



Screening for Diabetic Retinopathy in Primary Care

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Key concepts

- Sight-threatening diabetic retinopathy is largely preventable through regular retinal screening and prompt treatment
- Retinal screening should be carried out at least every two years. More frequent screening regimens are indicated by clinical risk factors such as the duration of diabetes and the degree of pre-existing retinopathy.
- Primary care plays a critical role in ensuring that patients are referred for and attend retinal screening so they can be treated before there is visual deterioration
- Maintaining good glycaemic control, treating hypertension and managing lifestyle risk factors, especially smoking cessation, is also essential

Supporting the PHO Performance Programme

“Get Checked” for diabetes complications

Approximately 5 – 7% of New Zealand adults have been diagnosed with type 1 or type 2 diabetes.¹ The actual number of people with diabetes is likely to be much higher than this. The self-reported prevalence of diabetes is two to three times higher among Pacific, Māori and Indo-Asian people.¹ Diabetes is a leading cause of blindness, end stage kidney failure and complications leading to lower limb amputation. It is a major risk factor for cardiovascular disease and early mortality.

Regular health checks are essential to reduce the frequency of complications from diabetes, as well as to minimise their impact. The “Get Checked” programme is a national initiative, offering free annual health reviews to people with diabetes, by their GP or practice nurse. The programme aims to promote early detection and intervention for problems associated with diabetes.

The “Get Checked” annual health review includes:

- A HbA_{1c} level
- Cardiovascular risk assessment, including blood pressure, lipid profile, height and weight
- Kidney function (microalbuminuria)
- Sensation and circulation of feet
- Retinal check (at least every two years)
- Follow-up plan for care

The annual check for people with diabetes is also a PHO Performance Programme (PPP) indicator.

The PPP goal is for at least 80% of all people with diabetes enrolled in a practice to have had a full annual “Get Checked” review each year.

In 2009 53% of the estimated number of people with diabetes in New Zealand had an annual “Get Checked” review.² This is an improvement from the previous year (46%),² but this number still falls considerably short of the PPP target of greater than 80%.

Between 2008 and 2009 annual reviews in the high needs population (identified as Māori and Pacific peoples and those living in lower socioeconomic areas) improved from 52% to 57%.²

There is much variation throughout DHB regions and PHOs, with some areas achieving better results. Technical difficulties in data collection may contribute to lower figures in some areas.

Consider the barriers to achieving this goal and ways in which the practice can address this. People with diabetes who are not receiving an annual free review, are potentially at a greater risk of developing harm from complications, which could have been treated if detected early enough.

Diabetic retinopathy is one of the leading causes of blindness in New Zealand

Diabetic retinopathy has been, until recently, the leading cause of preventable adult blindness and vision

impairment in New Zealand. Factors such as advances in treating retinal damage and more effective screening are slowly decreasing the prevalence of diabetic retinopathy in some areas.

The exact incidence of diabetic retinopathy is unknown but it is estimated that 30% of people with diabetes have some degree of retinopathy, with 10% having sight-threatening retinopathy.³ A New Zealand study of almost 12 000 people with diabetes, conducted between 2003 and 2005, found that almost one-third (32%) had some signs of diabetic retinopathy.⁴ There was also evidence that Māori were accessing the retinal screening service at a lower rate than other ethnic groups.⁴ As this study was based in one particular region of New Zealand (Wellington), incidence of diabetic retinopathy and disparities in accessing services, may be even greater in other areas. An earlier small study of almost 500 people with type 2 diabetes in South Auckland found that the prevalence of moderate to severe retinopathy was 4% in Europeans, 13% in Māori and 16% in Pacific peoples.⁵

The longer the duration of diabetes, the greater the prevalence of retinopathy. A large longitudinal study, based in the United Kingdom, found that the incidence of sight-threatening diabetic retinopathy after five years, in patients with diabetes (type 1 or 2) who had no signs of retinopathy at baseline, was 3.9%. In patients who initially had mild diabetic retinopathy, 15% had developed sight-threatening retinopathy by five years.⁶

Sight-threatening diabetic retinopathy is largely preventable, through regular retinal screening and prompt treatment. Primary care plays a critical role in ensuring that patients are referred for and attend retinal screening so they can be treated before avoidable loss of vision occurs.

Detecting and preventing diabetic retinopathy

Diabetic retinopathy is asymptomatic until it is at an advanced stage and then it is usually too late for effective treatment. Therefore early detection and prevention are imperative.

In primary care the two key responsibilities are:

- Referral for regular retinal screening at least every two years (and following-up on attendance and subsequent treatment if needed)
- Management of risk factors

Early detection of retinopathy with regular screening can save vision

The objectives of retinal screening in people with diabetes are to:³

1. Screen those with known diabetes for the onset of diabetic retinopathy
2. Identify those with early microvascular disease so primary care and diabetes teams can optimally manage risk factors such as glycaemic control and hypertension
3. Refer those with more significant retinopathy who are at risk of visual impairment for management and treatment by an ophthalmologist, before avoidable loss of vision occurs

N.B.: People with pre-diabetes (impaired glucose tolerance and impaired fasting glucose) do not require retinal screening and should not be referred.



Referral process for screening

- Make a referral to the local retinal screening provider
- Check with the patient at their next consultation, that they have been assigned an appointment time for retinal screening (or they have attended the appointment) and follow-up with the provider if this has not occurred
- Request and review a copy of the screening results, ensure that appropriate follow-up has occurred e.g. check that referral to an ophthalmologist has occurred if indicated, or make a note in the patient record that a more frequent screening interval has been recommended
- Place an automatic recall on the patient's notes for when screening is next due
- Follow-up patients who do not attend for screening, ask them what their difficulties in attending are, consider barriers to screening and how your practice may help address these

Each DHB has an individual arrangement with local providers for retinal screening (contact your local DHB if you are unfamiliar with referral options). Screening is usually performed by a suitably trained optometrist, photographic technician, ophthalmologist or other clinician. A designated ophthalmologist usually oversees each local retinal screening programme, to ensure consistency in grading of retinopathy.


Some areas may be under-resourced for the numbers of patients who require retinal screening. In some cases, if the public waiting list is too long, patients may be referred privately. A new study, soon to be published, suggests that the waiting time for referral to an ophthalmologist for moderate background retinopathy or mild maculopathy varies considerably throughout the country, but in most cases is less than the recommended referral time for this grade of disease (four to six months).⁷

Do not wait for signs and symptoms to occur

Early retinopathy is asymptomatic. Signs of blurred or

fluctuating vision, spots or “floaters”, if related to diabetic retinopathy, are most often associated with advanced disease.

People with diabetes who present with an acute impairment of vision from any cause should be referred for urgent review with an ophthalmologist/eye clinic.

 **Best practice tip:** Retinal photo-screening for diabetic retinopathy does not constitute a full eye examination. Patients should still be regularly reviewed for other eye pathologies such as cataracts or glaucoma. Primary care clinicians can test visual acuity using an eye chart and pinhole. **As a general rule, if visual acuity improves with pinhole testing, then it is more likely that any reduction in visual acuity is due to a refractive error (and may require subsequent referral to an optometrist) rather than due to pathology in the eye (which would require referral to an ophthalmologist).**

Screening intervals


New Zealand guidelines recommend that retinal screening is carried out every two years for a person with diabetes who does not have retinopathy (Table 1).³

A referral for screening should be made at the time of a confirmed diagnosis for people with type 2 diabetes because many people already have some degree of retinopathy at this stage. With type 1 diabetes, vision threatening retinopathy is very rare in the first five years after diagnosis or before puberty so screening may commence after this time.³

For people with diabetes who have early signs of retinopathy, screening should be more frequent. The frequency of screening is determined by the Guidelines and the clinician's opinion, taking into consideration factors such as the severity of the retinopathy, glycaemic control, blood pressure and the risk of progression (see sidebar).³

Diabetic retinopathy can progress rapidly during pregnancy. Women with diabetes who become pregnant should be

screened in the first trimester of their pregnancy. If no retinopathy is detected and the diabetes is well controlled the two-yearly screening schedule may be continued. If a minimal degree of retinopathy is detected or if the diabetes is not well controlled, three-monthly screening for the remainder of the pregnancy is recommended. Referral to an ophthalmologist is required if more than minimal retinopathy is detected.³ N.B. Women who develop gestational diabetes during pregnancy are not generally at increased risk of retinopathy unless they have pre-existing disease.

 Copies of the Ministry of Health retinopathy screening guidelines and a CD for training purposes can be obtained from:

www.moh.govt.nz/moh.nsf/indexmh/retinal-screening-grading-and-referral-guidelines-2006-resources-2008

Retinal screening methods

The current “gold standard” method for screening for diabetic retinopathy in New Zealand is digital photography of the retina while the pupil is dilated. Non-mydratiac photography is however widely used and, although not

suitable for every patient, does avoid the inconvenience of pupil dilation. If retinal photography is unavailable the fundus (interior surface of the eye) can be examined through a dilated pupil using slit-lamp biomicroscopy. An assessment of visual acuity should also be carried out.³

Conventional retinal examination involves using an ophthalmoscope to view the fundus through a dilated pupil, in a darkened room. However it is difficult for even the most experienced examiners to achieve high sensitivity of retinopathy detection with this method. Macular oedema is also not generally able to be detected with an ophthalmoscope.

After screening, the examiner grades the degree of retinopathy in each eye and applies an overall grading, depending on the worst affected eye. It is important that the examiner follows standardised New Zealand screening protocols for grading.³ The grade of retinopathy determines what follow-up action is taken.

Fluorescein angiography can be used to detect macular oedema if this is suspected. This involves dye being injected into the arm and images taken as the dye progresses through the blood vessels in the retina.

Table 1: Summary of screening recommendations for diabetic retinopathy³

	First retinal screen	Screening interval: no retinopathy	Screening interval: retinopathy detected
Type 1 diabetes	Five years after diagnosis or after puberty	Two-yearly	More frequent than two-yearly, determined by severity, glycaemic control and other risk factors
Type 2 diabetes	Soon as possible after confirmed diagnosis	Two-yearly	
Pregnancy + diabetes	First trimester of pregnancy	Two-yearly	Frequent throughout pregnancy (also if poor glycaemic control, even if no retinopathy)

Management of risk factors for diabetic retinopathy

The duration of diabetes is the most significant risk factor for diabetic retinopathy.^{3, 6, 9} Poor glycaemic control is also a major contributor to both the risk of development and progression of diabetic retinopathy.³ Other modifiable risk factors include hypertension and nephropathy.³ Elevated blood lipid levels have a weaker association with diabetic retinopathy but contribute to overall cardiovascular risk in a patient with diabetes.

If a person with diabetes is found to have signs of mild retinopathy, managing risk factors can help prevent more advanced changes from developing.

To reduce the risk of progression of diabetic retinopathy, focus on:

- Maintaining good glycaemic control – establish an individualised HbA_{1c} target (see Page 8)
- Managing hypertension – New Zealand cardiovascular guidelines recommend reducing blood pressure to < 130/80 mm Hg for people with diabetes,¹⁰ however this level may not be achievable for some people. In the presence of microalbuminuria or renal disease more aggressive control may be required to reduce blood pressure to < 125/75 mm Hg.¹⁰
- Advising on management of lifestyle factors, especially smoking cessation and promoting exercise and a healthy diet
- Reducing blood lipid levels as part of overall cardiovascular health – aim for a reduction towards the target level of total cholesterol < 4.0 mmol/L,¹⁰ although this level may not always be achievable (see Page 16)

Intensive glycaemic control

Factors that worsen the general prognosis for people with diabetes also worsen diabetic retinopathy. Intensive glycaemic control has been found to reduce the rate of

Clinical factors that may affect screening intervals

In some circumstances, risk factors may be present indicating that earlier re-screening or referral to an ophthalmologist should be considered.

These factors include:³

- Poor compliance – failure to attend appointments for screening on two or more occasions
- Poorly controlled diabetes – HbA_{1c} > 75 mmol/mol (> 9%)
- Duration of diabetes – including type 1 diabetes for greater than seven years
- Rate of progression of retinopathy
- Insulin treatment in people with type 2 diabetes
- Poorly controlled hypertension
- Renal failure
- Ethnicity – Māori, Pacific and Asian peoples are at a higher risk of complications of diabetes
- Asymmetrical disease – i.e. significant worsening in one eye

Pupil dilation

Retinal examination usually involves pupil dilation using tropicamide 1% eye drops. This is safe for most people with diabetes, including those being treated for chronic open angle glaucoma (but not closed angle glaucoma).

Patients should be warned that pupil dilatation may temporarily cause:³


- Distorted vision
- Disturbance of balance
- Lack of tolerance to bright light or sunlight – advise patients to take sunglasses with them to their clinic appointment
- Impairment in driving or using machinery – advise patients to arrange transport to and from their appointment and to **avoid driving** or using machinery for several hours afterwards (until vision returns to normal)

In extremely rare cases, dilation of the pupil can cause acute closed-angle glaucoma. This occurs because the iris is pushed into the angle (the junction of the iris and the cornea), blocking drainage and increasing intraocular pressure, which in turn can damage the optic nerve. Symptoms include pain, redness and decreased vision in the affected eye, coloured halos around lights, headache, nausea and vomiting. Acute closed-angle glaucoma is an emergency situation but is usually able to be treated using a regimen of ocular drugs and laser treatment.⁸



progression of diabetic retinopathy. The Action to Control Cardiovascular Risk in Diabetes (ACCORD) study placed people with type 2 diabetes on either intensive glycaemic control (target HbA_{1c} level of <6.0%) or standard treatment (target HbA_{1c} level of 7.0 – 7.9%). After four years, diabetic retinopathy had progressed in 7.3% of people on intensive glycaemic control compared with progression in 10.4% of people on standard therapy (P = 0.003).¹²

Intensive glycaemic control appears to reduce the risk of most complications of diabetes, including retinopathy but it has to be balanced against the increase in risks, such as severe hypoglycaemia, and some concerns about increased risk of mortality.

 See article “HbA_{1c} targets in people with type 2 diabetes – do they matter” on Page 8 for more information about intensive glycaemic control, including discussion of risk factors and adverse effects.

Hypertension as an independent risk factor

Hypertension is an independent risk factor for visual degradation. Over time, hypertension directly damages the retina, choroid and optic nerve and can result in events such as retinal vein and artery occlusion, retinal emboli, ischaemic optic neuropathy, glaucoma and age related macular degeneration. People with diabetes and poor blood pressure control are at an increased risk of progression of diabetic retinopathy.¹³

Management of hypertension is essential for people with diabetes, however there is conflicting evidence as to whether intensive control is beneficial for reducing progression of diabetic retinopathy, and what blood pressure level is required. The UK Prospective Diabetes Study (UKPDS) found a 34% reduction in the risk of progression of retinopathy after nine years in patients with tight control of blood pressure (target < 150/85 mmHg, using captopril or atenolol).¹⁴ However, the later ACCORD study found no significant benefit for diabetic retinopathy progression in those on intensive hypertensive control (< 120 mmHg, 10.4%) compared with standard treatment (<140 mmHg, 8.8%), nor a benefit in cardiovascular

outcomes.¹² The differing parameters for intensive blood pressure control and time frames of these studies may have contributed to the different conclusions.

Intensive lipid control

Optimal lipid levels are advised in people with diabetes for cardiovascular risk reduction, but the beneficial effect of intensive lipid control on diabetic retinopathy is less clear, and there is little or no evidence that statins have any benefit for retinopathy.

The Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study, which involved patients from New Zealand, Australia and Finland showed that there was a beneficial reduction in microvascular complications in people with type 2 diabetes taking fenofibrate for lipid control.¹⁵ Researchers found that laser therapy for retinopathy was needed more frequently in people with poorer glycaemic control or blood pressure control, but plasma lipid concentrations were not associated with the need for laser treatment. Patients on fenofibrate therapy (3.1%) had a reduced need for laser treatment for retinopathy compared to placebo (14.6%), but only if they had a degree of pre-existing retinopathy before lipid-lowering treatment commenced ($P = 0.004$). There was no significant difference in the need for laser treatment between people taking fenofibrate (9.6%) and placebo (12.3%) overall ($P = 0.19$) or in the subset of patients without pre-existing retinopathy (11.4% fenofibrate group vs. 11.7% placebo, $P = 0.87$). Researchers concluded that fenofibrate appears to reduce the need for laser treatment for diabetic retinopathy, but that this mechanism was not related to plasma concentrations of lipids.¹⁵

The recent ACCORD study found that diabetic retinopathy had progressed in 6.5% of people on intensive dyslipidaemia therapy (160 mg daily of fenofibrate plus simvastatin) compared with 10.2% on standard therapy (simvastatin plus placebo) ($P = 0.006$).¹²

Fenofibrate is not currently registered in New Zealand or funded on the pharmaceutical schedule. It is in the fibric acid derivative class of drugs which also includes

Aspirin and retinopathy

Many patients with diabetes and a high cardiovascular risk will be taking aspirin. If the patient also has retinopathy, should aspirin be continued?

The Early Treatment Diabetic Retinopathy Study found that 650 mg aspirin per day had no effect on the progression of retinopathy or the development and duration of vitreous haemorrhage. It was concluded that aspirin is not beneficial for the treatment of retinopathy, but there is normally no contraindication to its use in patients with retinopathy, when required for cardiovascular disease or other indications.¹¹



bezafibrate (registered and funded) and gemfibrozil (registered). Statins remain the primary agent for lipid control in people with type 2 diabetes.

Other micro- and macrovascular complications

There is a strong association between diabetic retinopathy and other microvascular complications such as nephropathy and neuropathy, and all are associated with a higher cardiovascular risk. Managing risk factors for retinopathy will generally also decrease the risk of these other complications of diabetes.

A person with coexisting retinopathy, renal and foot disease is at a high cardiovascular risk as well as high risk for requiring amputation.

Diabetic “foot-eye” syndrome is a distinct set of symptoms that has been observed in people, usually with type 2 diabetes. It is characterised by:¹⁶

- A foot ulcer attributable to peripheral neuropathy
- Diabetic retinopathy
- Self neglect, indifference towards illness

Patients who present with “foot-eye” syndrome have a poor prognosis due to underlying severe cardiovascular disease.¹⁶

Genetic factors may also play a role

Despite good diabetic control and management of risk factors, diabetic retinopathy still progresses in some people. Conversely, some people with uncontrolled risk factors do not progress to diabetic retinopathy. Genetic factors are thought to play a role in the susceptibility to diabetic retinopathy.¹⁷

Getting help with reduced vision

Community organisations and agencies such as the Royal New Zealand Foundation of the Blind offer information about low vision counselling, training, and other special services for people with vision impairments.

For further information visit: www.rnzfb.org.nz



Characteristics of diabetic retinopathy

What causes diabetic retinopathy and how does it manifest?

The microvascular changes that occur throughout the body as a result of diabetes, also affect the eye. Microvascular changes within the retina are the most likely to adversely affect vision.

Diabetic retinopathy can be classified as non-proliferative (background) or proliferative. Visual loss and blindness occurs via two different mechanisms – macular oedema and proliferative retinopathy. Macular oedema can occur at any stage of diabetic retinopathy and is caused by blood vessel leakage and retinal thickening, due to breakdown of the blood-retinal barrier. It is the most frequent cause of vision loss in people with type 2 diabetes. Proliferative retinopathy occurs as a result of vascularisation induced by ischaemia, causing loss of vision due to haemorrhage or retinal detachment.¹⁸

Non-proliferative retinopathy

Lesions include:

- Microaneurysms – small swellings that form on small retinal blood vessels; if the swellings leak or burst, plasma or blood can leak into the adjacent tissue.
- Haemorrhages – leakage of blood from the small vessels into adjacent tissues; haemorrhages deeper in the retinal tissue (dot and blot) are more common in diabetes, superficial haemorrhages (flame) in the nerve fibre layer are more common with hypertension
- Hard exudates – leakage of serum proteins and lipids caused by breakdown of the blood-retina barrier; appearing as white or yellow crystalline deposits in the retina, sometimes forming a circular



Above: A normal retina



Above: Retina showing signs of retinopathy including exudates and haemorrhages³

pattern around a leaking microaneurysm

- Cotton wool spots – formed from an accumulation of axoplasm due to occlusion of pre-capillary arterioles; appearing as soft, fluffy, white lesions, often at right angles to the direction of the nerve fibre layer
- Macular oedema – damage to the central vision caused by functional damage and necrosis of retinal capillaries; the leading cause of visual impairment in people with diabetes
- Venous loops and beading – damage to vessel walls resulting in formation of loops or beading (sausage shaping) of the blood vessels; often a sign that more severe proliferative retinopathy is developing

Proliferative retinopathy

- Restricted blood supply to the retina (retinal ischaemia) caused by diabetes, can lead to the release of Vascular Endothelial Growth Factor (VEGF) which initiates the formation of new blood vessels (neo-vascularisation)
- The new blood vessels break through and grow along the surface of the retina, the posterior hyaloid face (the wall between the retina and the vitreous cavity inside the eye) and in severe cases, the iris
- The new blood vessels are fragile and easily broken, leading to haemorrhage in the vitreous cavity or pre-retinal space, compromising vision almost immediately
- Over time, as the density of the newly formed blood vessels increases, fibrous tissue is formed which can adhere to the retina and posterior hyaloid face of the eye
- The traction between the vitreous and the fibrous tissue connections can cause retinal oedema, tears and detachments

Symptoms

Early to moderate stages of diabetic retinopathy are usually without any noticeable symptoms. Symptoms may be experienced in proliferative retinopathy, particularly with haemorrhage and retinal detachment.

Vitreous haemorrhage occurs suddenly but is not usually associated with any pain. The blood which enters the vitreous cavity occludes the vision and is seen as spots or areas of visual loss. If not treated, repeated haemorrhages result in progressive visual loss in most cases.¹⁹

Retinal detachment is described as flashing lights and floating spots in the peripheral vision or as a “curtain” progressing across the visual field.²⁰

NB: Sudden onset of visual symptoms or deterioration requires referral for specialist assessment, rather than for routine screening for retinopathy.

How is diabetic retinopathy treated?

Laser photocoagulation is the primary treatment for sight-threatening diabetic retinopathy, however it is not always completely successful in restoring visual loss. Better results are achieved if treatment is carried out earlier in the disease process.¹⁸ For the majority of cases the goal of treatment with laser photocoagulation is to reduce the rate of visual loss or to stabilise visual acuity.

During laser photocoagulation, a laser is applied to the retina, causing burns. Different methods of laser surgery (or combinations of methods) are carried out depending on the pathology being treated.

Surgery is usually completed in one session, but if both eyes require treatment, this normally occurs several weeks apart.

Adverse effects and complications of laser photocoagulation can include:

- Headache – usually relieved with rest and simple analgesia, but if severe or persistent, glaucoma must be ruled out
- Pain – anaesthetic drops are applied, but an uncomfortable stinging sensation can occur as time progresses
- Blurred vision (temporary)
- Visual field restriction
- Decreased contrast sensitivity
- Impaired night vision or colour vision

Vitreous surgery

Vitreous surgery may be required in patients with some types of retinal detachment, vitreous haemorrhages and severe proliferative retinopathy. Vitreous surgery has the potential for serious complications including significant visual loss and eye pain.

A vitrectomy is performed under local or general anaesthesia. A tiny incision is made in the eye and the vitreous gel clouded by blood is replaced by a saline solution. After the procedure the eye may be red and sensitive, an eye patch may be required for a few days or weeks to protect the vision, and antibiotic eye drops may be required to reduce the risk of infection.

Intravitreal corticosteroids

If laser photocoagulation has been unsuccessful, in some cases patients may be trialled on corticosteroids, injected into the vitreous of the eye. This method can be effective, but re-injections are usually needed and there is a potential for significant adverse effects such as infection, glaucoma and cataract formation.¹⁸



“The capacity to blunder slightly is the real marvel of DNA. Without this special attribute, we would still be anaerobic bacteria and there would be no music.” — Lewis Thomas

Improve patient safety by sharing solutions and prevent these incidents from occurring again. Report patient safety incidents here:

www.bpac.org.nz/safety

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References

1. Ministry of Health (MoH). A portrait of health: key results of the 2006/2007 New Zealand Health Survey. Wellington: MoH, 2008. Available from: www.moh.govt.nz (Accessed August, 2010).
2. District Health Boards New Zealand (DHBNZ). Reporting back to the public - DHB Performance results June 2009. Wellington: DHBNZ, 2010. Available from: www.dhbnz.org.nz (Accessed August, 2010).
3. Ministry of Health (MoH). National diabetes retinal screening grading system and referral guidelines 2006. Wellington: MoH, 2008. Available from: www.moh.govt.nz (Accessed August, 2010).
4. Frederikson L, Jacobs R. Diabetes eye screening in the Wellington region of New Zealand: characteristics of the enrolled population (2002 - 2005). *N Z Med J* 2008;121(1270).
5. Simmons D, Clover G, Hope C. Ethnic differences in diabetic retinopathy. *Diabet Med* 2007;24(10):1093-8.
6. Younis N, Broadbent D, Vora J, Harding S. Incidence of sight-threatening retinopathy in patients with type 2 diabetes in the Liverpool Diabetic Eye Study: a cohort study. *Lancet* 2003;361:195-200.
7. Hutchins E, Sanderson G, Rahman A, et al. Factors affecting effective implementation of the National Diabetes Retinal Screening grading system and referral guidelines: A multi centre analysis: Unpublished report to the Health Research Council New Zealand (pers comm G. Sanderson).
8. Merck. The Merck manual for healthcare professionals; Angle-closure glaucoma: Merck, 2008. Available from: www.merck.com/mmpe/sec09/ch103/ch103c.html (Accessed July, 2010).
9. Cikamatana L, Mitchell P, Rochtchina E, et al. Five-year incidence and progression of diabetic retinopathy in a defined older population: the Blue Mountains Eye Study. *Eye* 2007;21(4):465-71.
10. New Zealand Guidelines Group (NZGG). New Zealand cardiovascular guidelines handbook. 2nd ed. Wellington: NZGG, 2009.
11. Early Treatment Diabetic Reintopathy Study (ETDRS) Research Group. Effects of aspirin treatment on diabetic retinopathy. ETDRS report number 8. *Ophthalmology* 1991;98(5 Suppl):757-65.
12. The ACCORD Study Group and ACCORD Eye Study Group. Effects of medical therapies on retinopathy progression in type 2 diabetes. *N Engl J Med* 2010;363(3):233-44.
13. Spurling G, Askew D, Jackson C. Retinopathy screening recommendations. *Aust Fam Physician* 2009;38(10):780-3.
14. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. *BMJ* 1998;317(7160):703-13.
15. Keech A, Mitchell P, Summanen P, et al. Effect of fenofibrate on the need for laser treatment for diabetic retinopathy (FIELD study): a randomised controlled trial. *Lancet* 2007;370(9600):1687-97.
16. Walsh C, Soler N, Fitzgerald M, Malins J. Association of foot lesions with retinopathy in patients with newly diagnosed diabetes. *Lancet* 1975;1(7912):878-80.
17. Liew G, Klein R, Wong T. The role of genetics in susceptibility to diabetic retinopathy. *Int Ophthalmol Clin* 2009;49(2):35-52.
18. Simo R, Hernandez C. Advances in the medical treatment of diabetic retinopathy. *Diabet Care* 2009;32(8):1556-62.
19. Watkins P. ABC of diabetes: retinopathy. *BMJ* 2003;326:924-6.
20. Goold L, Durkin S, Crompton J. Sudden loss of vision: history and examination. *Austr Fam Phy* 2009;38(10):764-8.