Diabetes in Optometry Practice

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Prevalence

1% of the 60 million population in the UK suffers from Diabetes. The prevalence increases with age to 3 to 4% of the population over the age of 40 years.

Norway, with a population of 4.5 million inhabitants has approximately 130,000 people with diabetes. This amounts to some 2.9% of the population. About 0.45% have IDDM and 1.8% suffer from NIDDM. It is estimated that a further 0.65% of the population are undiagnosed.

Incidence of blindness

In the UK there are 1500 new cases of blindness per year.

Non-Insulin Diabetes Mellitus (NIDDM)

These patients can survive without exogenous insulin although many eventually require insulin to improve control. The prevalence in the UK is approximately 1% in UK and in Norway 1.8%.

The prevalence in elderly and some groups in the UK, for example Asian communities are higher at around approximately 3 to 5%.

The prevalence in Lima Indians of Arizona is reportedly 50%.

Migrants to western world seem to be particularly susceptible.

NIDDM is correlated with a combination of relative insulin deficiency and insulin resistance. The latter may be exacerbated by obesity.

There is a strong genetic predisposition with twin concordance for NIDDM being 90% compared to only 40% for IDDM.

NIDDM accounts for about 75 to 80% of the diabetic population.

NIDDM gives high risk of macrovascular complications and usually presents as a syndrome of anomalies including hypertension, hyperlipidaemia, obesity, and insulin resistance.

Very often optometrists will hear from their patient that “I have a just a little sugar” or “I have mild diabetes”. In fact, there is no such thing as “mild diabetes”.

Whatever the control required, if not adhered to, the disease will lead to retinal and other damage.

**Insulin-Dependent Diabetes Mellitus (IDDM)**

These patients cannot survive without insulin. In the UK the prevalence is about 0.2% with an incidence of 15/100,000/year aged < 21. Most cases present before 30 years and there is a peak incidence at 11-13 years.

A possible cause is destruction of the $\beta$ cells of the Islets of Langerhans perhaps through an autoimmune response.

Retinopathy is unusual before puberty and usually only presents after at least 10 years.

Mortality in < 50 age group is about 5 x higher than non-diabetics.

**Ocular manifestations of diabetes**

**Introduction**

Although we often think of retinal changes, it must not be forgotten that other parts of the eye and visual system may be affected including the anterior segment with changes occurring to the iris and lens. Changes in the iris include micro aneurysms and neovascularisation, the latter leading to a secondary glaucoma.

The classic “diabetic snowflake cataract” that occurs in acute diabetes is rarely seen. If the patient is swiftly treated, the cataract substantially resolves. Diabetics tend to develop prematurely age related lens changes. The optometrist is often the first person to suspect diabetes in a patient because of apparent fluctuations in refraction, along with reported symptoms of an unusual thirst and possible a general feeling of malaise or repeated illnesses.

The visual pathway may be affected in a variety of ways with, for example, haemorrhages producing visual field defects. Similarly diabetics are more prone to suffering lesions of the ocular motor system with consequent gaze palsies and incomitant strabismus.

**Diabetic retinopathy**

The term background retinopathy is used to describe the clinical features first seen within the diabetic retina and therefore the appearance that most
optometrists are probably most familiar. It is this type of diabetic retinopathy that optometrists in general practice need to make clinical judgments about.

Over the years there have been a number of classification systems proposed for describing diabetic retinopathy severity.

The classification of diabetic retinopathy as outlined by the Early Treatment of Diabetic Retinopathy (ETDRS) is now widely accepted.

**The ETDRS grading system can be summarised as follows:**

<table>
<thead>
<tr>
<th>Mild Non proliferative diabetic retinopathy – NPDR</th>
<th>At least one microaneurysm, but not as severe as moderate NPDR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate NPDR</td>
<td>Extensive intra-retinal haemorrhages, and/or microaneurysms, and/or cotton wool spots, venous beading or intra-retinal macrovascular abnormalities (IRMA) definitely present, but not as severe as severe NPDR</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>Cotton wool spots, venous beading, and IRMA, all present in at least two quadrants; two of them present in at least two quadrants with intra-retinal haemorrhages and microaneurysms present in all four quadrants; or IRMA present in each quadrant, being severe in at least one of them and no PDR.</td>
</tr>
</tbody>
</table>

This can be simplified to:

1. Intra-retinal haemorrhages in four quadrants
2. Venous beading in two quadrants
3. Severe IRMA in one quadrant

<table>
<thead>
<tr>
<th>Proliferative retinopathy - PDR</th>
<th>Neovascularisation of the disc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early PDR</td>
<td>Neovascularisation of the retina</td>
</tr>
<tr>
<td>PDR with high risk criteria</td>
<td>Pre-retinal haemorrhage</td>
</tr>
<tr>
<td>PDR including advanced diabetic eye disease</td>
<td>Vitreous haemorrhage</td>
</tr>
<tr>
<td></td>
<td>Tractional retinal detachment</td>
</tr>
<tr>
<td></td>
<td>Neovascularisation of the iris/angle</td>
</tr>
</tbody>
</table>

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**Pre-clinical retinopathy and histopathology**

One of the earliest reported features of diabetic pre-retinopathy has been the presence of dilated retinal veins. This is extremely difficult to assess and its importance in clinical management is somewhat dubious.
The main pathophysiological processes to occur prior to fundus changes seen clinically are:

**Retinal vascular leakage and occlusion**

This is characterised by alterations to the capillary wall and a reduction in blood flow and oxygenation. Vascular occlusion is thought to have some role in the formation of microaneurysms.

Microvascular leakage and microvascular occlusion are the two main pathological processes responsible for the development of diabetic retinopathy. The structural abnormalities that develop within the retinal capillary wall include the following:

- Pericyte loss
- Loss of endothelial cells
- Basement membrane thickening
- Endothelial cell dysfunction

All these alterations result in loss of autonomic auto-regulation, regulatory function, leakage from capillaries into the extra-cellular space of the sensory retina. The rheological changes which may result from hyperglycaemia affect the plasma, red blood cells and the platelets. The plasma changes include an increase in fibrinogen, $\alpha_2$-globulins and a decrease in serum albumin levels, resulting in decrease in fibrinolysis and increased viscosity. The alterations within red blood cells are decreased deformability and, within blood platelets, an increased tendency to clump together. The end result of this process is thrombosis within the retinal capillaries leading to the development of areas of capillary non-perfusion. Subsequently these areas may enlarge.

**Increased retinal vascular permeability**

This results in haemorrhages, exudates and retinal oedema.

**Non-proliferative (background) diabetic retinopathy – NPDR**

**Microaneurysms**
The earliest visible changes that develop are microaneurysms usually temporal to the fovea and representing mild NPDR. They can be classified into two distinct types:

- **Saccular** – Sac like extensions presumed to evolve from weak points in the capillary wall and be abetted by intra-luminal pressure. Reduced structural support from pericytes has been suggested as a contributory factor to their formation. An active cellular response, possibly as a result of a reduced inhibitory action by pericytes, has been proposed as a model for microaneurysm formation. In this case there would be an active budding of the cells of the cell wall.

- **Loop** – originate as kinks in a capillary, these appear to be uncommon in non-diabetics in contrast to saccular microaneurysms which are common in many vascular disorders. They are thought to develop from the fusion of contiguous arms of kinked segments of a capillary.

Microaneurysms may occur at any level between the superficial and deeper retinal capillary networks or even from the choroidal circulation, though the inner nuclear layer is the usual location. They vary in size from ~10 to 100µm but only those greater than 30 µm are visible clinically. The ETDRS gives an upper limit of 125µm diameter and the requirement of sharp borders to be considered a microaneurysm.

Larger red spots would be considered to be haemorrhages. Usually microaneurysms appear as bright red spots but occasionally they appear yellowish due to endothelial lining proliferation and hyalinisation of their cavities. They may well be associated with circinate cavities.

**Intra-retinal haemorrhages**

These appear secondarily to ruptured microaneurysms, capillaries and venules. Their appearance and therefore their classification depends on their location within the retinal layers.

- **Flame shaped haemorrhages**

  These occur in the superficial nerve fibre layer where the blood tends to follow the course of the nerve fibres

- **Dot haemorrhages**

  These are located in the outer-plexiform and inner nuclear layers of the retina. On fluorescein angiography dot haemorrhages exhibit hypofluorescence as opposed to hyperfluorescence of microaneurysms.
Blot haemorrhages

These are similar to dot haemorrhages but with less distinct borders.

Some haemorrhages have white centres due to the presence of platelets and fibrin. Intra-retinal haemorrhages are usually found scattered throughout the posterior pole and usually resolve within 4-6 months. Haemorrhages can of course be caused by many other conditions.

Hard exudates

Hard exudates consist of accumulated and condensed plasma and so are made up of mainly serum lipoproteins. They represent a leakage from the circulation at a level which probably requires a degree of structural damage to vascular endothelium. Capillaries with microaneurysms are the principle source.

Fluid plasma leaks through the abnormal permeable vascular wall and seeps into the outer-plexiform layer where it collects. It is thought that the interphotoreceptor Muller cell junctional complexes present an obstacle to the further movement of molecules the size of lipids and proteins, whilst allowing the unimpeded passage of water towards the choroid. In this the exudate becomes progressively concentrated to leave semi-solid residues which acquire a characteristic hard or waxy appearance.

Hard exudates may be re-absorbed either spontaneously or following laser photocoagulation. This is due to phagocytosis by macrophages.

Cotton wool spots

These are essentially a feature of moderate to severe NPDR (pre-proliferative). However less than 5 cotton wool spots are often considered as part of a background retinopathy scenario in screening protocols provided other pre-proliferative changes are not also present.

Cotton wool spots are small infarcts within the nerve fibre layer caused by reduction or blockage of flow within an arteriole. This results in a stasis of axoplasmic flow within nerve fibres and a subsequent swelling of the neural tissue supplied by the arteriole. Clinically this is seen as a poorly defined white spot often marked by striations from the nerve fibre layer.

Retinal oedema

The increased permeability of retinal vessels allows leakage of plasma constituents which accumulate in the extra-cellular spaces, initially at the outer-
plexiform layer and inner nuclear layer level and later extending to involve the entire retinal thickness. Such oedema at the macula is the most common cause of reduced vision in NPDR.

**Diabetic macular oedema**

Diabetic macular oedema according to the ETDRS can be defined as hard exudates and retinal thickening involving the macular area. Although these features lie in the macular area the fovea is not yet involved.

**Clinically significant macular oedema**

Clinically significant macular oedema is defined as any one of the following:

1. Retinal thickening at or within 500µm of the centre of the macula
2. Hard exudates at or within 500µm of the centre of the macula if associated with adjacent retinal thickening.
3. A zone or zones of retinal thickening one disc area in size at least part of which is within one disc diameter of the centre of the fovea.

**Diabetic maculopathy**

Diabetic retinopathy is well recognised as the commonest cause of blindness in the working age group (20-65 years) in the United Kingdom. It is responsible for 12% of all new cases of blindness in America each year. One of the ways in which blindness occurs is when the central macular area of the retina is damaged, causing maculopathy. The frequency of maculopathy varies between type I (juvenile onset) and type II (maturity onset) diabetes.

In mild non-proliferative diabetic retinopathy (NPDR), the earliest visible changes that develop are microaneurysms, usually in the area temporal to the fovea. At this early stage macular oedema with retinal thickening or hard exudate formation is rare but may be a threat to macular function. Such patients require follow up every ~6 months. If macular oedema becomes clinically significant then laser treatment should be undertaken.

In type I diabetes, macular oedema is not found before 5 years duration of the disease. In contrast with type II diabetics, the risk is 3% at less than 5 years duration. In both types the cumulative risk rises to approximately 30% after 20 years duration.

Risk factors for the development of diabetic maculopathy include the duration of diabetes, particularly in type I diabetes, pregnancy, hypertension, chronic hyperglycaemia, renal disease and hyperlipidaemia.
Aetiology of maculopathy

The underlying changes which occur in diabetic maculopathy are the same as in background or NPDR. However they are classified separately due to the special anatomy and function of the macula. The macula is defined as the central area of retina between the superior and inferior temporal arcades, from the disc and 2 disc diameters temporal to the fovea.

Classification of diabetic maculopathy

There are 4 main types of maculopathy according to clinical examination and fluorescein angiography. These are:

- **Focal:** Leakage from dilated segments of capillaries and microaneuerysms
- **Diffuse:** Characterised by the presence of diffuse oedema.
- **Ischaemic:** Capillary shut down results in retinal non-perfusion and ischaemia. It is characterized by the presence of large blot haemorrhages, multiple cotton wool spots and IRMAs.
- **Mixed:** It is not uncommon to see a combination of focal, diffuse and ischaemic maculopathy.

Focal diabetic maculopathy

The features are well defined focal areas of leakage with microaneurysms, haemorrhages and retinal thickening. These areas are often surrounded by circinate hard exudates.

In focal exudative macular oedema, discrete leakage sites are a consistent feature. Leakage may occur from retinal microaneurysms or areas of dilated retinal capillaries. The extent of the vascular changes can vary considerably. Leakage from capillaries can occur from the deep or superficial capillary network in the retina. Leakage from either micro-vascular abnormality gives rise to intra-retinal oedema with a bulk fluid flow towards competent capillaries. At these sites the fluid is reabsorbed into the relatively normal retinal capillary bed, causing the deposition and accumulation of the large molecules such as proteins and lipids which are seen as hard exudates.

The exact configuration of the exudate depends not only on the degree and sites of leakage, but also on the characteristics of fluid movement and absorption. Therefore exudates found at the macula vary considerably.
In many patients with focal exudative macular oedema the areas of leakage are well away from the fovea and central vision is preserved. This is where macular oedema is not clinically significant. Most patients in this group are asymptomatic, although some may suddenly notice fluctuations in their vision or even paracentral scotoma. In patients who do suffer with disturbed central vision, the degree of visual loss is usually related to the extent of retinal oedema and hard exudate formation. Once retinal thickening or hard exudates are coming as close as 500µm to centre of the fovea, focal laser treatment is advised, even in patients with perfect vision.

With direct focal treatment to leaking microaneurysms, the aim of treatment is to stop the leakage from the microvascular abnormalities and to allow hard exudates and fluid to be absorbed. Treatment should be applied to leaking microvascular lesions and to achieve this goal it is often useful to have a fluorescein angiogram available at the time of treatment. A fluorescein angiogram is particularly useful when it is suspected that these leaking lesions themselves are very close to the macula.

The results of laser treatment in this group are extremely good. Laser photocoagulation used in this type of maculopathy is the most straight forward and often fluorescein angiography is not necessary if the area of leakage is clinically obvious and sufficiently far away from the fovea.

The best results are obtained in those eyes with well-defined circinate, hard exudate rings, but minor or no involvement of the fovea and good vision.

The poorest response is seen in eyes in which central vision is reduced as a result of a foveal plaque of exudate or a long standing significant cystoid macular oedema.

Usually patients should be evaluated 3 months after treatment. Any residual oedema should be assessed and any additional leaking points should be considered for more treatment. It is not uncommon for more retinal microaneurysms to be seen at this visit, because they will become visible as the surrounding semi-opaque oedema subsides.
Laser treatment for diabetic maculopathy

The ETDRS demonstrated that the most effective strategy for eyes with macular oedema and mild, non-severe NPDR was immediate focal laser treatment with delayed scatter initiated only when more severe retinopathy developed. This reduced the risk of moderate visual loss by 50% compared to treatment deferral. Eyes in the group assigned to immediate full scatter and delayed focal photocoagulation had an increased risk of moderate visual loss during the first 16 months and were similar thereafter to eyes assigned to treatment deferral. Either strategy therefore was shown to have a decreased incidence of severe visual loss at 5 years compared to the treatment deferred group.

For eyes with macular oedema and severe NPDR or early PDR, the 5 year rate for the combined end point of severe visual loss or vitrectomy was 10.3% for eyes assigned to the treatment deferred group. This compared to 5.6% for those receiving full scatter and 6.9% for those assigned mild scatter photocoagulation.

A major objective of the ETDRS was to evaluate the effectiveness of laser photocoagulation in the management of diabetic macular oedema. When all eyes with diabetic macular oedema were considered (regardless of whether the macular oedema was “clinically significant”), treatment with immediate focal or grid-pattern laser photocoagulation significantly reduced the incidence of moderate visual loss compared to the subgroup in which focal laser photocoagulation for macular oedema was deferred. Immediate photocoagulation reduced the incidence of moderate visual loss by ~ 50% at all time points.

Specifically, the incidence of moderate visual loss was 5% in the immediate photocoagulation subgroup versus 8% in the deferral group at 1 year; 7% versus 16% at 2 years and 12% versus 24% at 3 years.

There is emerging evidence for anti-VEGF therapy as an effective treatment for macular oedema.

Pre-proliferative retinopathy

The term pre-proliferative diabetic retinopathy (PDR) is used to describe a clinically identifiable stage of retinopathy that precedes proliferative retinopathy. It is characterised by significant retinal ischaemia which reflects the underlying condition of retinal capillary closure. It is important to be able to recognise this stage because patients can quickly progress to proliferative retinopathy. Patients are at increased risk of visual loss from development of disc new vessels (NVDs) and/or new vessels elsewhere (NVEs) which may give rise to vitreous haemorrhage.

Signs of pre-proliferative retinopathy include
Blot haemorrhages

These originate from the deep capillary plexus. They are located in the inner plexiform and outer plexiform layers of the retina. Due to the compact structure of the retinal elements in this region and the relative depth at these locations, the haemorrhages assume a dark, blot like appearance. They are easily differentiated from the brighter red, flame shaped retinal haemorrhages. These originate from the superficial capillary plexus and in this less compacted nerve fibre layer they can then produce a flame shape.

Cotton wool spots

These are a common feature in DR and are a consequence of capillary occlusion in the nerve fibre layer and are arranged along its long axis. The resulting ischaemia interrupts the normal energy dependent axoplasmic flow in the nerve fibre layer. There is a build up of transported material in the nerve axons of the retinal ganglion cells. This gives rise to the fluffy white cotton wool-like lesions seen on slit lamp biomicroscopy. Most cotton wool spots have a fairly common size, being less than half a disc diameter. Small superficial, flame shaped haemorrhages are often related to cotton wool spots. On fluorescein angiography in the area of a cotton wool spot, there is capillary non-perfusion and it is sometimes possible to identify the arteriole that has become occluded. In DM this is usually due to capillary closure rather than arteriolar closure however. Cotton wool spots are almost always confined to the area adjacent to the major vascular arcades of the posterior pole.

Venous changes

Tortuosity and dilatation occur when there is sluggish retinal circulation and are the most important signs of pre-proliferative disease. Beading and looping are caused by increasing hypoxia. Focal vitreous traction may contribute to the formation of venous loops. Beading is the most important sign.

Intra-retinal microvascular anomalies (IRMA)

IRMA are defined as dilated retinal capillaries which are often difficult to assess and fluorescein angiography may well be needed. IRMA are a hall-mark of severe non-proliferative diabetic retinopathy and they are a pre-cursor of proliferative retinopathy. Today IRMA and intra-retinal new vessels are considered to be the same. By definition, intra-retinal new vessels are IRMA as long as they are not breaking through the internal limiting membrane.
Proliferative diabetic retinopathy

Proliferative diabetic retinopathy (PDR) is characterised by development of new vessels from the surface of the retina or optic disc as a result of retinal ischaemia.

Optic disc neovascularisation in a diabetic subject generally indicates advanced diabetic retinopathy and is an indication for pan-retinal photocoagulation (PRP)\(^4\). As with new vessels elsewhere NVEs, the stimulus for NVDs is retinal ischaemia. It is uncommon to see NVD when the area of capillary non-perfusion is less than a quarter of the whole retina.

New vessels elsewhere (NVEs) nearly always develops from the venous sides of the capillary network adjacent to an area of retinal ischaemia.

Pathogenesis of neovascularisation

The exact pathogenesis of retinal neovascularisation is not fully understood, although there have been some significant advances in our understanding of this condition in recent years.

Glucose toxicity is one of the main causes of retinal micro-vascular cell damage in diabetes. This includes damage to the retinal vessel walls with loss of pericytes and endothelial cells, an increase in thickness of capillary basement membrane in addition to an alteration in red blood cell and platelet function. The resulting capillary closure leads to retinal ischaemia which in turn causes an upset in the complex interaction of local and systemic factors which maintain the normal status quo. This results in sufficient amounts of angiogenic growth factors to overcome the natural growth inhibitory mechanisms normally present. This imbalance leads to the dissolution of the extra-cellular matrix material of the vessel wall followed by cellular migration, proliferation, and finally formation of abnormal blood vessels.

The factors that control intra-ocular angiogenesis are still not fully understood. It is thought that systemic activation of leucocytes, particularly monocytes leads to capillary closure through increased adhesiveness of the leucocytes to the damaged endothelial cell surface of the capillary walls. Activated macrophages in the tissues, probably derived from a pool of circulating cells and from a resident microglial population, provide a rich source of growth factors and other molecules such as matrix-modifying enzymes which lead ultimately to an angiogenic response surrounding healthy endothelial cells. It is thought that, for example, heparin binding growth factors, transforming growth factors and endothelial growth factor (VEGF) which have all been identified in vitro angiogenetic studies, all may play a part.
NVD may be derived from the retinal or choroidal circulation, although it is more likely to be from the choroidal circulation if the new vessels originate from the deeper part of the cup. It is, however, difficult to be absolutely sure of the exact origin of the vessels and in any case makes no difference to the clinical management.

In the early stages it is easy to confuse NVD with fine, slightly dilated disc capillaries or even small disc collaterals. NVD does not however develop in the absence of signs of retinal ischaemia.

**Subsequent development of untreated NVD**

NVD may extend over the surface of the disc and across the disc margin in one or more quadrants. Often NVD follow the retinal vessels, especially along the temporal arcades. At the advancing edge of each vessel is a loop, the tip of which serves as a focus for new growth and extension of the vessel. The vessels usually grow between the internal limiting membrane of the retina and posterior vitreous face to which they eventually become adherent. Fibrous tissue accompanies the development of the new vessels and becomes progressively more clinically obvious.

**Treatment of NVD**

All patients with NVD should be considered for full scatter PRP\(^5\). This treatment should be undertaken as a matter of urgency.

The aim of the initial treatment session is to place as many laser applications as possible. It is best to separate each application by the diameter of the spot size used. Care should be taken to avoid the retinal vessels and, if present, any area of retinal detachment or fibrosis.

In a light PRP, the duration of the burn should be short, between 0.05 and 0.2s, whereas spot size can vary between 200\(\mu\)m and 500\(\mu\)m. Full thickness retinal burns are not required to achieve involution of new vessels. Therefore the power setting should be continually adjusted to achieve an end point equivalent to mild blanching of the retinal pigment epithelium (RPE).

Following treatment it important to advise the patient that their central vision is likely to be blurred for up to 10 days. The mechanism of this transient visual disturbance is unclear. Some loss is usually ascribed to temporary macular oedema and in others to altered accommodation. Permanent changes in colour vision and dark adaptation have been documented following PRP, although this may remain sub-clinical. Colour appeared normal in this case.
INDICATIONS FOR REFERRAL TO AN OPHTHALMOLOGIST

Protocols vary but the following are useful guidelines

1. Same day casualty referral
   - Retinal detachments
   - Rubeosis

2. Urgent Referral (ideally <2 weeks)
   - Definite new vessels on the disc (NVD)
   - Pre-retinal and/or vitreous haemorrhage

3. Early referral (ideally within 6-8 weeks)
   - Possible/ not definite NVDs
   - New vessels elsewhere
   - “High risk”, pre-proliferative retinopathy: IRMAS, venous beading, venous loops, clusters of large blot haemorrhages, multiple cotton wool spots.
   - Non proliferative retinopathy with macular involvement.
   - Haemorrhages and/or hard exudates within one disc diameter from the centre of the fovea not just microaneurysm.
   - Reduced visual acuity not corrected by pinhole, suggestive of macular oedema (not amblyopia)

4. Routine referral (within 13 weeks)
   - Non-proliferative retinopathy with large circinate or plaque exudate within the major temporal arcade but not threatening the macula
   - Retinal findings that are not characteristic of diabetic retinopathy
   - Background retinopathy with reduced vision but without maculopathy to determine cause of visual loss

5. No need for referral
   - Background retinopathy including microaneurysms within 1 disc diameter from the centre of the fovea, where the visual acuity (if necessary corrected by pinhole) is not reduced. This can include poor vision due to long standing amblyopia.
6. Review by optometrist in 12 months
   - Normal minimal and mild NPDR

7. Review by optometrist in 6 months
   - Two or more microaneurysms (MA s) within one disc area centred on foveola with no clinically significant macular oedema
   - Any definite exudate within 2 disc diameters (but more than 1 disc diameter) from fovea (however if exudate extensive consider referral)
   - Moderate NPDR

Conclusions

The mechanisms involved in the development of microangiopathy are complex and still not yet fully understood. Clinical features and their natural progression are described along with recommendations for referral criteria and management options. These are considered for each stage in the progression of diabetic retinopathy.
Example Referral letters

Example 1

18th July 2012

Dear J

Re: Mr. J S D.O.B. 14.2.1954
37 ………..London N………..

Routine report No diabetic retinopathy

This is a routine report following Mr. S's annual check. I understand that his medication is unchanged.

On examination I found his refraction and visual acuities to be R. -2.25/-1.25 x 80 = 6/5-. L. -2.25/-1.50 x 80 = 6/5-. Add +2.50DS for reading = N5 at 40cms.

External eyes, pupillary reflexes, ocular motility, ocular media are normal. The anterior chamber angles were grade 2. The ocular fundi appeared normal. There are no retinal diabetic changes. I photographed the retinas (OptoMap laser scanning 200° field).

Intraocular pressures were R. 11 L. 13 mmHg. Visual fields were full to Humphrey C40 screening. I have advised a routine examination in twelve month time.

Regards

Dr. Simon Barnard PhD, BSc, FCOptom, FAAO, DCLP, DipClinOptom DipTh(IP)

Example 2

……..  
London N…..

28th November 2012

Dear Dr M

Re: Mr D D DOB 05/01/1953
9…. London, N………..

Reason for referral: Macula oedema and cystic changes left macula Background retinopathy RE & LE

I saw Mr D for review today. He is happy with his vision. I understand he underwent laser therapy since I last saw him.

Refraction today was R. -5.50 /-1.25 X100 = 6/5-1 L. -3.50 /-2.75 X15 = 6/7.5+2 Add +2.50DS for reading = N4 at 40 cm.
Both eyes show diabetic retinopathy. The left macula shows cystic macula oedema with an increase in retinal thickness of 50 microns since I last saw him in September 2011. Intraocular pressures were R. 18 L. 19 mmHg. Visual fields were full to Humphrey C40 screening.

In conclusion, since I last saw Mr D the L. VA has reduced to 6/7.5 and there are cystic changes at the left macula. I would be grateful if he could be reviewed by his consultant ophthalmologist I have copied this to Dr E his endocrinologist with a further copy for the ophthalmologist.

Regards

Dr Simon Barnard PhD FCOptom FAAO DipCL DipClinOptom DipTh(IP)

Cc       Mr D
        Dr E

Reference


Acknowledgments

I wish to fully acknowledge the adaption of notes written by Chris Steele, OEL