



Diabetes: Indications for treatment

Dr Brinda Muthusamy MRCP MRCOphth



It is estimated that there are currently 180 million people worldwide affected with diabetes. Diabetes is the leading cause of blindness for people under the age of 65 years in the Developing World. The prevalence and incidence of diabetes in our modern society is currently on the increase. It is important to recognise this and the impact it has on the provision of care to the diabetic patient in the ophthalmic setting. It is also important to realise the overall impact of diabetes on the eye. Although this article concentrates on the retina, diabetics can suffer a myriad of signs and symptoms: cataracts, cranial nerve palsies, pupillary abnormalities, neovascular glaucoma; and are more susceptible to vein and artery occlusions and ocular ischaemic syndromes. The treatment of

such conditions should ideally be based on current scientific evidence and clinical trials showing the effectiveness of the treatment provided. However, it is important to consider each individual patient and their specific needs as well as taking into account the experience of the practitioner administering the treatment needed.

Diabetic retinopathy

Ideally, management of diabetes and diabetic retinopathy should be based on evidence gained from research studies conducted in this field. This would include randomised controlled trials, case series, case studies and other forms of non-randomised prospective or retrospective patient data analysis. The key studies in the management of diabetic retinopathy which set some of the standards of current practice are:

1. The United Kingdom Prospective Diabetes Study (UKPDS)
2. The Diabetic Retinopathy Study (DRS)
3. The Early Treatment of Diabetic Retinopathy Study (ETDRS)
4. The Maculopathy Photocoagulation Study (MPS)
5. The Diabetic Retinopathy Vitrectomy Study (DRVS).

As stated in previous articles in the series, diabetes is generally classified as Type 1 or Type 2. Type 1 refers to patients with reduced insulin

production who require insulin replacement. These patients tend to be younger. Type 2 diabetics develop insulin resistance and can be managed by diet, drugs and/or insulin replacement. Although Type 2 diabetes tends to be a disease of the older population, we are now seeing a younger population diagnosed with this condition due to the increase in childhood obesity. There are other subtypes of diabetes such as people who have suffered damage to the pancreatic cells that produce insulin, for example in chronic pancreatitis. Women can develop diabetes during pregnancy and this is referred to as gestational diabetes.

The UKPDS has shown that close monitoring of blood glucose and tight diabetic control, reduces the rate of developing diabetic retinopathy both in Type 1 and Type 2 diabetics. For this reason, management of diabetic retinopathy requires a multidisciplinary approach involving physicians,

ophthalmologists, optometrists, pharmacists, nursing and non-medical specialist staff.

Diabetic retinopathy occurs largely due to ischaemic changes in the retina. Retinal ischaemia is thought to be caused by factors such as altered platelet function and blood viscosity. Increased intravascular osmotic pressure due to high glucose levels also contributes to intracellular electrolyte imbalances and damage to the retinal vascular cells. It is damage to the cells of the retinal vessels and their supporting cellular structures (pericytes) that lead to the signs we see in diabetic retinopathy.

Vascular endothelial growth factors (VEGF) are produced by retinal cells in an ischaemic environment. This leads to development of retinal vascular abnormalities and new vessel formation.

The ocular manifestations of diabetes are summarised in Table 1. These signs are listed in order of the severity of ischaemia and stages of diabetic

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retinopathy. In mild retinopathy, one may see only microaneurysms and as the severity of the retinopathy progresses, haemorrhages, cotton-wool spots and intra-retinal microvascular abnormalities (IRMA) develop. Unchecked ischaemia ultimately leads to proliferative changes with new blood vessel formation (Figure 1) and risk of vitreous haemorrhage (Figure 2).

When examining the retina, systematically examine the macula, retinal vasculature and the four quadrants of the retinal vascular arcade for signs of diabetic retinopathy. The optic nerve should also be carefully examined for proliferative changes.

Look for changes in the macular area to determine if these changes are 'clinically significant' (Figure 3). It is important to realise that potentially sight threatening diabetic maculopathy can exist in the absence of severe diabetic retinopathy. Then examine the rest of the retina and attempt to grade the severity of the diabetic retinopathy. Severe changes need close monitoring by an ophthalmologist and involvement of a multidisciplinary diabetic team. Proliferative changes with 'high risk' characteristics will require treatment.

The definition of clinically significant macular oedema (CSMO) is defined as one or more of the following:

- Retinal thickening at or within 500µm of the centre of the macula
- Hard exudates at or within 500µm of the centre of the macula associated with retinal thickening
- An area of retinal thickening one disc area in size, at least one part of which



➔ **Figure 1**
Proliferative diabetic retinopathy

Name	Description
Microaneurysms	Seen as small red dots in the middle retinal layers. Weakening of the capillary wall causes dilatation and aneurismal changes.
Exudates	This is a collection of serum and breakdown products of neurones. They are seen as discrete yellow dots often surrounding areas of retinal thickening where fluid has leaked (e.g. from microaneurysms)
Haemorrhages	Rupture of weakened capillaries leads to haemorrhages. Deep haemorrhages (inner nuclear layer/outer plexiform layer) are round/oval and are often called 'dot-blot haemorrhages'. Superficial haemorrhage (nerve fibre layer) is flame shaped.
Cotton wool spots	Local ischaemia causes swelling of the retinal nerve fibres due to obstructed axoplasmic flow. These are seen as white, fluffy spots.
Venous beading and venous loops	Sign of slow retinal circulation. Important indicator of ischaemia and areas of capillary non-perfusion.
Intra retinal microvascular abnormalities (IRMA)	Dilated capillaries representing collaterals that open up between the arterial and venous circulation.
Neovascularisation on the disc (NVD)	New vessels (NV) on or within one disc diameter of the optic nerve. NV appear as fine fronds that either lie flat on the surface of the disc or protrude into the vitreous gel. They tend to arise from retinal veins.
Neovascularisation elsewhere (NVE)	New vessels more than one disc diameter from the optic nerve. Fine vessels can lie flat on the retina or protrude into the vitreous gel.
Vitreous haemorrhage	Bleeding from NVE or NVD. May be subhyaloid (between the vitreous face and the retina) (figure 2) or intravitreal.
Fibrovascular change	Proliferating NVE and NVD can develop fibrous changes. They appear as white bands that follow the NV.
Tractional retinal detachment	Posterior vitreous detachment and shrinkage can cause traction on NV and leads initially to vitreous haemorrhage and later to areas of tractional retinal detachment.

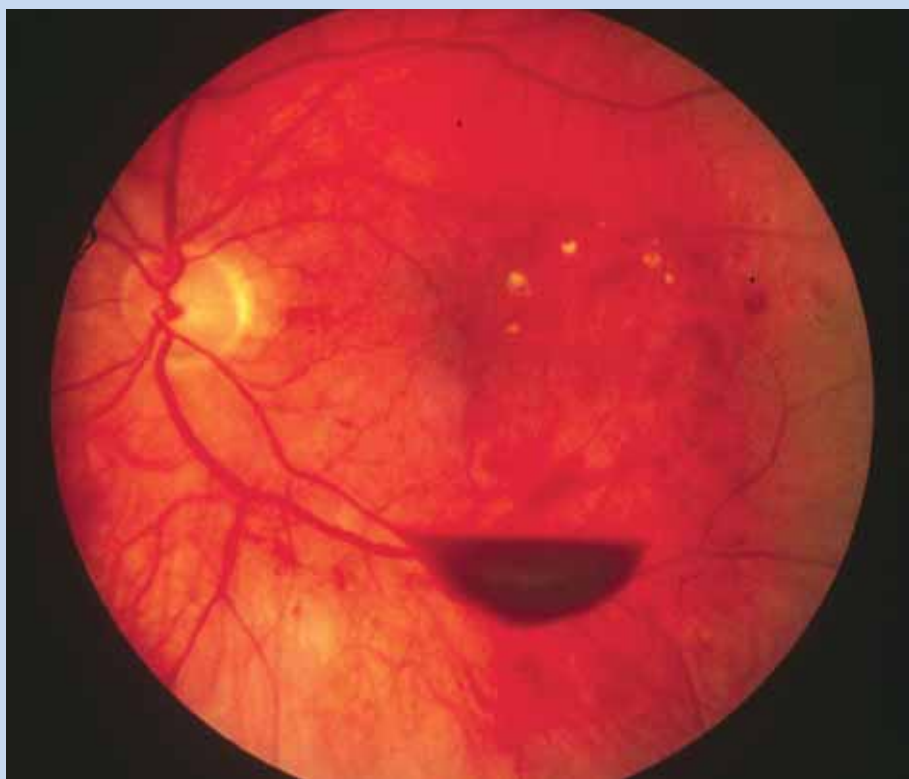
➔ **Table 1**
Ocular manifestations of diabetes

is within one disc diameter of the centre of the macula.

CSMO should be referred promptly to an ophthalmologist to consider argon laser photocoagulation. The clinician may or may not perform a fundus fluorescein angiogram (FFA)

prior to treatment.

The overall retina is evaluated, looking at signs to establish the degree of ischaemia and if 'high risk characteristics' are present. Non-proliferative diabetic retinopathy (NPDR) (Figure 4) is now classified as



➔ **Figure 2**

Proliferative diabetic retinopathy with subhyaloid haemorrhage

mild, moderate and severe based on standard photographs from the ETDRS (Table 2). These are available to view at: <http://eyephoto.opth.wisc.edu/ResearchAreas/Diabetes/DiabStds.htm>

Once neovascularisation occurs, this is called proliferative diabetic retinopathy (PDR). The ETDRS recommendation is to treat PDR with 'high risk' characteristics, defined below, although each clinician should treat each patient as an individual, using the recommendations as a guideline. The presence of these risk factors carries an increased risk of visual loss within two years:

1. NVD over $\frac{1}{3}$ or more of the optic disc surface
2. NVE more than $\frac{1}{2}$ of an optic disc area
3. Vitreous or preretinal haemorrhages (Figure 5)

Management of the diabetic patient requires a holistic approach. It is not uncommon to have patients present late in the disease process and with already advanced diabetic retinopathy. As mentioned previously, good

diabetic blood sugar control is essential to control the progress of diabetic retinopathy. It is also important to realise that these patients can have other vascular pathology affecting their cardiovascular and renal systems. Many also suffer other systemic pathology and managing hypertension, hypercholesterolaemia and risk factors such as smoking are important adjuncts in managing retinal disease.

Finally, initial progression of diabetic retinopathy is seen when tight control of blood sugar is instituted in a poorly controlled diabetic. This does recover but may require close monitoring. Rapid deterioration of retinopathy is also recognised in pregnancy and should be monitored closely and treated as necessary.

Diabetic screening

The National Screening Committee has established a population screening programme in England and Wales to detect diabetic retinopathy that is severe enough to warrant review and possible treatment by an

ophthalmologist. The aim of the programme is to reduce the risk of sight loss amongst people with diabetes, by the prompt identification and effective treatment of sight threatening diabetic retinopathy. All diabetics from the age of 12 years are offered annual photographic screening. Systematic screening involves digital photography of the retina, followed by a two-stage or three-stage image grading process to identify sight-threatening diabetic retinopathy in the retina. The pupils are routinely dilated to obtain an optimal image during screening in England and Wales. In Scotland, the pupil is only dilated when unable to obtain an optimal image.

The photographic screening is provided by ambulatory units that visit the general practitioner or photography in optometry practices. A group of trained professionals then grade the digital retinal photographs and the information is sent both to the patient's GP and the co-ordinators of the National Screening Programme. If diabetic retinopathy that requires closer monitoring (e.g. a pregnant woman with gestational diabetes) or changes requiring treatment are detected (e.g. a diabetic with the early signs of CSMO), the patient is sent an appointment to attend their local ophthalmology unit. An adult who has been treated for proliferative diabetic retinopathy would continue being screened following treatment.

We have previously described the grading of diabetic retinopathy as mild, moderate and severe NPDR and PDR. For screening purposes this has been modified to 'low risk' which includes mild/moderate NPDR (background diabetic retinopathy) and 'high risk' which includes severe NPDR (preproliferative). These have been assigned different levels as described in Table 3.

To reiterate, 'low risk' characteristics are: microaneurysms, mildly dilated veins, dot haemorrhages, exudates and a few cotton wool spots. 'High risk' characteristics are: IRMA, venous beading with venous loops, clusters of large haemorrhages and multiple cotton wool spots.



Maculopathy is classified as focal oedema, diffuse oedema, ischaemic or mixed. Focal maculopathy refers to well defined areas of oedema often surrounded by exudates. These may or may not be 'clinically significant' as described before. Diffuse maculopathy refers to general thickening of the central macula. In some patients, the visual acuity (VA) is reduced despite the lack of evident oedema. This is due to ischaemic changes affecting the macula. Fluorescein angiography can be useful to identify this and will show enlargement of the foveal avascular zone (FAZ). Mixed maculopathy is when oedema and ischaemia co-exist.

Diagnosis

Fundus fluorescein angiography

FFA is useful to determine the severity of diabetic retinopathy. The patient is counselled and consented. An inert fluorescein dye is then injected into the peripheral venous circulation after the patient is suitably positioned at a fundus camera poised to photograph the transition of dye through the retinal circulation. The dye travels first through the venous circulation, through the capillaries then the arterial circulation. Dye leaks through weaknesses in the vascular walls and areas of non-perfusion are not highlighted by the circulating dye. A rapid sequence of photographs of one or both eyes is taken. This sequence of photographs enables the ophthalmologist to determine areas of fluid leakage, for example, in macular oedema and shows areas of capillary non-perfusion (ischaemic areas) in the retina. It can also help differentiate between IRMA and new vessels (NV), as the latter leak fluorescein dye.

A FFA is not always necessary before proceeding to treat diabetic retinopathy. It is usually performed when the clinical picture is not straightforward or a patient has undergone previous treatment that has been unsuccessful.

Optical coherence tomography

Optical coherence tomography (OCT) is a transpupillary method of imaging the layers of the retina. It is non-invasive and produces a cross sectional

image of the retina with a resolution of 10 to 17µm that allows differentiation of the retinal layers. It also allows measurement of retinal thickness. In the diabetic patient, OCT can be used to identify macular oedema and vitreous traction on the macular region. It can also be used to monitor the progress of macular oedema following treatment.

Treatment

Laser

Treatment of diabetic retinopathy and maculopathy is performed using ophthalmic lasers. There are many forms of lasers currently in use and new ones are being developed for the purpose of treating eye disease.

Laser treatment is applied to the retina through a dilated pupil either at the slit-lamp using a contact lens, with an indirect ophthalmoscope or through the sclera. The mainstay of treatment is done at the slit lamp using various forms of contact lenses. These lenses allow application of the laser to the peripheral retina, the retina surrounding the arcades and also provide a magnified view of the macula for detailed laser application. When

using these lenses, it is important to remember that the image is inverted and that the lens in use can magnify the applied spot of laser, so the necessary adjustments need to be made. The laser beam is usually focused onto the area to be treated using a red HeliumNeon (HeNe) laser which acts as a pointer. In some situations, laser photocoagulation can be applied using a non-contact condensing lens such as a 90 or 78 dioptre lens.

Indirect ophthalmoscopy is used to apply laser photocoagulation to the peripheral retina usually in the setting of general anaesthesia. There are some patients, however, who undergo this procedure under local anaesthesia. A diode laser can be used to apply photocoagulation using a trans-scleral route.

The pigment in the retinal tissue (lutein, melanin or haemoglobin) absorbs the energy emitted by the laser and this causes thermal damage to the retinal cells. The target of photocoagulation when treating diabetic retinopathy is the retinal pigment epithelium. The most common laser used in ophthalmic practice is the argon laser. This is a gas



➔ **Figure 3**

Severe non-proliferative diabetic retinopathy with clinically significant macular oedema



laser that produces a beam in the visible light spectrum. The laser produces two main peaks of energy in the 514nm and 488nm wavelength. The latter is filtered out because it is absorbed by luteal pigment and so can potentially damage the macula.

Other lasers includes the Krypton laser, Dye laser and the Diode laser (used with a contact lens). There is some evidence to show that the frequency doubled YAG laser can be used to treat diabetic maculopathy. When using ophthalmic lasers any attendants in the room should wear protective goggles. The room should have windows closed and darkened and reflective surfaces such as mirrors removed.

In pan-retinal photocoagulation (PRP), the aim of treatment is to destroy areas of retina that show ischaemia or reduced capillary perfusion. Application of the laser to the retina causes blanching of the retinal tissue. If the laser energy used is too high, the retina will whiten significantly. The aim is to cause mild to moderate blanching of the retinal tissue. It is also important to remember that the scar caused by laser photocoagulation will enlarge in time and that the spots of laser application should be adequately spaced (one spot size width apart). The

usual application is a burn spot size of 500µm diameter.

The aim of PRP is to treat proliferative diabetic retinopathy with 'high risk' characteristics. Treatment is applied to the entire retina, avoiding the macular area within the temporal arcades. This should ideally be done using 1500 to 2000 burns with a spot size of 500µm diameter. The treatment is done under local anaesthesia and can be done in a single sitting. If the treatment is not well tolerated, the patient is brought back within a week to complete the treatment. The patient is then reviewed in one to three month's time to assess if regression of NV has occurred. If treatment has not been entirely successful, further laser can be applied.

Shorter-pulsed patterned scanning laser photocoagulation using the new PASCAL laser (Optimedica Corp, Santa Clara, California) allows application of multiple burns at a time. This increases the precision of application and reduces the overall time needed for PRP. This procedure also shortens the time each laser burn is applied from 0.1 seconds (s) down to 0.01s, allowing for a more comfortable experience and less overall laser exposure.

There are recognised complications

of PRP. These are:

• **Reduction in visual function**

The visual field can be reduced by up to 50% following PRP causing a loss of peripheral vision. Patients should be counselled prior to treatment that this may affect their ability to drive in future. The aim of treatment is to preserve vision and not to necessarily improve it. It is the responsibility of the patient to inform the UK Driver and Vehicle Licensing Agency (DVLA) that they have undergone retinal laser treatment. Colour vision can also be affected.

• **Vitreous haemorrhage can occur following PRP**

Some patients have NV extending from the retina into the vitreous. Photocoagulation can cause separation of the vitreous from the retina (posterior vitreous detachment) causing traction on these NV with subsequent vitreous haemorrhage.

• **Pain**

Photocoagulation can be a painful experience for some patients. Some can tolerate treatment with topical anaesthesia only. Others may require a subtenon anaesthesia. In extreme cases, a general anaesthetic may be required.

• **Macular oedema**

In some patients, PRP can precipitate macular oedema. This often resolves spontaneously but patients should be warned about this.

• **Secondary choroidal neovascularisation**

This is a condition similar to that seen in wet age related macular degeneration. Choroidal neovascularisation can occur if very high laser energy is applied very close to the macula.

Before any treatment is given, the patient needs to be informed of the risks and benefits and written informed consent should be obtained.

The primary treatment of diabetic maculopathy involves careful application of laser energy to the macular region. Again, this is usually done with an argon laser and is applied either to focal lesions or as a grid to diffuse oedema. The infra-red diode laser is also useful in treating macular oedema with a wavelength of

Diabetic Retinopathy (DR) Severity Level	Findings Observable with Dilated Ophthalmoscopy
No apparent DR	No abnormalities
Mild nonproliferative DR	Microaneurysms only
Moderate nonproliferative DR	More than "mild" but less than "severe"
Severe nonproliferative DR	Any of the following: 20 or more intraretinal haemorrhages in 4 quadrants Definite venous beading in 2 or more quadrants Prominent IRMA in 1 or more quadrants and no neovascularisation
Proliferative DR	1 or more of the following: Definite neovascularisation Preretinal or vitreous haemorrhage

➔ **Table 2**

International Clinical Diabetic Retinopathy (DR) Disease Severity Scale



Disease Grade	Disease Severity	Grade Level	Management
Retinopathy (R)	None Background Pre-proliferative Proliferative	Level 0 (R0) Level 1 (R1) Level 2 (R2) Level 3 (R3)	Annual screening Annual screening Refer to hospital Refer to hospital urgently
Maculopathy (M)	None Present	Level 0 (M0) Level 1 (M1)	None Refer to hospital
Photocoagulation (P)	None Present	Level 0 (P0) Level 1 (P1)	None If new patient, refer to hospital If stable post treatment, annual screening
Other Lesions (OL)	N/A	N/A	Refer to primary care or hospital
Unclassifiable (U)	N/A	N/A	If biomicroscopy possible then refer to hospital. If unscreenable, discharge and inform general practitioner

➔ **Table 3**

Disease grading and management protocol from the National Guidelines on Screening

810nm. Laser treatment is indicated with CSMO when the VA measures Snellen 6/9 or below. If the VA is better, the patient should be counselled and the risks need to be balanced against any potential benefits.

If specific lesions that are causing fluid leakage are identified, either clinically or using fluorescein angiography, these areas are targeted using a scatter of laser burns. These are often microaneurysms. The laser spot size is usually 50-100µm and the laser energy used should cause the faintest blanching of the retina. The laser is usually applied for a duration of 0.1s, or less. These are guidelines only, and different operators will modify the settings according to their level of experience and the individual patient.

If diffuse oedema is present at the macula, the laser is applied in a grid pattern. This form of oedema is more difficult to treat. The argon laser is applied in a grid pattern, with a spot size of 50-100µm, at least one burn width apart, to cause a faint blanching of the retina. The fovea is avoided at all costs!

When ischaemia is present at the macula, VA is reduced despite a

relatively 'dry' appearance. Fluorescein angiography shows enlargement of the FAZ and areas of capillary dropout. This form of maculopathy does not respond to laser treatment. However, if it is felt that the ischaemia is severe enough to be driving proliferative changes in the retina, laser treatment is indicated.

Following laser treatment for diabetic maculopathy, the patient should be followed up after three months to monitor their progress.



➔ **Figure 4**

Severe non-proliferative diabetic retinopathy

When proliferative neovascularisation co-exists with maculopathy, there is some debate about which to treat first. In young Type 1 diabetics with active proliferative changes, it is recommended that PRP is given first followed by treatment of the maculopathy. In older, Type 2 diabetics, the maculopathy should be treated first. This is because PRP has been shown to cause progression of the maculopathy in these patients. PRP can be applied in a few sessions to reduce the risk of developing maculopathy. These, however, are merely guidelines and each patient should be considered individually.

Once treated, proliferative changes may regress completely or may stabilise leaving some NV that do not progress. Signs of ischaemia such as venous beading and haemorrhages resolve with time. There are however, patients who do not respond to PRP and other causes of their ischaemic drive need to be addressed as well.

Surgery

Surgical intervention, in the form of pars plana vitrectomy, has proved



➔ **Figure 5**
Neovascularisation at the optic disc with vitreous haemorrhage

useful in the management of diabetic retinopathy. This needs to be performed by a trained vitreoretinal surgeon. Vitreous haemorrhage can occur in a diabetic eye when NV grow into the vitreous gel that subsequently separates from the retina. This produces traction on the vessels and subsequent bleeding.

If vitreous haemorrhage fails to clear spontaneously in a previously untreated eye, a vitrectomy is indicated to clear the view for both the patient and the ophthalmologist and enable retinal laser to be administered where appropriate. The vitreous gel is cleared of haemorrhage and any source of haemorrhage is identified and dealt with.

Surgery is also indicated if recurrent haemorrhage fails to clear spontaneously after a period of observation in a previously treated eye. The haemorrhage usually clears within a month. If the patient only has one good eye that develops a vitreous haemorrhage or bilateral vitreous haemorrhage occurs, a vitrectomy may need to be performed to enable the patient to carry on with

activities of daily living.

When tractional retinal detachment threatens the macular area, vitrectomy can be used to release the traction from the vitreous. This happens when NV undergo fibrous change and contract causing traction on the retina (Figure 6). Delamination and segmentation of the overlying fibrous tissue can also be beneficial in releasing any horizontal tractional elements.

Some diabetic patients develop chronic macular oedema secondary to traction and/or thickening of the overlying vitreous face. Surgical removal of this posterior vitreous face can be beneficial in relieving the macular oedema. This is done as a pars plana vitrectomy, with or without, a membrane peel.

In patients requiring a vitrectomy, there is often co-existing lens opacification. In some cases, a combined vitrectomy, with or without, lensectomy is indicated.

Following cataract surgery, diabetic eyes are known to develop posterior capsule opacification (PCO) more rapidly than the non-diabetic

population. This is especially the case in younger diabetics. PCO can be treated by YAG laser capsulotomy.

Cranial nerve palsies and pupillary abnormalities

Part of the spectrum of diabetic complications is neuropathy. This can affect one or a number of nerves anywhere in the human body. Nerves are supplied by a network of fine blood vessels called vasso nervorum, which may be damaged by diabetes. Diabetes also increases the risk of suffering a cerebrovascular accident either in the form of a stroke, leading to disability that lasts more than 24 hours, or a transient ischaemic attack that resolves within 24 hours. This can affect both the visual field and/or ocular motility.

If a diabetic patient presents with a recent onset visual field defect that is not explained by retinal pathology, they should be referred urgently to either a physician or an ophthalmologist who can involve the necessary medical team.

A patient may present with a cranial nerve palsy affecting ocular movement leading to diplopia. These patients also require urgent referral to an ophthalmologist to rule out other causes of cranial nerve palsies such as a space occupying lesion or an aneurysm. A headache and dilated pupil are highly suggestive of a compressive lesion. If it is a cranial nerve palsy due to diabetes affecting the microvascular circulation to the nerve concerned, the patient needs orthoptic follow up and monitoring. Most recover spontaneously after three to six months and just need orthoptic support during this time. Temporary spectacle prisms are helpful in dealing with diplopia. If the affected extraocular muscle does not recover, the patient may eventually require permanent prisms or strabismus surgery to provide some improvement to their quality of life.

Some diabetic patients can develop pupillary abnormalities such as an Argyll-Robertson pupil. This is where the pupil accommodates to a near target but not to light.



Cataracts

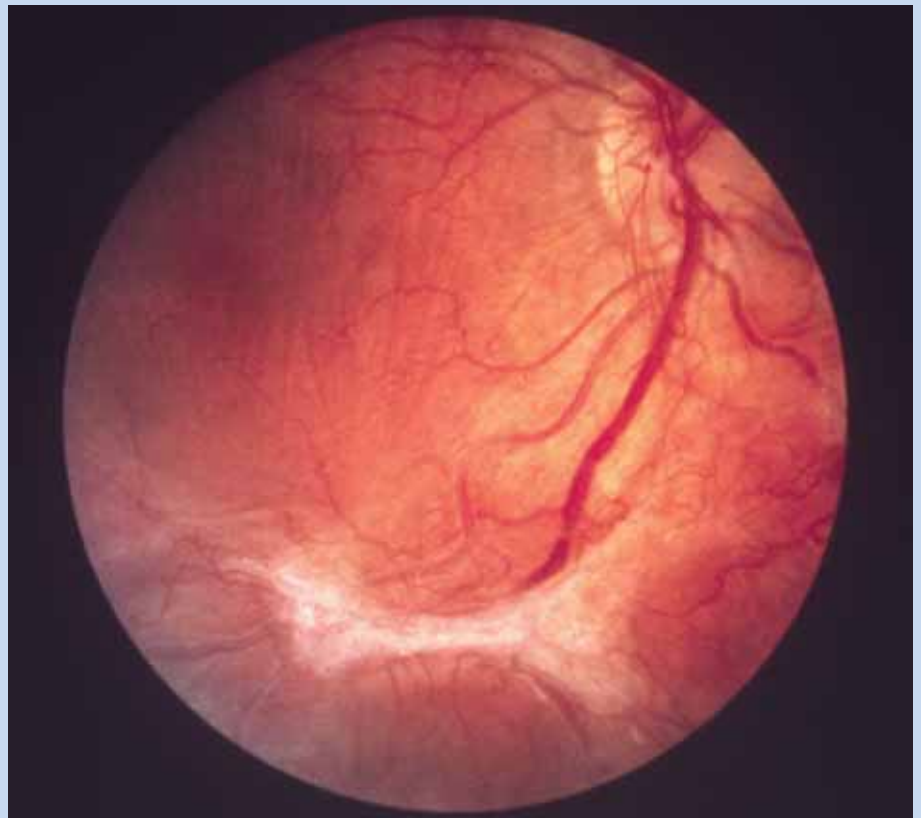
Cataract refers to opacification of the crystalline lens of the eye. The vast majority of cataracts seen in ophthalmic practice are in the elderly population. Opacification of the lens is both more prevalent and occurs more rapidly in the diabetic patient population. It is not uncommon to see cataracts in young diabetics with suboptimal control of their blood sugar levels.

If a young patient presents to you with a cataract and is not known to be diabetic, it is important to refer them to their GP/ophthalmologist to have their diabetic status checked.

The implications of early onset cataract in a diabetic who is still in employment and with an active lifestyle may be significant. Even with a minimal reduction in VA, the patient may experience symptoms including glare or anisometropia from uniocular index myopia which can affect driving or the ability to perform certain fine motor tasks at work. Young diabetic patients with cataracts should be considered for early referral, as it is not uncommon for these cataracts to progress quite rapidly.

Cataract surgery should also be considered if the cataract obscures the view of the fundus and prevents optimal retinal examination. In a patient who presents with moderate to severe NPDR and poor diabetic control, good fundal examination is important. Cataract surgery is indicated if this is not possible due to lens opacification. Lensectomy now is routinely done using phacoemulsification with intra-ocular lens implantation.

It is important to remember the specific risks of cataract surgery to the diabetic patient. If possible, diabetic maculopathy and/or retinopathy need to be addressed before proceeding with cataract surgery. Diabetic patients who undergo cataract surgery, are more at risk of progression of their diabetic retinopathy and of developing post-operative cystoid macular oedema. They should be followed up by an ophthalmologist post-operatively due to both the risk of cystoid macular oedema and the



➔ **Figure 6**

Proliferative diabetic retinopathy with fibrovascular proliferation

possible progression of pre-existing diabetic retinopathy. Diabetic patients may be at greater risk of post-operative infections due to their slightly more immunocompromised state.

If there was no view of the diabetic fundus prior to cataract surgery, the retina should be examined post-operatively following lensectomy. If sight threatening disease is found, there is an argument for treating the patient in theatre with laser photocoagulation. If this is not possible, it should be done at the earliest opportunity.

Once the patient is treated, they are often followed up in hospital for a period of time to ensure the retinopathy has regressed or stabilised. They can then be discharged back to the screening programme. The patient should then have annual photographic screening and also at least an annual review with their optometrist for general ophthalmic and refractive follow up.

Conclusion

Diabetes is a difficult condition to treat and manage. A diabetic has to live with this condition every day of his or her life. Though many cope well with these demands, many find it intrusive and challenging. All too often, these are the patients who have suboptimal diabetic control and develop complications of the disease: cardiovascular, neurological, renal and ophthalmic. By the time they reach the ophthalmologist for treatment, many have other sequelae and need more than just ophthalmic input. Merely concentrating on the eye disease ignores the true drive of the condition and the multidisciplinary approach is imperative. We need to always consider treating the patient, not just the eye.

About the author

Dr Brinda Muthusamy MRCP MRCOphth is currently a registrar in ophthalmology at the Bristol Eye Hospital.



Module questions

Course code: c-10559/0

Please note, there is only one correct answer. Enter online or by the form provided

An answer return form is included in this issue. It should be completed and returned to CET initiatives (c-10559/0) 07, Ten Alps plc, 9 Savoy Street, London WC2E 7HR by April 6 2009

1) Which one of the following is incorrect?

- a. exudates are a collection of serum and breakdown products of neurones
- b. cotton wool spots are due to obstructed axoplasmic flow
- c. microaneurysms are due to high pressure blood flow
- d. intra retinal microvascular abnormalities are dilated capillaries representing collaterals that open up between the arterial and venous circulation

2) Which one of the following is incorrect regarding deep haemorrhages?

- a. they are found in the inner nuclear layer
- b. they are found in the outer plexiform layer
- c. they are called 'dot and blot' haemorrhages
- d. they are found in the nerve fibre layer

3) Which one of the following is correct?

Venous beading and venous loops are signs of:

- a. slow circulation
- b. ruptured capillaries
- c. dilated capillaries
- d. fast circulation

4) Which one of the following is correct?

Clinically significant macular oedema is classified as:

- a. retinal thickening at or within 500µm of the centre of the optic nerve
- b. hard exudates at or within 500µm of the centre of the macula associated with retinal thickening
- c. always due to ischaemia of the macula with an enlarged FAZ
- d. an area of retinal thickening one disc area in size, at least one part of which is within one disc diameter of the centre of the optic nerve

5) All of the following increase the risk of visual loss within two years except:

- a. NVD over 1/3 or more of the optic disc surface
- b. microaneurysms
- c. NVE more than ½ of an optic disc area
- d. vitreous or preretinal haemorrhages

6) Which one of the following people are eligible for community diabetic screening?

- a. a diabetic with the early signs of clinically significant macular oedema
- b. a pregnant woman with gestational diabetes
- c. a child aged eight years old with Type 1 diabetes
- d. an adult diabetic with treated proliferative diabetic retinopathy

7) Which one is correct? If a patient is classified as R3, they require:

- a. annual screening
- b. referral to hospital
- c. referral to hospital urgently
- d. discharge

8) All of the following are recognised complications of panretinal laser photocoagulation except:

- a. loss of peripheral vision
- b. optic nerve neovascularisation
- c. macular oedema
- d. vitreous haemorrhage

9) Which one is incorrect? If diffuse oedema is present at the macula:

- a. the laser is applied in a grid pattern
- b. this form of oedema is difficult to treat
- c. a spot size of 50-100µm is used
- d. the laser is applied at least ½ a burn width apart

10) All of the following are features of diabetic retinopathy that require follow-up by an ophthalmologist except:

- a. vitreous haemorrhage
- b. hard exudates within 500µm of the macula associated with retinal thickening
- c. new vessels at the disc
- d. multiple laser scars in the peripheral retina

11) A 65 year old Type 2 diabetic presents with sudden onset diplopia and headache. His right eye is deviated laterally and inferiorly with a dilated pupil. There is no obvious ptosis. You:

- a. advise him to alter his head posture to minimise the diplopia
- b. prescribe prisms in his spectacles to reduce the degree of diplopia
- c. refer him to his GP advising to refer him to the hospital eye service
- d. refer him urgently to the hospital eye service

12) A 55 year old Type 2 diabetic is reviewed by an ophthalmologist. Fundoscopy shows an area of neovascularisation in the supratemporal arcade measuring two disc areas. The next course of action is:

- a. follow up is arranged for one month's time to monitor progress
- b. to book annual screening
- c. the patient is booked for urgent pan-retinal photocoagulation
- d. urgent referral is made to the diabetic physician before treating the eye

Please complete online by midnight on April 6 2009 - You will be unable to submit exams after this date - answers to the module will be published in our April 10 2009 issue

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