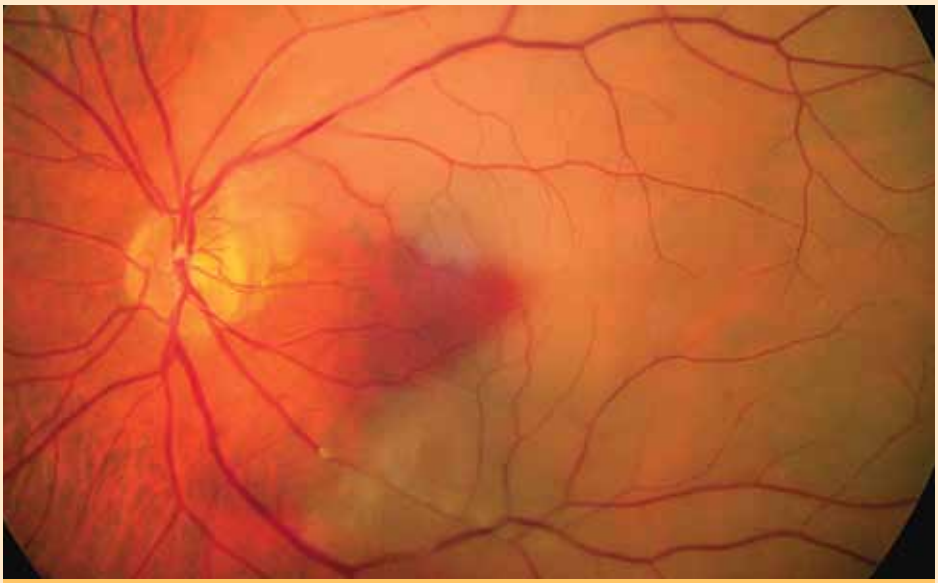
Management & Investigation of Vascular Conditions

MODULE 13 PART 6: CLINICAL OPTOMETRY
COURSE CODE: C-13921 O/D



Louise O'Toole MMedSci FRCSI(Ophth) MRCOphth FEBO

The previous article in this series discussed the investigation and management of retinal conditions that may be seen in clinical optometric practice. The present article covers the more common vascular conditions of the eye that could be encountered. This article does not, however, discuss diabetic retinopathy, since this topic has been comprehensively covered in previous articles in *Optometry Today.*

Hypertensive retinopathy

Hypertension may be classified as either essential hypertension, or the less common malignant hypertension. Essential hypertension is diagnosed when the average systemic blood pressure measures >140mmHg systolic or 90mmHg diastolic, on two subsequent occasions. Malignant hypertension occurs when there is a rapid and severe elevation of blood pressure with a systolic component greater than 200 mmHg.

Systemic arterial hypertension will affect the retinal, choroidal and optic nerve head circulation. Ocular manifestations of chronic essential hypertension include focal constriction and dilatation of the retinal arterioles, "nipping" of the veins by overlying arterioles (Figure 1), an increase in the arteriolar light reflex, tortuosity of the retinal vessels, and loss of transparency of the intra-arterial blood column. There is the Scheie system[1] of classification for grading fundal changes in hypertensive retinopathy (Table 1). In this system of classification, Scheie quantifies the changes of hypertension and arteriolosclerosis separately, in a five-stage classification ranging from normal to the most severe changes visible in the retina. The arteriolar changes of hypertension have been considered to result primarily from vasospasm whereas the arteriolosclerotic changes result from thickening of the arteriolar wall. Retinal microaneurysms occur in a wide variety of retinal vascular diseases, including hypertension, and represent a non-specific finding. There is an association of microaneurysms, cotton wool spots, and non-perfusion of retinal capillaries in hypertension, but these types of changes are also seen in other vascular conditions such as diabetes. Indeed, there may be a crossover between clinical conditions, as chronic hypertensive changes are part of the pathogenesis of the ocular ischaemic syndrome and arterio-venous ("a/v") nipping is frequently seen in cases of branch retinal vein occlusion (see later sections).

Acute hypertension can enter an exaggerated or malignant stage characterised by fibrinoid necrosis of the arterioles and papilloedema (Figure 2). Symptoms include headache, scotoma, diplopia, dimness in vision, and photopsia. Ophthalmic findings in acute malignant hypertensive retinopathy include focal arteriolar narrowing, cotton wool spots, intra-retinal transudates, macular oedema and retinal haemorrhages. There are also choroidal changes seen in malignant hypertension, with focal occlusion of the choriocapillaris leading to necrosis and atrophy of the retinal pigment epithelium (RPE), forming Elschnig's spots. There is vasoconstriction of the posterior ciliary arteries supplying the optic nerve head, which leads to optic disc oedema. In severe hypertensive retinopathy there may be a macular star and even exudative retinal detachments.

Pregnancy-induced hypertension affects both the retinal and choroidal vasculature with clinical findings resembling hypertensive retinopathy. Involvement of the occipital cortex in pregnancy-induced hypertension can lead to transient cortical blindness.

Management of Hypertensive Retinopathy

Treatment of hypertensive retinopathy is aimed at bringing the underlying systemic

hypertension under control in cases of essential, malignant, or pregnancy-induced hypertension, and therefore appropriate referral to the patient's GP is required. However, patients presenting with clear signs of malignant hypertension, most notably the presence of papilloedema, should be referred to the nearest hospital as an emergency.

Retinal arterial occlusion

Retinal arterial occlusions (RAO) may be divided anatomically into central (CRAO) or branch (BRAO) forms, depending on the site of obstruction. A CRAO occurs when the blockage is within the optic nerve substance itself and therefore the site of obstruction is generally not visible on ophthalmoscopy. BRAO occurs when the site of blockage is distal to the laminar cribrosa of the optic nerve, and therefore a cause in the arterial circulation of the retina may be observable. The mean age of patients who experience retinal arterial occlusions is approximately 60 years at presentation. Men appear to be more susceptible than women, with a male to female ratio of 2:1. The cause of retinal arterial obstruction may be either due to an embolus from another site or the development of a thrombosis at the site of obstruction. Vasculitis may present as an arterial occlusion. Structural abnormalities of the eye such as pre-papillary arterial loops or optic disc drusen have also been associated with CRAO. Vasospasm induced by either a migraine or cocaine use has also been linked to retinal arterial occlusion.

Central Retinal Artery Occlusion (CRAO)

In a CRAO, patients experience painless vision loss occurring over several seconds. In some instances, there may be a preceding history of amaurosis fugax. The visual acuity (VA) is reduced to 6/120 or worse. However, if there is a patent cilio-retinal artery present, there may be normal foveal perfusion with no loss of central VA; this artery is a branch of the posterior ciliary artery and therefore macular perfusion and function

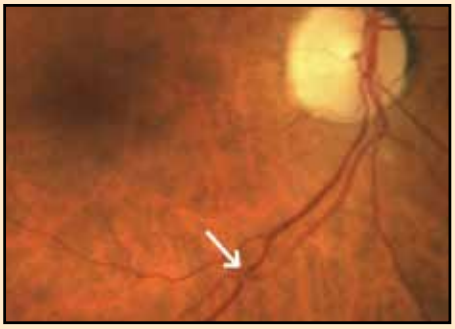


Figure 1
An arteriole crossing a vein, illustrating "a/v" nipping

can be maintained (Figure 3). There will always be a relative afferent pupillary defect (APD) present in the affected eye.

Within the first few minutes to hours after the obstruction, the fundus may appear relatively normal. However, the decreased blood flow to the eye will result in ischaemic whitening of the retina in the territory of the obstructed artery. In the acute phase, the retinal arterioles appeared thin and attenuated and may exhibit segmentation of blood flow termed "box-carring". The characteristic finding of a CRAO is a cherry red spot at the macula. At this point the choroidal circulation is visible and contrasts against the pale white ischaemic retina.

A CRAO is an ophthalmic emergency and patients should receive active treatment in the acute phase. Ocular massage should be attempted either digitally or using a Goldman contact lens. In rare instances, this manipulation can dislodge an obstructing embolus. Massage should increase the intraocular pressure (IOP) for 10 to 15 seconds, followed by a sudden release, which can dislodge the obstruction.

Changes of Hypertension	
Stage 0	Although the patient has diagnosed hypertension, there are no visible retinal vascular abnormalities
Stage I	Diffuse arteriolar narrowing is seen, especially in the smaller vessels. Arteriolar calibre is uniform, with no focal constriction
Stage II	Arteriolar narrowing is more pronounced, and there can be focal areas of arteriolar constriction
Stage III	Both focal and diffuse arteriolar narrowing is more obvious and severe, and retinal haemorrhages may be present
Stage IV	Stage I, II, or III abnormalities may be present, along with retinal oedema, exudates, and optic disc oedema
Changes of Arteriolosclerosis	
Stage 0	Normal
Stage 1	There is broadening of the light reflex from the arteriole, with minimal or no arterio-venous compression
Stage 2	Light reflex changes and crossing changes are more prominent
Stage 3	The arterioles have a "copper wire" appearance and there is more arterio-venous compression
Stage 4	The arterioles have a "silver wire" appearance, and the arterio-venous crossing changes are most severe

Table1
The Scheie Classification of Hypertensive Retinopathy changes

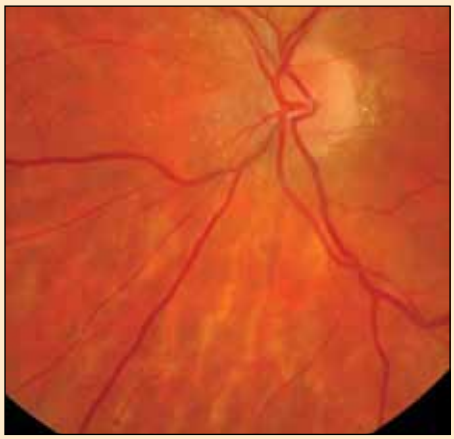


Figure 2
Fundal picture in a patient with a history of malignant hypertension

The use of an oxygen and carbon dioxide (95% O_2, 5% CO_2) mixture (carbogen) has been advocated by some authors but dismissed by others.[2] Hyperbaric oxygen therapy has been shown to have some beneficial effects in cases of CRAO, but only if commenced within hours of the arterial occlusion. An increase in retinal arterial blood flow can be achieved by lowering IOP either physically, by performing a paracentesis, or pharmacologically, by using topical or systemic medication. The most important therapeutic intervention is where the CRAO is associated with giant cell arteritis (GCA) and high dose systemic steroids must be administered before the fellow eye is affected.

The underlying cause of the CRAO needs to be determined and effective treatment of this will reduce the risk of repeat vascular occlusion in the same, or a fellow, eye as well as preventing other associated conditions such as stroke. This will involve the investigation and control of systemic hypertension, diabetes and hypercholesterolaemia. The use of aspirin or warfarin may be indicated. Some patients with carotid artery disease may require carotid endarterectomy. The heart may also act as an embolic source.

Figure 3
Central retinal artery occlusion (CRAO) with sparing of the macula due to the cilio-retinal artery, a branch of the posterior ciliary artery

Branch Retinal Artery Occlusion (BRAO)

A BRAO generally occurs secondary to an embolus. However, it may also be secondary to inflammation or coagulopathies. Cholesterol emboli originate from the ipsilateral carotid artery. They are small and refractile with a yellow-orange colour (Figure 4). Platelet-fibrin emboli are long and white in colour. They are generally associated with carotid or cardiac disease. Calcific emboli are associated with classification of the heart valves or the aorta. Over 90% of BRAOs involve the temporal retinal vessels.

There is generally a history of acute, unilateral, painless visual field loss occurring over several seconds. As with a CRAO, there is retinal whitening but usually localised to the retina surrounding the affected artery. The majority of patients achieve a VA of 6/12 or better with conservative management but a persistent visual field defect may be present.

Retinal arterial macroaneurysms

Retinal arterial macroaneursyms may occur as either saccular dilations or out-pouchings from the arterioles. They may be an incidental observation or else present as a tri-laminar haemorrhage affecting the sub-retinal, retinal and pre-retinal spaces. Retinal arterial macroaneurysms can also present with surrounding retinal oedema. The presence of multiple bilateral arterial aneurysmal abnormalities occurring principally at arterial bifurcations has been described in association with the disorder Idiopathic Retinal Vasculitis, Aneursyms and Neuro-retinitis (IRVAN). The macroaneurysm can spontaneously involute following thrombosis and fibrosis (Figure 5). Eyes with macular oedema, serous elevation of the sensory retina, or exudation should be treated by gentle laser photocoagulation to thrombose the aneurysm.

Central retinal vein occlusion (CRVO)

A central retinal vein occlusion (CRVO) occurs when there is a blockage of blood flow within the central retinal vein; thrombosis is thought to occur at the lamina cribrosa. A CRVO has an increased incidence with increased age and is found in association with diabetes mellitus and hypertension. It is thought that these conditions promote atherosclerosis and the thickened arterial wall compresses the venous wall and promotes thrombosis. There may also be an increased risk of thrombosis in cases of hyperviscosity. Retinal vein thrombosis is a complication of polycythaemia, multiple myeloma and leukaemia. It may be precipitated by diuretics, dehydration, or the oral contraceptive pill in predisposed patients.

If the IOP is raised it increases the risk of venous thrombosis; it has been theorised that the increased pressure bows the lamina cribrosa posteriorly, inducing turbulence, consequent endothelial damage, and thrombosis. Venous thrombosis can also occur if the wall is inflamed, as occurs in episodes of periphlebitis. Inherent ocular predisposing factors include hypermetropia and congenital anomalies of the central retinal vein.

The visual loss associated with a CRVO is painless and may be acute or sub-acute in onset. The extent of retinal ischaemia determines the clinical findings. In an ischaemic CRVO, the VA is generally 6/60 or less and there is a marked relative afferent pupillary defect (RAPD). On

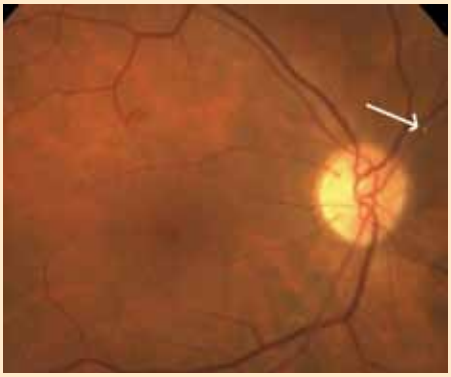


Figure 4
Embolus in a retinal arteriole. This patient requires a full cardiovascular assessment as they are at a high risk of developing a stroke

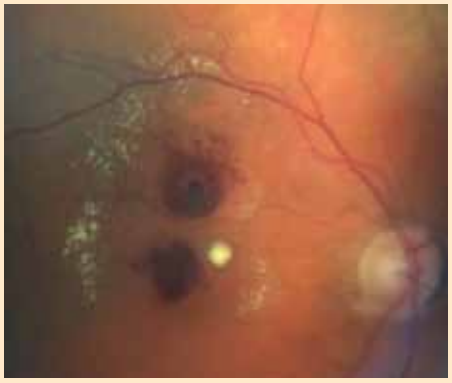


Figure 5
Spontaneous involution of macroaneursyms following thrombosis. There are multiple retinal exudates and tri-laminar haemorrhages.

examination of the posterior pole there are marked haemorrhages in the four quadrants, associated with disc swelling and venous tortuosity (Figure 6). Cotton wool spots may also be a feature. Close follow-up is required as these patients run the risk of developing retinal and iridal neovascularisation (iris rubeosis) consequent to the retinal ischaemia. Non-ischaemic CRVOs are more common and typically the presenting VA is better than 6/60 and the RAPD is not marked. It is important to note that as many as 20% of those classified as non-ischaemic on presentation can subsequently convert to the ischaemic subtype. Visual outcomes are dependent of the degree and persistence of macular oedema.

Branch Vein Occlusion

A branch retinal vein occlusion (BRVO) is three times more common than a CRVO. The supero-temporal vein is most commonly affected (Figure 7). A BRVO occurs most commonly in the seventh decade of life. A BRVO typically occurs at an arterio-venous crossing site. Histological studies have demonstrated that as the branch retinal artery and vein converge on each other, there is fusion of the adventitia - the outermost components of their walls. The adjacent artery is usually narrowed and sclerotic, the obstructed branch vein dilated. Typical features include oedema, and scattered superficial and deep retinal haemorrhages over a triangular retinal sector whose apex is located to the occlusion site. The obstructed vein is characteristically dilated and tortuous distal to the occlusion. Retinal oedema in the involved area is usually present. The involved retina demonstrates variable degrees of scattered superficial and deep retinal haemorrhages, which respect the horizontal midline.

Weeks to months after the onset of BRVO, collateral vessel formation can be observed characteristically located at the edge of the involved area. Collaterals are usually small tortuous venous channels that cross the horizontal raphe mostly temporal to the fovea and drain into the venous circulation of the uninvolved quadrant.

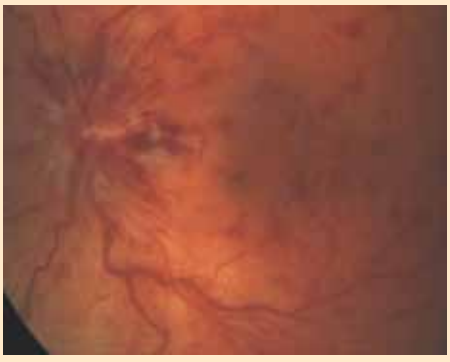


Figure 6
Central Retinal Vein Occlusion (CRVO)

Management of Retinal Vein Occlusions

There are two aims in the management of retinal vein occlusions: the identification of modifiable risk factors and their medical management, and the recognition and management of sight-threatening complications.

Patients presenting with a retinal vein occlusion should have a medical screen to detect hypertension, diabetes, and/or hyperlipidemia. Retinal vein occlusions are associated with an increase in vascular causes of death. Treatment of hypertension reduces the severity of its complications, and additional therapy of aspirin in well-controlled hypertensive subjects given as a prevention therapy reduces cardiovascular event rate. Cholesterol-lowering statins reduce cardiovascular morbidity and mortality. In patients presenting with a retinal vein occlusion under the age of 50 years, a full thrombophilia screen should be included in their medical workup. In females, the contraceptive pill is the most common underlying association, and is contraindicated in patients with retinal vein occlusion.

There is no proven early treatment that will alter the visual prognosis in established CRVO. The main management problem is to differentiate ischaemic from non-ischaemic sub-types. An ischaemic CRVO may develop neovascularisation and if this occurs, pan-retinal photocoagulation is indicated. Macular laser photocoagulation in isolation has been shown not to be of use in treating chronic macular oedema in CRVO. Several clinical trials are in progress looking at the long-term

Figure 7
Branch Retinal Vein Occlusion (BRVO)

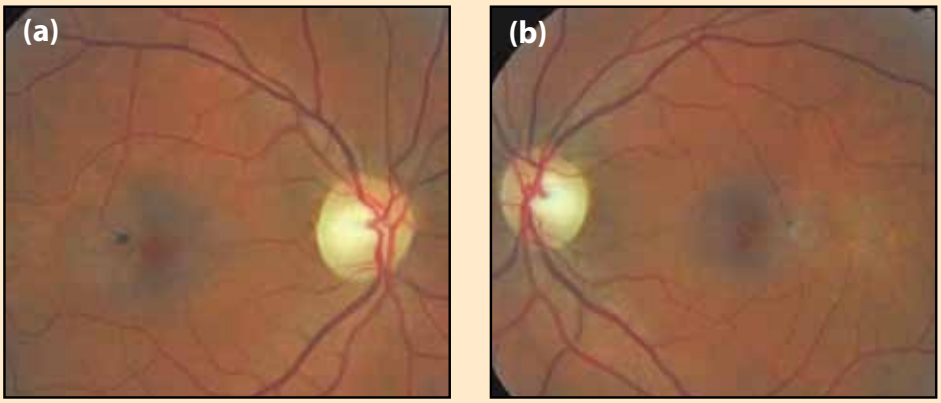


Figure 8
Right and left eye idiopathic juxtafoveolar retinal telangiectasias (IJRT)

outcome of intra-vitreal agents such as ranibizumab (Lucentis), bevacizumab (Avastin) or steroids used either in isolation or in combination for the treatment of macular oedema in CRVO.

Approximately 50% of untreated eyes with BRVO retain a VA of 6/12 or better whilst 25% will have a VA of <6/60. Randomised clinical studies in the laser treatment of macular oedema have demonstrated that a grid pattern of photocoagulation in the distribution of leaking capillaries is beneficial but it is recommended only after a period of three to six months following the initial event and following absorption of the majority of the haemorrhage and if VA is 6/12 or less.[3] Intravitreal triamcinolone acetonide (IVTA) has been shown to be effective in improving vision and reducing macular oedema secondary to BRVO. The complications include cataract formation and increased IOP. The long-term safety and efficacy of IVTA is currently being investigated in a multi-centre clinical trial known as the Standard Care Versus Corticosteroid for Retinal Vein Occlusion Study (SCORE).[4] Secondary neovascularisation can also complicate a BRVO, and if this occurs then sector argon laser photocoagulation to the ischaemic area is indicated.

Ocular Ischaemic Syndrome

The ocular ischaemic syndrome has also been termed "slow flow retinopathy" and occurs when there is severe and chronic arterial hypoperfusion. This condition is more common in men compared to women by a ratio of 2:1, reflecting the higher incidence of atherosclerotic cardiovascular disease in men. It tends to be a condition of elderly people and may be associated with a dull ache. Typically, a 90% or greater stenosis of the ipsilateral carotid arterial system is present in eyes with the ocular ischemic syndrome. Flow abnormalities within the vessel are seen when the stenosis reaches 70%, and it has been demonstrated that a 90% carotid stenosis reduces the ipsilateral central retinal artery perfusion pressure by about 50%. The obstruction can be present within the common carotid or internal carotid artery. A carotid endarterectomy is indicated when there is less than 100% stenosis.

In the ocular ischaemic syndrome central vision tends to be reduced. On examination of the anterior segment there may be neovascularisation of the iris. Posterior segment examination reveals retinal arterial narrowing and straightening, dilated and beaded retinal veins, retinal haemorrhages and microaneurysms. Neovascularization is a complication of chronic retinal ischaemia. Because of the poor arterial perfusion, minor pressure to the globe will cause the retinal arterioles to visibly pulsate. This is a very sensitive and useful clinical observation.

Retinal telangiectasias

The retinal telangiectasias comprise a group of rare idiopathic retinal vascular anomalies characterised by dilatation and tortuosity of retinal vessels, formation of multiple aneurysms, leakage and lipid exudation in the macula, and rarely combined with vascular changes in the retinal periphery. Idiopathic juxtafoveolar retinal telangiectasis (IRT) must be differentiated from other systemic and ocular diseases associated with telangiectasias like diabetic retinopathy, retinal vascular occlusions, Eale's disease, retinopathy of prematurity, or sickle cell retinopathy. There is an association with parafoveal telangiectasia and abnormal glucose tolerance tests.

Idiopathic juxtafoveolar retinal telangiectasia (IJRT)

IJRT encompasses a group of disorders characterised by retinal telangiectasias, superficial retinal crystalline deposits, right-angle venules and intra-retinal pigment plaques (Figure 8). It is capable of causing visual loss in otherwise healthy patients, although there is an association between IJRT and glucose intolerance.

Coats Disease

Coats disease is characterised by telangiectatic retinal vascular abnormalities in association with lipid exudation. The majority of cases are diagnosed before the age of 20 years, with the peak incidence at the end of the first decade. It may present with leukocoria or strabismus. It is invariably unilateral and more common in males compared to females. Complications include secondary cataract, rubeosis iridis, uveitis, secondary glaucoma, retinal detachment and phthisis bulbi (atrophy of non-functional eye). Treatment is either with laser photocoagulation or cryotherapy to the peripheral vascular abnormalities.

Sickle Cell Disease

Sickle cell haemoglobinopathies all share the common feature of an abnormal globin chain, which leads to sickling

of erythrocytes and obstruction of the microcirculation. Sickle vaso-occlusive events are insidious and affect virtually every vascular bed in the eye. There are various subtypes of sickle cell disease; the "SS disease" is associated with more severe systemic disease, whist the "SC disease" tends to cause more advanced ocular disease.

A salmon patch haemorrhage is observed in sickle cell disease and appears as an oval-shaped area of intra-retinal or pre-retinal blood, believed to occur secondary to an obstructed retinal arteriole, which subsequently ruptures. After re-absorption of the salmon patch haemorrhage, the retina may appear entirely normal, without any evidence of residual blood. In the location of the haemorrhage, however, there may also be a faint indentation or depression representing thinning of the inner retina. This appears on ophthalmoscopy as a dimple. Black sunburst lesions have also been associated with intra-retinal haemorrhages; these are flat, round to oval, black patches about 0.5–2mm in size. Venous tortuosity is an early sign of ophthalmic sickle cell disease and "silver wiring" of the arterioles is also a feature.

Sickle cell disease can also cause proliferative retinopathy. Peripheral retinal neovascularisation often assumes a frond-like configuration, resembling the marine invertebrate Gorgonia flabellum (sea fan), but intra-retinal microvascular abnormalities (IRMA) can also be found at sites of active angiogenesis. The majority of the neovascular sea fan formations are found at the interface between perfused and non-perfused peripheral retina, growing toward the ischaemic pre-equatorial retina. Sea fans may have multiple feeding arterioles and draining venules, probably due to their origin from multiple buds of angiogenesis that break through the internal limiting membrane of retina and grow along the surface of the retina at the vitreo-retinal interface. Neovascularisation can result in vitreous haemorrhage or retinal detachment and so photocoagulation is indicated when signs of proliferative retinopathy appear.

Radiation Retinopathy

Radiation retinopathy can be a complication of either localised irradiation (brachytherapy) or external beam irradiation (teletherapy). Brachytherapy is used when radioactive plaques are placed on the sclera to irradiate choroidal tumours. Indications for external beam radiation include the treatment of structures adjacent to the eye – tumours of the orbit or nasopharynx. Radiation retinopathy tends to occur 12 to 18 months following exposure to radiation. Retinal changes include cotton wool spots, retinal haemorrhages and hard exudates. Ischaemic retinal necrosis can involve both the central and peripheral retina. Neovascularisation may be a complication of this condition, which is then treated by argon laser photocoagulation.

Choroidal Haemangioma

A choroidal haemangioma is a benign vascular tumour of the choroid (Figure 9a). There are two distinct subtypes – the diffuse and the circumscribed. The median age of onset of ocular symptoms in patients with the diffuse type is 8 years of age, contrasting with the age of 39 years for the circumscribed type.

In the well-circumscribed type of choroidal haemangioma, there is a well-defined orange red dome-shaped mass visible. Most tumours are 3-9mm in diameter and are typically located in the posterior pole. If external pressure is placed on the globe, it may cause blanching of the tumour.

The diffuse choroidal haemangioma is seen in Sturge-Weber syndrome. It is found in 40% to 55% of Sturge-Weber patients and is bilateral in 23.5% of these patients. The haemangioma is usually ipsilateral to the port wine stain; however, variants have also been reported. Typically, it appears as a diffuse, reddish-orange thickening of the choroid that may cause the fundus to appear darker than the fundus of the fellow eye. The description, "tomato ketchup" fundus, is characteristically applied to the diffuse choroidal haemangioma.

Presenting symptoms include vision loss, foveal distortion or hypermetropia from choroidal thickening. Patients with port wine stains may present asymptomatically when undergoing screenings with dilated fundus examinations. Vision loss in affected patients is secondary to exudative retinal detachment, cystoid degeneration or photoreceptor loss of overlying retina, or fibrous metaplasia of the overlying RPE. Exudative retinal detachment may be seen in up to 50% of patients with Sturge-Weber syndrome and diffuse choroidal haemangioma. Choroidal haemangiomas show a very good treatment response to photodynamic therapy (PDT) (Figure 9b).

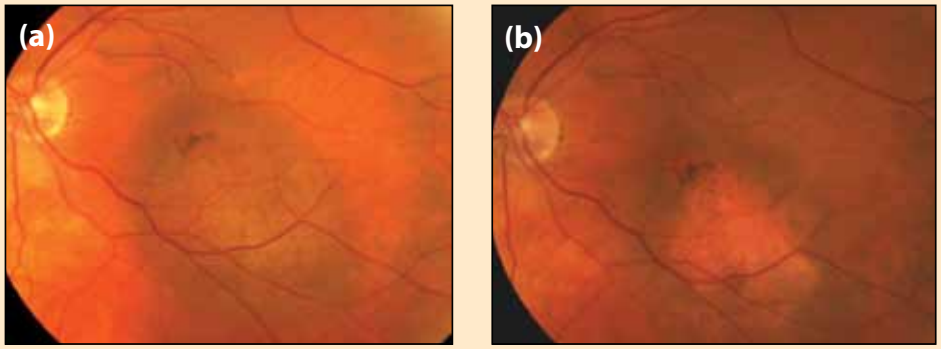


Figure 9(a) and (b)
(a) Choroidal haemangioma, and (b) choroidal haemangioma after treatment with PDT

Optic Nerve Ischaemia

Optic nerve ischaemia most frequently occurs at the optic nerve head, where structural crowding of nerve fibres and reduction of the vascular supply may

combine to impair perfusion to a critical degree and produce optic disc oedema. The most common such syndrome is termed anterior ischaemic optic neuropathy (AION). Generally AION is categorised as either arteritic (AAION) or non-arteritic (NAAION). Less frequently when optic nerve ischaemia affects the intra-orbital portion of the nerve, with no visible disc oedema, this has been termed posterior ischaemic optic neuropathy.

NAAION (Figure 10) is the most common acute optic neuropathy in patients over the age of 50 years. It is thought that insufficiency of the optic disc circulation, exacerbated by structural crowding of nerve fibres and supporting structures at the nerve head, eventually reaches a point at which inadequate oxygenation produces ischaemia and swelling of the disc. Periodic nocturnal hypotension may be an associated risk factor. In contrast to patients with retinal arterial occlusion, those with NAAION are not at an increased risk of early death from systemic vascular disease.

Visual impairment is often first noticed upon awakening. The initial course of visual loss may be static or progressive. Fellow eye involvement is estimated to occur in up to a fifth of patients by five years after onset. The optic disc in the contralateral eye typically is small in diameter and has a small or absent physiological cup. The optic disc oedema in NAAION may be diffuse or segmental, hyperaemic or pale. The disc oedema then resolves and optic disc pallor may become evident as early as six weeks after the vascular insult. The associated visual field defect is typically altitudinal. The VA in one third of patients is normal or slightly reduced. A small number of patients develop progressive visual loss.

AAION is most commonly associated with Giant Cell Arteritis (GCA) but has been reported in association with other inflammatory conditions such as Wegener's granulomatosis. It is an ophthalmic emergency as there is a risk of visual loss in the contralateral eye, as well as systemic associations such as aortic aneurysms, myocardial infarctions and renal failure.

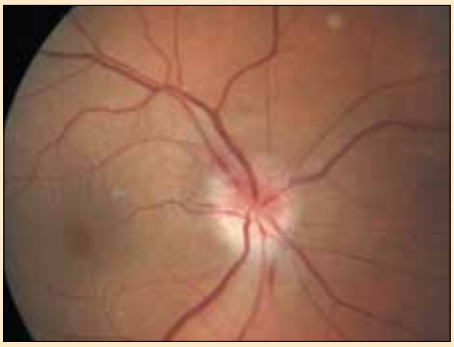


Figure 10
Non-arteritic anterior ischaemic optic neuropathy (NAAION)

GCA presents in the seventh and eighth decades of life. There may be an antecedent history of general malaise and weight loss. If the superficial temporal artery is involved, the patient will complain of temporal tenderness and the superficial artery feels enlarged and nodular. Patients may notice scalp tenderness when brushing their hair. There may be an associated headache, which tends to be localised to the frontal, occipital or temporal areas. Patients may also experience ischaemia of the masseter muscles leading to pain when eating; this is termed jaw claudication. GCA may be the underlying cause of amaurosis fugax or cranial nerve palsy. Any neurological disturbance in elderly people must have GCA ruled out as the underlying cause, as it is a treatable condition. If GCA affects the posterior ciliary arteries, the patient will present with AAION. The vision will be profoundly reduced, there will be a RAPD, the optic disc will appear swollen and chalky white, and there may be associated haemorrhages.

If GCA is suspected, the patient should be referred to the nearest hospital as an emergency. Confirmatory laboratory investigations include a raised Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP). A temporal artery biopsy provides a histological diagnosis. It should be noted that the arteritic process affects the artery in a non-diffuse or "skip lesion" pattern so that a negative biopsy does not rule out GCA.

Treatment of GCA is with high dose systemic steroids. The patient will need to remain on immunosuppressive therapy for months to years and have regular consultations to detect reactivation.

About the Author

Louise O'Toole MMed Sci, FRCSI(Ophth) MRCOphth FEBO is a consultant medical ophthalmologist in the Mater Private Hospital, Eccles Street, Dublin. She is also a lecturer to undergraduate optometrists in the Dublin Institute of Technology She has been involved in teaching on the MSc in Clinical Optometry at City University, London, particularly in the area of Ocular Therapeutics, and has written several articles in the field for *Optometry Today.*

References

See www.optometry.co.uk and search 'references'

Suggested reading

Royal College of Ophthalmologists Retinal Vein Occlusion (RVO) Interim Guidelines February 2009 http://www.rcophth.ac.uk/docs/publications/published-guidelines/RVO_Guidelines_Feb_2009.pdf

MSc in Clinical Optometry

CITY UNIVERSITY and OT have joined forces allowing readers to achieve CET points through to a full Masters in Clinical Optometry. MSc courses running at City University include: Principles of Therapeutics (apply anytime – web-based), Independent Prescribing (June 20-22nd 2010), Glaucoma (July 18-20 2010), Posterior Segment Eye Disease (September 5- 7 2010) and Binocular Vision (November 14-16 2010). For further information please contact Dr Michelle L Hennelly by emailing (m.hennelly@city.ac.uk) or call 0207 040 8352.

Module questions

Course code: C-13921 O/D

1. Which of the following statements about hypertensive retinopathy is FALSE?
a) The arterioles may assume a "silver wire" appearance
b) The arterioles may assume a "copper wire" appearance
c) Retinal haemorrhages are a feature
d) Optic disc oedema is unilateral

2. Which of the following statements about retinal arterial occlusions is FALSE?
a) Men tend to be more frequently affected compared to women
b) They may occur secondary to an embolus
c) Vasculitis can cause an arterial occlusion
d) They are not associated with Giant Cell Arteritis

3. All of the following are treatments for a central retinal artery occlusion EXCEPT:
a) Ocular massage
b) Physical lowering of intraocular pressure
c) Use of carbogen
d) Anti-VEGF agents

4. Which of the following statements about retinal arterial macroaneursyms is FALSE?
a) They are more common in hypertensive patients
b) They are treated by pan-retinal photocoagulation
c) They may spontaneously rupture
d) They can chronically leak

5. Which of the following statements about a central retinal vein occlusion is FALSE?
a) If it is ischaemic, there will be no RAPD
b) It is associated with disc swelling/oedema
c) It has retinal haemorrhages involving the four quadrants of the fundus
d) It may lead to neovascularisation

6. Which of the following statements about the ocular ischaemic syndrome is FALSE?
a) It is associated with ipsilateral carotid stenosis
b) It is associated with retinal arterial pulsations
c) It can have rubeosis as a feature
d) Like a CRVO it presents with tortuosity of the venules

7. Which of the following statements about Coats disease is FALSE?
a) It is an important differential for leukocoria
b) It is typically bilateral
c) It is more common in males than females
d) It may cause an exudative retinal detachment

8. Features of sickle cell retinopathy include all of the following EXCEPT:
a) Elschnig spots
b) Salmon patches
c) Sea fans
d) Starburst exudates

9. Which of the following statements about choroidal haemangiomas is FALSE?
a) Diffuse haemangiomas are a feature of the Sturge Weber syndrome
b) They can be effectively treated with PDT
c) They cause an axial myopia
d) They may present before adolescence

10. Which of the following statements about non-arteritic ischaemic optic neuropathy is FALSE?
a) It is treated with high dose systemic steroids
b) It presents with disc swelling/oedema
c) It is associated with postural hypotension
d) It has an associated visual field defect

11. Which of the following statements about arteritic ischaemic optic neuropathy is FALSE?
a) It occurs when there is occlusion of the posterior ciliary artery
b) The disc is swollen and pale
c) There is a RAPD
d) There is no risk to the fellow eye

12. Which of the following statements about Giant Cell Arteritis is FALSE?
a) It may present with jaw claudication
b) It may cause temporal tenderness
c) It causes intracranial aneurysms
d) It responds well to steroids

PLEASE NOTE There is only one correct answer. All CET is now FREE. Enter online. Please complete online by midnight on July 7 2010 - You will be unable to submit exams after this date – answers to the module will be published on www.optometry.co.uk