Cataract in diabetes

Diabetic patients develop cataract more frequently than non-diabetic patients and at a younger age. Diabetes is the most common risk factor for cataracts in the developed world. It has been estimated that 10% - 20% of all cataract operations carried out in the UK are on diabetic patients.

Cataract in diabetics is significant for a number of reasons:

1. It impairs the recognition of sight-threatening diabetic retinopathy, both in the screening situation and in the detection of subtle macular oedema.
2. There is an increased surgical complication rate of cataract surgery in diabetics especially those with DR.
3. There is a risk that retinopathy can deteriorate and that diabetic maculopathy (DM) in particular can be exacerbated by cataract surgery.

It is very important to detect any DR present and in particular any DM. Patients with diabetic maculopathy should be closely monitored prior to and following cataract surgery. Ideally, maculopathy should be fully treated with any oedema fully resolved prior to cataract surgery, but for some cases macular oedema persists despite treatment at the time of cataract surgery. Essentially, if there is any DR present it should be treated appropriately prior to surgery. Patients should be offered cataract surgery when they become symptomatic and preferably before the view of the fundus deteriorates to a significant level.

Modern cataract surgery using small incision phacoemulsification has greatly reduced the prevalence of surgical complications in diabetic patients, but they can still occur and clinicians need to be aware of their potential occurrence. The following are the most common complications following cataract surgery in diabetics:

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**Outline:**

Co-existing diabetes and other eye disease can make diagnosis and decision-making more problematic, and treatment more challenging. This article provides a research-based exploration of co-existing eye disease in diabetics, and what the eyecare practitioner needs to know in order to make appropriate decisions for patient management, advice and referral.

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**Introduction**

Part 5 of this diabetic eyecare series discusses some of the ocular diseases and conditions that may co-exist in patients with diabetic retinopathy (DR). The most common is cataract (Figure 1).
Post-operative uveitis

Post-operative uveitis, particularly anterior uveitis, is more common in diabetics because of the increased levels of vascular-endothelial growth factor (VEGF) in diabetic eyes, especially in those with significant DR (figure 2). Patients with poorer metabolic control and uncontrolled glucose levels also have increased VEGF levels. The more inflammation there is after diabetic cataract surgery, the greater the potential for retinopathy exacerbation to occur. Surgical trauma therefore should be kept to the absolute minimum. The use of topical steroids such as prednisolone acetate 1% six to eight times daily starting one week before cataract surgery is often recommended in high risk cases as well as post surgery for longer than normal periods, then tapered slowly e.g. 6x per day for a week, then; 4x, 3x, 2x.

Endophthalmitis

Endophthalmitis means bacterial or fungal infection inside the eye involving the vitreous and/or aqueous humours (figure 3). The most common pathogens in endophthalmitis vary considerably depending on the surgical procedure involved. Coagulase-negative staphylococci are the most common causes of post-cataract endophthalmitis. Diabetes is probably a risk factor for endophthalmitis with the risk being approximately 1 case in every 1000 cataract operations. Presence of pre-existing DM is also a risk factor for endophthalmitis. Acute endophthalmitis is a medical emergency. The most important component of treatment is the intravitreal injection of antibiotics, along with vitrectomy in severe cases. Systemic antibiotics may also be used. Repeated intravitreal injections of antibiotics may be necessary if there is no response to the initial therapy. Many eyes that receive prompt and appropriate treatment will recover useful vision.

Posterior capsular opacity and anterior capsule phimosis

Both these are more common in diabetics, especially in patients with DR. To reduce the risk of these complications a large capsulorhexis (to reduce the risk of anterior capsule phimosis) just overlapping the optic edges should be performed and a large optic (6mm or more) acrylic lens preferably used.

Iris and irido-corneal angle rubeosis

This may occur secondary to the loss of the lens barrier and inflammation associated with surgery, particularly where lens capsule integrity is lost owing to e.g. rupture. Usually retinopathy has to be at least at the level of severe non-proliferative diabetic retinopathy (NPDR) or proliferative diabetic retinopathy (PDR) to put a patient at risk of experiencing this serious complication. Surgeons should be particularly wary of ischaemic eyes with very severe background retinopathy changes, but no new vessels in the presence of a posterior vitreous detachment. These eyes should be treated with pan-retinal photocoagulation (PRP) prior to surgery. Note that rubeosis can be very subtle and should be looked for carefully on the iris-pupil margin.

Progression of DR post-cataract surgery

There is evidence that DR progression can occur after cataract surgery, especially if complicated. The apparent progression in diabetic retinopathy after modern cataract surgery seems to reflect the masking of low grades of diabetic retinopathy by preoperative lens opacities. Active DR is also a very important determiate factor of visual outcome. Treated DR is in general far more likely to have a successful outcome than untreated DR at the time of surgery. Any laser treatment should therefore be undertaken prior to cataract surgery if at all possible.

Vitreous haemorrhage

This can occur following cataract surgery secondary to changes in the vitreous gel with increased vitreous syneresis. This is thought to lead to an earlier onset of posterior hyaloid separation from the retina and consequently vitreo-retinal traction on any new vessels present (figure 4).

Irvine-Gass related cystoid macular oedema (CMO) and exacerbation of diabetic macular oedema

CMO is more common in patients with diabetes, especially if there is any DR present (figure 3). Diabetic macular oedema can also be precipitated by cataract surgery and may be undiagnosed prior to cataract surgery, because of the cataract itself. Diabetic macular oedema (DMO) on its own is the main determinant of poor vision following cataract surgery in diabetic patients. It increases by 6x the risk of a Snellen visual acuity less than 6/12. In patients with stable diabetic retinopathy without significant macular oedema, intravitreal ranibizumab (Lucentis) injection at the time of cataract surgery may prevent the postoperative worsening of macular oedema and may improve the final visual outcome without affecting safety.
Management of patients with diabetes undergoing cataract surgery

The patient’s glycaemic and hypertensive control should be optimised prior to cataract surgery wherever possible. Diabetic patients with no DR can be treated as normal patients with cataract surgery. Patients with mild or moderate retinopathy with no macular oedema may be treated post-operatively with extended use of steroids e.g. Predforte. Wherever possible patients with macular oedema should have this maximally treated prior to cataract surgery. Similarly patients with active PDR or marked pre-proliferative features should be treated maximally with PRP prior to cataract surgery. If PRP is not possible because of the density of the cataract then intra-operative PRP should be considered. For patients with active PDR with features suggesting vitrectomy may be needed (i.e. traction threatening/ affecting the fovea or vitreous haemorrhage) a combined cataract/ vitrectomy surgical approach should be considered.

Primary open angle glaucoma (POAG)

Open-angle glaucoma may be present especially if there is a family history of glaucoma in diabetic patients. The optic disc should be assessed (including disc photography) as in the non-diabetic patient with visual field and IOP monitoring. The visual fields of diabetic patients who have had laser treatment, particularly if extensive, may be difficult to assess. Treatment of glaucoma in diabetic patients is similar to that in the non-diabetic.

Corneal and ocular surface disorders in diabetes

Decreased corneal sensitivity and improper neural regulation in the diabetic cornea apparently leads to delayed epithelial wound healing and occurrence of recurrent erosions and persistent epithelial defects. Abnormalities of the epithelium and basement membrane may occur in diabetic patients with poor adhesiveness of the corneal basement membrane complex. This results from impaired hemidesmosome formation.

The risk of contact lens associated bacterial keratitis in diabetics is increased, mainly explained by an impaired immune response in diabetics leading to an increased susceptibility to infection.

Poor metabolic control, laser PRP treatment, and PDR are high risk factors for ocular surface disorders in type 2 diabetes. These patients should be followed more carefully, and should be referred to an ophthalmologist when required.

Anterior adnexae related conditions

Dry eyes are up to 50% more common in diabetic patients compared with non-diabetics. Other conditions such as xanthelasma, styies and blepharitis are also more common. Poor metabolic control and proliferative retinopathy are high risk factors for ocular surface disorders in type 2 diabetes and require careful follow up.

Neuro-ophthalmological manifestations in diabetes

Neuro-ophthalmological manifestations of diabetes mellitus may involve defects related to vascular, neuropathic or metabolic changes:

Diabetic papillopathy

In diabetic papillopathy (DP) there is a unilateral or sometimes bilateral transient swelling of the optic discs in patients with longstanding DM, but visual acuity remains relatively good (figure 6). Usually this condition resolves spontaneously over a period of several months. It is important to differentiate DP from more serious disorders of the optic nerve head. In cases of bilateral optic nerve involvement, malignant hypertension and papilloedema must first be ruled out by measuring blood pressure, undertaking neuro-imaging and then lumbar puncture, respectively. The pathogenesis remains largely unknown, but there has been evidence suggestive of its associations with a small cup/disc ratio and rapid reduction in glycaemia. There is no validated therapy for diabetic papillopathy, however, current case reports have shown promising results after local injections of corticosteroids as well as bevacizumab (Avastin).

Non-arteritic ischaemic optic neuropathy (NAION)

Non-arteritic ischaemic optic neuropathy (NAION) is a common cause of optic disc oedema, particularly in older patients. Risk factors for the development of NAION include ischaemic heart disease, hypercholesterolaemia and diabetes mellitus (DM). In patients with DM who develop NAION, this typically presents with quite profound sudden onset loss of vision, acquired colour vision defects (dyschromatopsia), optic disc oedema, moderate or marked relative afferent papillary defect and usually altitudinal visual field defects. Approximately 30%-40% of patients with NAION improve spontaneously. Duration of diabetes is an important risk factor for both presence and severity of DR in subjects with NAION.

Ocular motor disorders

The most common neuro-ophthalmological manifestation of DM is diplopia, which most often results from ischaemia to the third, fourth or sixth cranial nerves.

1. Sixth (VI) nerve palsy

The most common reason for a neuro-ophthalmological referral in a diabetic patient is ischaemic sixth (VI) nerve palsy. A VI nerve palsy presents with convergent strabismus and sudden onset horizontal diplopia. There are a number of possible causes of VI nerve palsy including hypertension, compression (e.g. acoustic neuroma, nasopharyngeal tumour), trauma and ophthalmoplegic migraine.

VI nerve palsy may also be a sign of raised intracranial pressure associated with posterior fossa tumours or benign intracranial hypertension. Hypertension is the commonest underlying association in diabetics.

An isolated VI nerve palsy is often due to focal small vessel occlusion with ischaemia of the VI nerve. Small brain stem infarctions are increasingly recognised as a cause of isolated ocular motor and vestibular nerve palsies in diabetic and/ or hypertensive patients. Recovery with time is to be expected. However if there is failure to recover, then other investigations should be considered e.g. neuro-imaging.

2. Third (III) nerve palsy

This usually presents with sudden onset diplopia, ptosis and pain may be present.

When a patient presents with isolated third (III) nerve palsy, the cause is usually an ischaemic event or compression from an expanding aneurysm, which may rupture imminently causing mid-brain haemorrhaging. It is always important to look for other cranial nerve involvement and to investigate these thoroughly.

Until proven otherwise, a III nerve palsy with pupillary involvement i.e. a dilated pupil should be assumed to be caused by an intracranial
aneurysm. This requires urgent management and neuro-surgical assessment. Anisocoria of greater than 2mm should always be fully investigated using neuro-imaging whenever a CN III nerve palsy is suspected.

Diabetic III nerve palsies may be caused by ischaemia in the mid-brain or along the peripheral nerve. Most commonly however, the location of the ischaemia is peripheral rather than in the mid-brain.

3. Fourth (IV) nerve palsy
Recent onset vertical diplopia is the most common presenting symptom with IV nerve palsy, particularly when the patient is attempting to read. This condition is usually painless. On cover test, the hyper-deviation will increase in contralateral gaze, reduce in ipsilateral gaze, increase on ipsilateral head tilt, and decrease on contralateral head tilt. Other symptoms may also include:

- horizontal diplopia
- a head tilt contralateral to the affected superior oblique muscle
- the patient’s chin may also be deviated downwards
- visual acuity is unaffected and there is very rarely pain
- In bilateral cranial nerve IV palsy, the patient will present with a hyper-deviation which reverses in opposite gaze.

In diabetics a IV nerve palsy is commonly caused by vascular infarct. Usually this will spontaneously resolve over a period of three to six months and the patient will not require further management beyond periodic observation or press-on Fresnel prisms.

4. Ocular motility defects
In addition to having cranial neuropathies, patients with DM may present with a number of rare ocular motility defects from brain stem ischaemia.

5. Ischaemic stroke
Ischaemic stroke, often seen in diabetics, is the most common type of stroke, where an artery is blocked by a blood clot, which interrupts the brain’s blood supply. This may be due to a cerebral thrombosis (sometimes called a thrombotic stroke) where a blood clot forms in the main artery leading to the brain, or to a cerebral embolism (sometimes called an embolic stroke) in which a blood clot forms elsewhere in the body and is swept into the arteries serving the brain.

Transient neurological deficits have been associated with hypoglycaemia, which may be seen in children with type 1 diabetes.

On administration of glucose, the prognosis is usually very good and further investigation is not indicated. Acute hypoglycaemia also has a number of effects on vision which include blurred vision, impaired information processing and reduced contrast sensitivity.

Ocular ischaemic syndrome
Ocular ischaemic syndrome refers to the changes in the anterior and posterior segments of the eye that result from ischaemia, usually with carotid artery occlusion and poor collateral flow. Visual loss of varying degrees occurs and iris neovascularisation is quite common. Most patients with ocular ischaemic syndrome also have diabetes.

Vascular occlusions
Vascular events are common in diabetics (with or without DR), including retinal artery occlusion, vein occlusion and ischaemic optic neuropathy. This may be due to an increase of cardiovascular risk factors (e.g. 70% of type II diabetics are hypertensive).

Medical management in general includes the following:

- good metabolic control of diabetes including weight control, healthy diet and regular exercise
- stopping smoking
- control of hypertension
- prescribing statins for blood lipid control e.g. Atorvastatin
- treatment of co-existing cardiac problems e.g. atrial fibrillation - consider electrocardiogram (ECG)
- exclude carotid disease - consider carotid Doppler scan
- treatment of blood hyperviscosity if present e.g. aspirin
- anti-platelet drugs e.g. aspirin
- exclude temporal arteritis in ischaemic optic neuropathy or retinal arterial occlusion.

Retinal vein occlusion
There are two aims in the management of retinal vein occlusion: the identification of modifiable risk factors and their medical management and the recognition and management of sight-threatening complications (figure 7). All patients with retinal vein occlusions should be referred at least to their GP for a full cardiovascular evaluation. The following investigations should now
be conducted according to the Royal College of Ophthalmologists recently published new guidelines:

- full blood count (FBC)
- erythrocyte sedimentation rate (ESR)
- blood glucose
- urea and electrolytes (U and Es)
- cholesterol
- thyroid function tests (TFTs)
- plasma protein electrophoresis
- blood pressure

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

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

1. **Central retinal vein occlusion (CRVO)**

Non-ischaemic CRVO is the most common type accounting for about 75% of all cases (figure 7). It presents with a moderate loss of vision. Mild to moderate retinal haemorrhages are seen distributed throughout all four quadrants, but cotton wool spots are usually absent. Mild to moderate disc oedema and/or macular oedema may also be present. The prognosis is reasonably good in about 50% of cases with vision returning to normal or near normal. Cystoid macular oedema is the most common cause of reduced vision in such cases.

Signs of ischaemic CRVO include very reduced vision, RAPD, a very hemorrhagic fundus appearance with cotton wool spots, retinal new vessels and there may even be vitreous haemorrhage. CRVO with retinopathy and neovascularisation, vitreous haemorrhage, markedly raised IOP or a painful eye, usually require urgent laser treatment. Other treatment such as diode laser to the ciliary body may be considered if the media is not clear. Ultrasound may also be helpful where there is retinopathy and dense cataract without a fundus view in order to rule out other significant pathology such as an intraocular tumour.

Ophthalmological management for CRVO may include photocoagulation (PRP) to prevent or treat new vessel formation and neovascular glaucoma or, more recently, Ozurdex® (Dexamethasone - a steroid) intra-vitreal implants. Photocoagulation does not alter the visual prognosis in CRVO and macular oedema.

Topical steroids and atropine may be indicated in order to help keep the eye comfortable in a patient with painful neovascular glaucoma.

2. **Branch retinal vein occlusion (BRVO)**

The diagnosis of branch retinal vein occlusion is clinical. In doubtful cases, especially with small BRVOs, fluorescein angiography may be indicated to confirm the diagnosis. Fluorescein angiography and optical coherence tomography (OCT) is particularly useful in determining the extent of macular oedema and ischaemia. Current treatments for macular oedema secondary to BRVO now include Ozurdex intravitreal implants or Ranibizumab (Lucentis) intravitreal injections, in addition to grid/PRP laser treatment.

**Retinal emboli**

Emboli are fairly common in DR and are often picked up during routine screening (figure 8). They appear as small white, grey or creamy spots, lumps or plaques within the retinal arterioles. There are three main types of retinal emboli, namely:

- cholesterol emboli
- fibrinoplatelet emboli
- calcific emboli.

They often present at a junction or branch of the vessel and calcific emboli are the most likely to cause visual symptoms as they often block the central retinal artery near to the optic disc. The most common kind of embolus is a cholesterol embolus. They are usually easy to determine from a single exudate because they are present within the vessel rather than scattered throughout the retina. Many diabetic patients will have a raised cholesterol level and many will be on some form of statin and aspirin. Cardiovascular evaluation and treatment should be carried out to reduce the risk of further cardiovascular events.

**Retinal artery occlusion (central and branch)**

With central retinal artery occlusion (CRAO) there is usually a sudden painless loss of vision and giant cell arteritis (GCA) should always be considered in all cases of CRAO affecting older patients. Therefore an urgent erythrocyte sedimentation rate (ESR) should be arranged, particularly if over the age of 65 years and if there are other typical symptoms of GCA. A general cardiovascular examination is required to exclude remedial cardiovascular risk factors such as...
atrial fibrillation, carotid bruits, heart murmurs and hypertension. If embolic disease is the cause then carotid ultrasound is indicated and possibly a cardiac echocardiogram. Other relevant investigations may include:

- full blood count (FBC)
- glucose
- fasting cholesterol
- urea and electrolytes (U&Es)
- thrombophilia coagulation screening

If the patient is a smoker they should be encouraged to stop. They can be referred if appropriate, to their GP for smoking cessation advice and support.

If the branch or central retinal artery occlusion has occurred within the last 24 hours, intravenous (IV) Diamox 500mg may be administered in addition to ocular massage. Paracentesis of the anterior chamber may also be considered if symptoms are less than 12 hours duration. If the sudden vision loss was more than 24 hours, then recovery of vision is very unlikely. Low dose aspirin is usually commenced by the patient.

Neovascular glaucoma (NVG)

In NVG elevation of IOP is caused by synechial angle closure through contraction of fibrovascular tissue. The common factor in all eyes with NVG is severe, diffuse and chronic retinal ischaemia. Ischaemic CRVO is the commonest cause, followed by patients with long standing diabetes who also have proliferative DR.

Ghost cell glaucoma in diabetes

This type of secondary glaucoma is associated with degenerated intraocular red blood cells that cause trabecular obstruction. It can occur after vitreous haemorrhage in any eye with communication between vitreous and anterior segment with disruption of the anterior hyaloid face (especially in eyes already aphakic). After approximately 2 weeks in the vitreous, the haemoglobin leaks out of the red blood cells, turning them into degenerated “ghost cells” which then pass through any defect in the anterior vitreous face into the anterior chamber, thus obstructing the trabecular meshwork.

Choroidal naevi

Choroidal naevi are fairly common occurring in approximately 5% of screened DR patients and are sub-retinal pigment patches found in the choroidal layer. They are usually flat, oval/circular and greyish in appearance. However naevi greater than 3 to 4 disc diameters in size should be considered for referral to evaluate any signs of choroidal melanoma (figure 9).

Dry age-related macular degeneration

It is important to remember that diabetic patients are not immune from macular degeneration whether or not retinopathy is present. Distinguishing exudates from drusen can be challenging but the key differences are listed in table 1.

Table 1

<table>
<thead>
<tr>
<th>Exudates</th>
<th>Drusen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other signs of DR especially microaneurysms</td>
<td>Deeper</td>
</tr>
<tr>
<td>Yellow hard appearance</td>
<td>Creamy/white</td>
</tr>
<tr>
<td>Streaks</td>
<td>Round</td>
</tr>
<tr>
<td>Circinate/semi-circinate pattern</td>
<td>Not typically circinate</td>
</tr>
</tbody>
</table>

The management of wet (exudative) macular degeneration is similar to that in the non-diabetic patient.

Cellophane maculopathy (epiretinal membrane (REM))

This may occur in diabetes with or without DR. This is caused by contraction of fibrous tissue on the surface of macula, usually because of the presence of a posterior vitreous detachment (PVD). Vision may be distorted and can be classified according to the severity of retinal distortion, associated slit lamp biomicroscopic changes and associated ocular disorders. If symptomatic the patient may be a candidate for pars plana vitrectomy and membrane peel and so should be referred for a vitre-retinal opinion. If the symptoms are mild or if VA is greater than logMAR 0.5 (Snellen 0.50) then surgery is best avoided.
Chorio-retinal folds

These may appear as horizontal, vertical or oblique folds that are parallel in appearance. On slit-lamp the crest (peak) of the fold appears yellow and the valley (trough) appears darker. These are often located supero-temporal and circumferential to the macula. If unilateral they are often associated with orbital masses. They can occur bilaterally in hypermetropes and may be asymptomatic. The management depends entirely on the presumed cause.

Asteroid hyalosis

Asteroid hyalosis comprises of lipid droplets suspended in the vitreous gel. If severe enough these can affect vision which in extreme cases may justify vitrectomy surgery. As far as screening for DR these can obscure the retinal image and make distinguishing exudates, if present, from asteroid quite difficult. The simplest way to distinguish between the two is to take a second photograph having asked the patient to move their eyes between shots. Asteroid droplets will move position whereas exudates will remain in their fixed location on the retinal image.

Conclusions

While examining diabetics with DR there are a wide variety of other medical retinal and anterior segment conditions that may give rise to the need for referral. However not all these conditions do actually need to be referred!

References

GOC’s Enhanced CET Scheme

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

When you have completed your reading, close this window and return to iLearn/Spectrum to choose your practitioner group and either interactive or non-interactive CET quiz.

If you choose ‘non-interactive’ you have to pass (>60%) a 6-question multiple choice quiz. If you choose ‘interactive’ you must pass a MCQ quiz and complete a further 30-minute discussion with a colleague, and upload a short summary of your discussion and reflections within 30 days. Note you must complete both tasks before your CET can be awarded. If you want the CET counted within a calendar year make sure you submit the online record of discussion and remind your colleague to verify it online at least 2 weeks before the end of the year.

Further instructions for interactive learning are as follows:

The following steps must be completed within 30 days of completing the MCQ quiz:

1. Discuss the interactive questions below with a registered colleague. Note if you are an optometrist, the colleague must also be an optometrist. If you are a dispensing optician, the colleague may be a dispensing optician, a contact lens optician or an optometrist. The discussion should be in a quiet environment where you are not interrupted for at least 30 minutes. Discuss the set questions and record a summary of the output of your discussion. Please ensure to create a paper copy of your record, sign and date the document and keep it safely stored in case your CET is audited in future by the GOC.

2. In the event of an audit we need to be able to show the GOC that the interaction has taken place in accordance with the instructions. Therefore, before you can be given points for this activity you must, within 30 days, record your answers to the set questions in the online Discussion Record and Reflection form (link provided on iLearn/Spectrum).

3. You will be asked for the GOC number, name and email address of the colleague who has completed the interaction with you, so please have those ready. Your colleague will be contacted by email (so please make sure you enter their correct email address) and will be sent a link to verify the interaction took place.

4. You can only be awarded interactive CET points if these steps are completed within 30 days.

The learning objectives for the interactive article are:

8.1.3 Dispensing opticians will have an evidence-based understanding of treatments for a range of eye conditions co-existing with diabetic eye disease enabling them to give advice /answer patients’ questions.

6.1.10 Optometrists will have an understanding of how the management and advice to patients with diabetic eye disease may change in the presence of co-existing ocular disease.

6.1.6 Optometrists will have an evidence-based understanding of management and post-operative complications of cataract patients with co-existing diabetes.

2.1.7 Therapeutic optometrists will have an evidence-based understanding of appropriate referral and treatment for patients with co-existing eye disease and diabetes.

2.7.5 Optometrists will have an evidence-based understanding of appropriate referral and treatment for patients with co-existing eye disease and diabetes.

The discussion tasks for the interactive learning option are as follows.

1. Discuss with your colleague the different types of surgical complications that a patient with diabetic retinopathy may potentially encounter during or after cataract surgery. Discuss also what strategies may be used to minimise the risks of these complications.

2. Discuss with your colleague most common neuro-ophthalmological conditions which may co-exist in patients with diabetic eye disease, and their causes.

3. Reflecting on the reading and discussion...
   a. what are the main things you learned from the reading?
   b. how will you apply this learning in your future practice?
   c. has this module identified for you any areas in which you wish to pursue further learning?