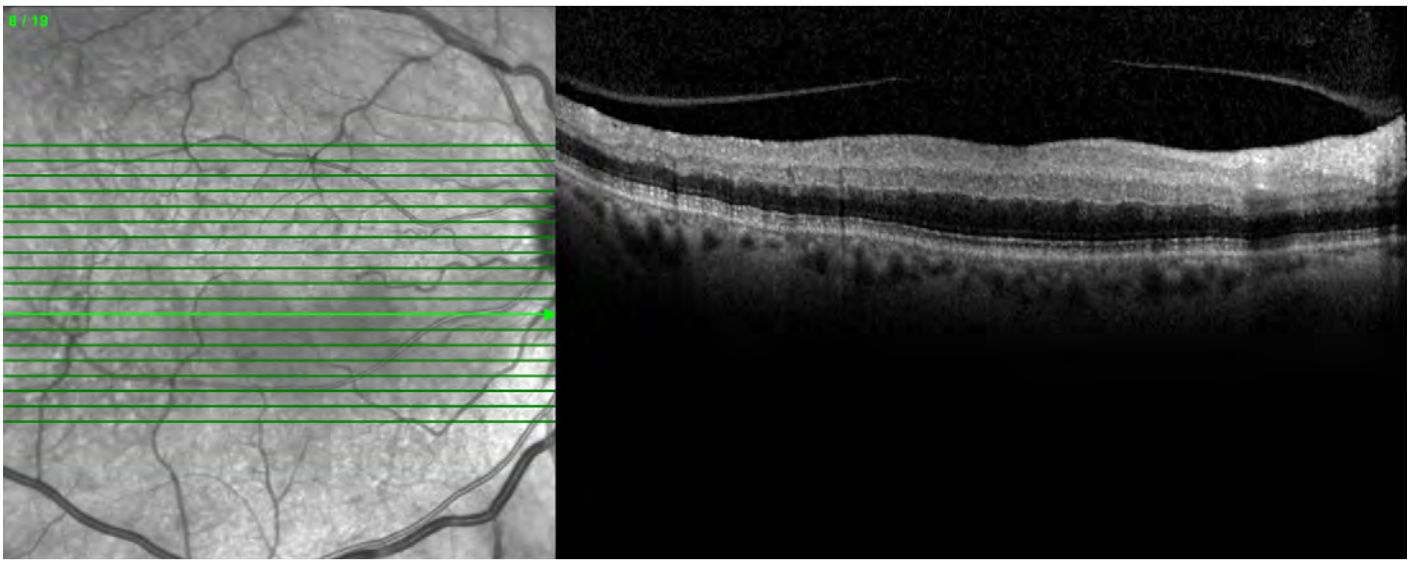

Figure 15: Possible hyperreflectivity of inner retinal layers (HIRL)

Using spectral domain OCT (SD-OCT) a recent study reported significant thinning of inner retinal thicknesses in the focal macular area after ranibizumab therapy in both CRVO and BRVO cases. Retinal thinning was predominantly in the inner plexiform layer (IPL) to outer nuclear layer (ONL) compartment in focal macular areas in patients with RVO.[32]

In another recent study patients with ischaemic and non-ischaemic CRVO whose macular oedema resolved after intravitreal anti-vascular endothelial growth factor injections and did not recur for at least 6 months, was compared with the normal contralateral eye. Each retinal layer thickness was compared according to Early Treatment Diabetic Retinopathy Study (ETDRS) sub-fields using SD-OCT. In CRVO eyes with resolved macular edema, the outer retinal layers were thinner as well as inner retinal layers, whereas IPL and ONL were thicker than normal fellow eyes. Additionally, photoreceptor layer thickness in foveal area had a significant impact on visual acuity in CRVO.[33]

Morphological optic nerve head changes on spectral-domain optical coherence tomography (SD-OCT) in RVO have also been recently described. In one recent study morphologic parameters of optic nerve head were assessed using Spectralis SD-OCT using a radial pattern. The length of Bruch's membrane opening (BMO) was measured in OCT scans, and optic disc diameters (DD) were assessed in infra-red fundus photographs. Most of the eyes with CRVO had normal optic disc diameter, but approximately 25% of patients with CRVO had reduced optic disc dimensions in terms of BMO and DD.[34]

Another recent OCT based study examined optic nerve head (ONH) anatomy in young adults with central retinal vein occlusion (CRVO), branch retinal vein occlusion (BRVO), central retinal artery occlusion (CRAO), branch retinal artery occlusion (BRAO) or non-arteritic anterior ischemic optic neuropathy (NA-AION) in order to look for associated, potentially predisposing anomalies.

Using OCT, the presence of optic disc drusen (ODD), pre-laminar hyper-reflective lines and peripapillary hyper-reflective ovoid mass-like structures (PHOMS) amongst other features was determined. ODD, prelaminar hyper-reflective lines and PHOMS were less common in RVO than in NA-AION patients. The prevalence of ODD in retinal vascular occlusion patients was similar to the reported prevalence in the general population.[35]

MultiColor fundus imaging

Retinal vascular occlusion (RVO) is one of the conditions in which MultiColor fundus imaging is at its most effective. Importantly, the findings in the MultiColor fundus images are consistent with the changes in OCT findings (**Figures 2, 12**)

Retinal artery occlusion

Central retinal artery occlusion

This is an ophthalmic emergency. It is a condition characterised by the impediment of blood flow affecting the whole retinal circulation caused by the occlusion (mostly embolic) of the central artery of the retina. At presentation the vision is usually severely reduced and the prognosis is usually poor, even with immediate treatment.

In the acute phase, the intra-cellular oedema secondary to hypoxia, causes an increase in thickness and reflectivity of the inner retinal layers. The retinal nerve fibre layer (NFL), inner plexiform layer (IPL), and ganglion cell layer (GCL) often appears as merged together. The outer retinal layers are also poorly visible due to a back shadowing effect. In the late stages of the disease the ischaemic retinal layers decrease in thickness and atrophy develops where the entire retina may become significantly thinner than normal.

Branch retinal artery occlusion (BRAO)

BRAO is caused by a blockage of the blood flow in a branch retinal artery. The cause can be embolic or inflammatory. The result is ischaemia of the retinal area deprived of blood supply containing oxygen and nutrients (**Figure 16**).

BRAO shows the same appearance of CRAO on OCT, but with abnormalities confined to the area of the retina originally perfused from the occluded artery branch. Intra-cellular oedema secondary to hypoxia, causes an increase in thickness and reflectivity of the inner layers (NFL-IPL-GCL) which appear merged into one layer. The outer retinal layers are poorly visible due to a back shadowing effect. In the late stages of the disease the ischaemic retinal layers reduce in thickness and chronic, atrophic changes develop as with CRAO.

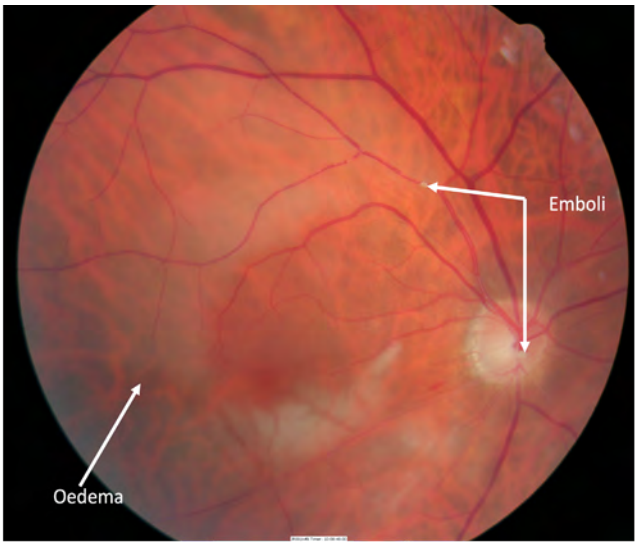

Figure 16: Branch retinal artery occlusion – infero-temporal oedema. Note two emboli (white arrows)

Optical coherence tomography angiography (OCTA)

Although FFA is the gold standard for evaluating the retinal vasculature, it has risks of adverse effects and known limitations in imaging all the layers of the retinal vasculature. Optical coherence tomography angiography (OCTA) can image vessels based on flow characteristics and may provide improved very useful information. Evaluation of OCTA mainly enables interpretation of the retinal vessel density in the different retinal vascular plexus or measuring the size of the foveal avascular zone. It can also visualise collaterals of the regular retinal microvasculature and be used to evaluate the extent of ischaemic areas.

It also provides a new non-invasive method for imaging of the superficial and deep capillary networks in the posterior pole[36] (**Figure 17**). This novel technique has been studied in both normal and pathologic eyes, particularly in patients with retinal vascular disorders such as diabetic retinopathy (see Part 2) and RVO.[37, 38] In RVO, capillary dropout is predominantly observed at the level of the deep capillary plexus (DCP), the perifoveal arcade is disrupted, and the foveal avascular zone (FAZ) enlarges.[39, 40] OCTA was shown to be more effective than FFA in RVO to display and analyse the macular capillary network, the perifoveal capillary arcade and to detect the presence of macular edema. Perifoveal capillary arcade disruption observed using OCTA correlates well with the presence of peripheral non-perfusion as seen on FFA. A significant correlation between quantified macular vascular density on OCTA and peripheral non-perfusion on FFA has recently been reported, with reduced vascular densities in the superficial capillary plexus (SCP) and more so in the deep capillary plexus (DCP), as well as increases in area of the foveal avascular zone in RVO.[41] This is despite the resolution of macular edema, in patients with RVO induced macular oedema who were treated with intravitreal injections of anti-vascular endothelial growth factors (anti-VEGFs) or with intravitreal steroid implant such as Ozurdex (Dexamethasone). Visual acuity closely correlates with SCP and DCP vascular densities as these quantitatively measure the degree of macular ischaemia which in turn determines level of visual function. OCTA is therefore becoming increasingly useful in identifying high-risk retinal vein occlusion patients who may benefit from further evaluation using FFA.[42] In comparison to FFA, OCTA can detect the extent of ischaemic RVO in most cases, but it does miss some.[43]

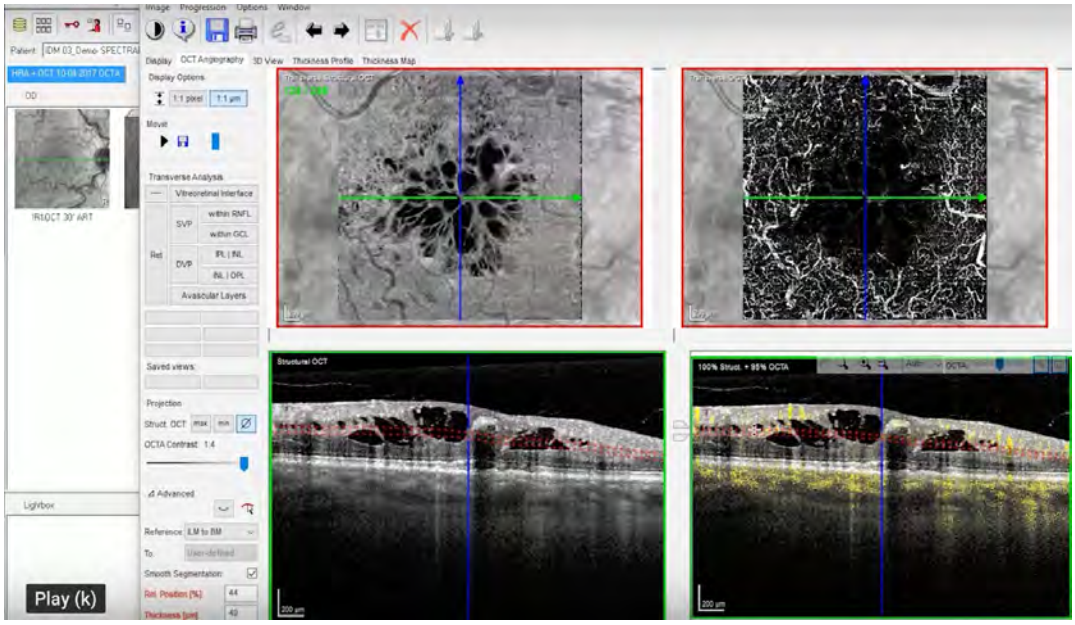

Figure 17: Retinal vein occlusion on OCTA

The exact impact of each feature in OCT and OCT angiography is still to be determined in large randomised clinical trials; therefore, the current recommendation is to monitor disease activity with OCT at regular intervals and retreat based on VA (as the strongest predictor for later VA) and CRT.

Therapeutic Strategies for RVO

Aims of treatment

Treatment for patients with central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO) is directed at eliminating any associated macular oedema, retinal neovascularisation, or anterior segment neovascularisation. Aims of treatment are to:

- Maintain visual acuity by minimising the effects of chronic macular oedema.
- Reducing the risk of bleeding into the vitreous by producing regression of retinal neovascularization.
- Preventing neovascular glaucoma that can occur in eyes with severe disease.
- Manage predisposing risk factors, such as diabetes and hypertension.

Laser therapy

Laser photocoagulation is the standard of care for the treatment of neovascular complications associated with RVO.[44, 45] The mechanism of pan-retinal laser photocoagulation (PRP) has been attributed to the destruction of ischaemic retina, leading to an improved blood supply to the remaining retina and decrease in vascular endothelial growth factor (VEGF) production.[46] Prior to the introduction of anti-VEGF therapy, focal laser photocoagulation was used to treat macular oedema secondary to BRVO.[47] The goal of laser treatment is not to improve vision, but to decrease neovascular changes and prevent the development of neovascular glaucoma.

For patients with CRVO, the CVOS provided evidence for indication, treatment, and follow-up for PRP.[42] Prophylactic PRP does not prevent the development of iris or angle neovascularisation in eyes with extensive capillary non-perfusion (10 or more disc areas). Therefore, PRP is only recommended after iris neovascularisation is detected.

In the past, grid pattern laser treatment has been used with variable success to treat macular oedema secondary to BRVO. Laser treatment for macular oedema secondary to BRVO has been shown to be effective for visual improvement but in view of the availability of anti-VEGF therapy, focal laser photocoagulation should be considered only as a second-line treatment.[48, 49]

Laser treatment is also indicated in CRVO where signs of clinically significant peripheral retinal non-perfusion are present, or as a supplementary treatment to either anti-VEGF or Ozurdez therapy where there has been an incomplete response observed to first line treatment.

Anti-vascular endothelial growth factor agents

The use of anti-vascular endothelial growth factor (VEGF) therapy for macular oedema following RVO is based on the observation that intraretinal VEGF mRNA transcription and intraocular VEGF levels were increased in patients with RVO compared with a control group in various studies.[50, 51] VEGF increases vessel permeability by increasing the phosphorylation of tight junction proteins and is thus an important mediator of the blood-retinal barrier breakdown leading to vascular leakage and macular edema. Therefore, therapy that inhibits VEGF is an effective therapeutic modality targeting the underlying pathogenesis of macular oedema in RVO.

Anti-VEGF intravitreal therapy is now the standard of care for treating RVO using either ranibizumab (Lucentis) and aflibercept (Eylea®). The action of anti-VEGFs results in reduced macular oedema and limits visual loss or improves vision. Anti-VEGF therapy requires frequent monitoring and intravitreal injections to maintain good vision (see below).[52]

Anti-VEGF treatment for BRVO and CRVO is preferred if:

- A younger patient (<50 years)
- Co-existing glaucoma or ocular hypertension
- Known steroid responder
- The patient is willing to commit to frequent hospital appointments for intravitreal injections and monitoring

Corticosteroids

As discussed above the pathogenesis of RVO (both CRVO and BRVO) involves an increase in capillary permeability that results in macular oedema as well as raised venous pressure and hypoxia. This is caused by a breakdown of the blood-retina barrier mediated in part by VEGF and in part by inflammatory cytokines. Although the mean vitreal levels of VEGF are elevated in both disease states (CRVO and BRVO) in one-third of the eyes, these may fall within the normal range despite the presence of macular edema.[53] This finding indicates the existence of VEGF-independent pathways leading to macular oedema that may be the reason why some patients are less responsive to anti-VEGF therapy alone.

The rationale for the use of steroids to treat macular oedema is because of their ability to reduce capillary permeability. Steroids inhibit the expression of the VEGF gene and the metabolic pathways of VEGF, and, in addition, that of inflammatory cytokines. Corticosteroids may also have a neuroprotective effect that is beneficial in eyes with RVO.[54] Several proinflammatory mediators, mostly cytokines such as TNFα, IL-1, MCP-1, and IL17-E, have been shown to be involved in macular oedema secondary to RVO.[55] Another interesting rationale for the use of steroids in RVO is improved retinal oxygenation following Ozurdex injection.[56]

Dexamethasone (Ozurdex)

NICE: TA229
Ozurdex

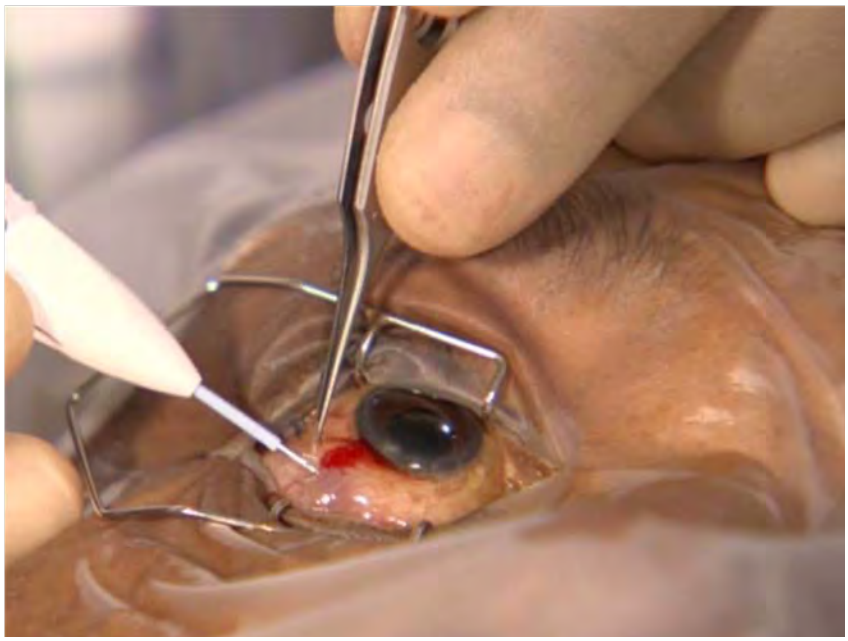

Figure 18: Oxurdex implant being inserted

This is a commonly used intravitreal implant option marketed by Allergan (Irvine California, USA) as Ozurdex (**Figure 18**). The evidence demonstrates that visual outcomes are superior to IVTA described above. The size of needle required for implantation is quite large compared to the fine needles used for injection of anti-VEGF drugs into the eye and can lead to complications if repeated implants are required.

Dexamethasone is a potent corticosteroid that suppresses inflammation in the eye by inhibiting oedema, fibrin deposition, capillary leakage and phagolytic migration. Corticosteroids inhibit the expression of VEGF. Corticosteroids also prevent the release of prostaglandins, some of which are mediators for cystoid macular oedema. The anti-oedematous response to a single intravitreal dexamethasone implant is maximal 1 to 3 months after receiving the implant. Intraocular pressure elevation and cataract formation should also be monitored following the use of intravitreal dexamethasone implants.

Corticosteroids are very effective for treating many patients with RVO, but largely on a second-choice level. Hence, switching to a steroid in non-responders who have already been treated with anti-VEGF (after 3–6 injections, depending on the specific response of each patient) is usually the case. Pseudophakic patients are also preferred as dexamethasone induces cataract, although phakic eyes may in certain cases be undertaken.

Steroids may be considered as a first-line therapy however, for patients who have a recent history (past 3 months) of a major cardiovascular event (myocardial infarction or stroke). All such patients were excluded from all the major anti-VEGF treatment trials, as there is a small risk that anti-VEGFs may exacerbate or complicate any previous cardiovascular event.

Another group of patients in whom corticosteroids may be considered as first-line therapy are those who are unwilling to come for monthly injections (and/or monitoring) in the first 6 months of therapy. However, these patients' IOP still needs to be monitored every 2 to 8 weeks following injection because of the significant risk of a steroid induced IOP response. The effects of Ozurdex can be sustained for around 4 months, when treatment may be repeated so long as it safe to do so and IOP levels are satisfactory.

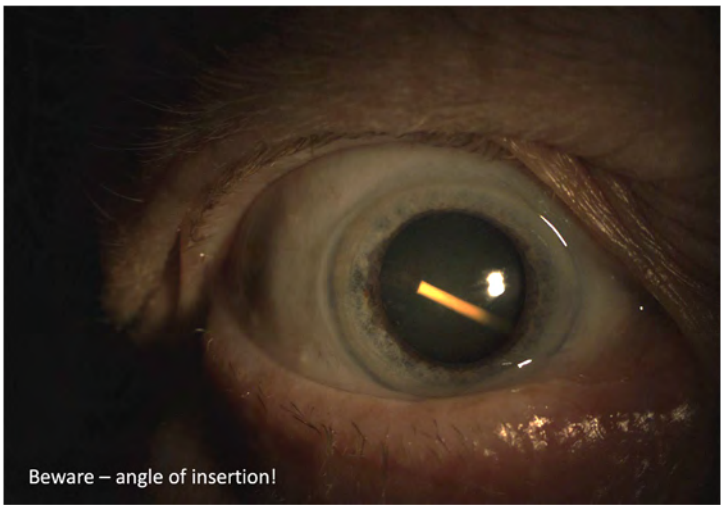

Figure 19: Ozurdex intravitreal implant in anterior vitreous – embedded in lens capsule!!

Appropriate Referral of BRVO and CRVO and factors affecting treatment plans

All patients with BRVO and CRVO should be referred to the Hospital Eye Service (HES) for further assessment. If the RVO is acute (within the past week) an urgent referral to the HES should be made to investigate major underlying causes e.g. hypertension. If the RVO has likely been present for some time (>2 weeks) then less urgent routine referral should be made.

Treatment for BRVO or CRVO should be instigated if there is macular oedema post BRVO or CRVO and no history of spontaneous improvement in visual symptoms. Treatment options include NICE approved anti-VEGF agents (ranibizumab and Aflibercept), Ozurdex and/ or laser.

At each clinic visit OCT and IOP measurement are essential components of the clinical assessment. As discussed earlier, OCTA is also becoming increasingly useful in RVO assessment.

Conclusions

Retinal vein occlusion is an important cause of visual loss, particularly in elderly patients. With an aging population, retinal vein occlusion is likely to become more common. Although we are still not able to predict, prevent or directly treat retinal vein occlusion, there are now robust means of treating the retinal complications of retinal vein occlusion. Previous standards of care often just involved observation. Anti-VEGF agents have made an enormous difference to patients' visual outcomes after retinal vein occlusion. The use of anti-VEGF agents are now first line therapy, whereas corticosteroids are reserved for pseudophakic patients unresponsive to anti-VEGF agents. This is owing to the increased risk of cataract formation and increased IOP. Treatment protocols are however still evolving and the long term outcomes are still unknown.

The role of OCT is now essential in the diagnosis and management of RVO. Structurally, an increase in CRT and the presence of SRF as well as HRF correspond markedly with reduced visual function. This differs from other retinal diseases such as nAMD or DMO, where other, more complex imaging biomarkers seem to have a more distinct impact on vision. Reviewing OCT images for the presence of several key OCT based biomarkers described above is therefore critical in the diagnosis, monitoring and treatment of RVO patients.

The SD-OCT signs of inner retinal ischemia in acute CRVO help the clinician understand the pathophysiology. Regular follow-up with OCT in the first year until the risk of anterior segment neovascularisation (rubeosis) reduces enough that monitoring intervals can be lengthened is important. Even though SD-OCT is an excellent imaging technique in patients with acute RVO there is still no reliable imaging characteristic on baseline SD-OCT that prognosticates subsequent neovascular outcomes.

The community optometrist should be aware of these significant changes in the management and treatment of retinal vein occlusions based increasingly on OCT and OCTA findings, so that timely and appropriate referrals can be made. It is also helpful if the community optometrist is able to discuss current management options with their patients in an informed way so that any advice given is accurate and contemporary, based on OCT imaging

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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.