

***Optometric Care of the Patient with Diabetes***

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## Introduction

The objective of this document is to provide the reader with an overview of the epidemic of diabetes currently facing Canada and the morbidity and mortality associated with this growing healthcare burden.

Specifically, an evidence based, patient centered, cost effective role of the optometrist in the eye care of Canadians with diabetes will be presented.

## Diabetes in Canada

The current number of people living with diabetes in Canada is approximately two million [1]. Type 2 diabetes is the most common form of the disease, accounting for 90% of cases. The prevalence of type 2 diabetes is increasing dramatically with current estimates confidently predicting a doubling of cases by 2025 [2,3]. In addition, the prevalence of diabetes in First Nations people in Canada is substantially higher than in the general population [4]. The “diabetes epidemic” is projected to increase the total number of cases of diabetes in Canada to three million by the end of this decade. Diabetes represents a potential doomsday threat to human health in the 21<sup>st</sup> century [5,6]. It has been estimated that diabetes is a contributing factor in the deaths of 41,500 Canadians per year and this number will undoubtedly increase. [7]

In terms of morbidity, diabetes affects almost every organ in the body. Diabetes has a strong association with hypertension, dyslipidemia and obesity. Diabetes doubles the risk of stroke, causes 33 percent of the new cases of end stage renal disease, quadruples the risk of heart disease, is the leading cause of non-traumatic lower extremity amputations, and is the leading cause of blindness in adults aged 25-75 and can also cause a painful form of nerve damage called diabetic neuropathy. [8] Statistics have shown that the life expectancy of a person with type 2 diabetes can be shortened by 5 - 10 years. [9]

The anticipated increase in type 2 diabetes over the next 10 to 20 years will have an unprecedented impact upon Canadian health care services. In 2003, Health Canada [10] estimated that at least \$13.5 billion was spent annually on treating people with diabetes and its complications. The American Diabetes Association clearly set the scene for the integration of healthcare professionals when it stated “*It is time for all health professionals to treat diabetes aggressively. It is also time for patients to take their diabetes with utmost seriousness. And it is incumbent upon the health care system to provide the necessary resources for both to be successful*” [11]. Stakeholders including the Canadian Medical Association, the Canadian Diabetes Association, the Canadian Ophthalmological Society and the Canadian Association of Optometrists have declared a need for all health professionals to develop an integrated delivery system that ensures a best quality of care approach for people with diabetes. [12, 13]

# Types of Diabetes

## Type 1 Diabetes

This type of diabetes accounts for approximately 10% of the cases of diabetes. Type 1 diabetes is characterised by autoimmune pancreatic  $\beta$ -cell destruction that usually leads to absolute insulin deficiency. It generally presents as an acute onset in young people who often have a history of a preceding infection, often viral. Although type 1 diabetes is no longer considered a disorder limited to young people [14], the initial presentation of the disease often occurs in a child who may present with a spectrum of symptoms from mild, non-specific to a coma. [15] People with newly diagnosed type 1 diabetes can present with weight loss, lethargy, polyuria, polydipsia, loss of appetite, nausea, vomiting, blurred vision and altered mental status. [16] Polyuria, polydipsia and blurred vision result from a hyperosmolar state, while weight loss results from break down of amino acids into glucose and ketones. The acute complications of diabetes include insulin shock, hypoglycemia, ketoacidosis (DKA) and hyperosmolar nonketotic syndrome (HNKS). DKA is a common presentation with this disease and there is an increased risk of cerebral edema (CE) in DKA. [17] Mortality rates from DKA are reported to be 0.15 – 0.31% and CE accounts for between 57 -87% of these deaths. [18, 19] Diabetic patients with DKA will often manifest ketone breath.

The patho-physiology of type 1 diabetes is believed to be an autoimmune process whereby the beta cells of the pancreas, which produce insulin, are destroyed [20]. The autoimmune process may be initiated by a reaction to an infection, such as German measles and cytomegalovirus, or even vaccine administration, although this process is not well understood and the evidence supporting it is very weak. Furthermore, the autoimmune response may be influenced by antibodies against proteins in cow milk. Indeed, the evidence supporting the role of environmental factors is so weak that some argue that they may merely serve as modifiers of disease pathogenesis rather than as triggers. A genetic vulnerability to develop type 1 diabetes has also been implicated.

Insulin acts as a gateway to allow glucose to cross the cell membranes of insulin dependant tissues; mainly adipose and muscle tissue. These tissues now must use other substrates such as free fatty acids for energy sources which leads to the formation of ketones and eventually DKA. Insulin independent tissues such as brain, nervous system, cortex of the kidney, and bone marrow are flooded with the increased blood levels of glucose and become damaged by the formation of advanced glycosylation / glycated end products (AGEs). In addition, the diversion of glucose metabolism into sorbitol pathway results in the excess production of sorbitol and fructose causing osmotic stress. Chronic hyperglycemia results in a host of additional metabolic events that further drive tissue damage, including abnormalities of lipid metabolism, increased oxidative damage, hyperinsulinemia, hyperperfusion, hyperviscosity, platelet dysfunction and the activation of growth factors. These events lead to cataract, microvascular disease of the eyes (neovascular glaucoma and retinopathy), kidney (nephropathy) and nerves (neuropathy).

People with type 1 diabetes rarely show ocular complications until at least 5 years after diagnosis. After 10 years, 60% will have some degree of diabetic retinopathy and, at 15 years, it is likely that all people living with type 1 diabetes will have diabetic retinopathy. After 15yrs duration of diabetes, 42% of people with type 1 diabetes will develop diabetic macular edema (DME) [21]. At the 15 year mark, 23% will progress to PDR and after 20 years 50% will progress to PDR. [22, 23, 24].

## **Type 2 Diabetes**

The incidence of type 2 diabetes increases significantly with increase in age, typically manifesting in middle age or the later part of life, although childhood / adolescent type 2 diabetes is now also apparent. Approximately 90% of the cases of diabetes are type 2. Type 2 diabetes is characterised by a mix of insulin resistance and insulin secretory defect (β-cell exhaustion). This disease generally develops slowly with the first sign being peripheral insulin resistance. The pancreas responds by increasing its output of insulin leading to hyperinsulinemia. This may keep blood glucose levels near normal for years until glucose intolerance develops and pancreatic function decreases. Eventually blood glucose levels increase as does the risk for microvascular disease. Like type 1 diabetes, the patho-physiology of type 2 diabetes is multi-factorial and not fully understood. A genetic link is strongly implicated and a change in lifestyles over the last century from “famine to feast” in combination with less physical activity is also suspected [6]. Understanding of the clinical presentation of type 2 diabetes is also changing. In particular, there appears to be a link between type 2 diabetes and mental illness, particularly depression and anxiety. There is a doubling of the risk of depression in people with diabetes and there is an association between depression and hyperglycemia and diabetes complication risk [25]. Furthermore, the prevalence of type 2 diabetes is far higher in patients with schizophrenia, bipolar disorder or a severe depressive disorder [26], to the extent that schizophrenia is now a recognized risk factor for the development of type 2 diabetes.

Type 2 diabetes in particular (and cardiovascular and cerebrovascular disease) is often preceded by metabolic syndrome. Metabolic syndrome is a cluster of metabolic risk factors that come together in a single individual. Those risk factors include insulin resistance, hyperglycemia, obesity, hypertension and dyslipidemia (elevated low density lipoproteins, LDLs / reduced high density lipoproteins, HDLs, which result in increased blood coagulation abnormalities). As a result, many patients with diabetes, especially type 2, also have concomitant hypertension and lipid abnormalities which generate the characteristic retinal signs of arterial / vein “nicking” and hard exudates, respectively, alongside the signs of diabetic retinopathy. Twenty to 30% of people in industrialized countries are thought to have metabolic syndrome and the prevalence rises to 60% in the obese.

It has been reported that 80-90% of newly diagnosed people with type 2 diabetes are obese. Obesity is clinically defined as a body mass index (BMI) of greater than 30. [27] There is an obesity epidemic in Canada with obesity rates in the last 15 years in children increasing from 2 – 10% in males and from 2 – 9% in females. [28] Obesity among adults

has doubled from 1980-2000. [29] Other risk factors for type 2 diabetes are greater than 9 lb birth weight, a female with a history of gestational diabetes, smoking, other vascular diseases and a prior history of glucose intolerance. [30] It has been shown that 20% of people with type 2 diabetes will have some diabetic retinopathy at the time of diagnosis. At 15 years 60-85% will have some diabetic retinopathy. Only 4% will develop PDR within 4 years of diagnosis and after 15 years 5- 20% will develop PDR. After 15yrs duration of diabetes, 80% of people with type 2 diabetes will develop DME [21].

## **Gestational Diabetes**

This type of diabetes occurs in about 5 % of all pregnancies. Gestational Diabetes increases the risk of hypertension and caesarean section. The risk of stillbirth in women with diabetes in pregnancy is almost twice as high as those women without diabetes in pregnancy. [31] Gestational diabetes is associated with enlarged fetal abdominal circumference and fetal macrosomia and offspring of women with gestational diabetes have been shown to be more at risk of obesity and to have a higher prevalence of type 2 diabetes later in life. [32] Gestational diabetes that occurs during pregnancy has not been associated with the development of diabetic retinopathy during that pregnancy.

## **Treatment of Type 1 Diabetes**

The person with type 1 diabetes must use exogenous insulin to live, typically injected sub-cutaneously, although insulin pump, oral, nasal, transdermal and inhalation methods of insulin delivery are also either established or in various phases of development. They are taught by the diabetes educator to titrate the amount and type of insulin with their blood glucose and caloric intake. Various types of insulin with differing modes of action (i.e. rapid, short and long acting) are now available that may be used in combination to provide improved diurnal fluctuation, and tighter long-term, glycemc control.

## **Treatment of Type 2 Diabetes**

Diet and exercise remain the cornerstones of type 2 diabetes treatment, along with appropriate control of blood pressure and treatment of dyslipidemia. With reduced caloric intake that results in an average loss of 5% of initial body weight there is an associated 58% decrease in progression to type 2 diabetes in those people with impaired glucose tolerance. [33] The Clinical Practice Guidelines of the Canadian Diabetes Association recommends that persons with type 2 diabetes accumulate at least 150 minutes of moderate intensity aerobic exercise each week, over at least 3 non-consecutive days, or 4 or more hours of exercise per week. [34]

If diet and exercise do not allow the person to meet their blood glucose targets then medications are indicated. The first choice medication is a biguanide (Metformin). It works by acting on the liver to produce less glucose and on the muscle to use glucose in the bloodstream. The second choice of medications are called the insulin sensitizers; rosiglitazone (Avandia) and pioglitazone (Actos). These medications work by making the body more sensitive to its own insulin. The third choice of medications are the insulin

secretagogues which fall into two categories; the sulfonylureas, glyburide (DiaBeta), glicazide (Diamicon and DiamiconMR) and glimepiride (Amaryl) and the nonsulfonylureas; repaglinide (Gluconorm) and nateglinide (Starlix). These medications work by stimulating the pancreas to release insulin. Also, the alpha-glucosidase inhibitors (Prandase) works by blocking an enzyme in the intestine that breaks down complex carbohydrates into glucose. The dipeptidyl peptidase 4 (DPP-4) inhibitor sitagliptin (Januvia) has been recently introduced which simultaneously stimulates the secretion of insulin and suppresses the release of glucagon (thereby reducing the amount of glucose released into the blood from the liver) by the pancreas.

Irrespective of type of diabetes, the key to the management of this condition is to keep blood glucose, blood pressure and cholesterol within healthy limits. In addition, diet and weight management and increased physical activity are essential, along with appropriate eyecare, footcare, etc.

## **Vision loss and aging**

Canadians fear loss of sight more than loss of any other sense. Vision loss has an enormous impact on quality-of-life and is extremely costly from a societal and economic perspective ([www.costofblindness.org](http://www.costofblindness.org)). The aging of the Canadian population is predicted to have disastrous consequences in terms of rates of vision loss, as the baby boom generation reaches their 60's [35, 36]. By 65 years of age, 1 in 9 individuals will experience severe vision loss and this will increase to 1 in 4 individuals at 75 years [37]; these trends are even more pronounced for people of First Nations origin.

## **Diabetes and the eye**

The effect of diabetes on the eye and vision can range from transient mild blurred vision to blindness. People with diabetes are at increased risk of extraocular muscle palsies, most commonly of cranial nerves 3,6 and rarely 4. Cataracts develop in persons with diabetes at an earlier age than the general population [38]. Neovascular glaucoma, thought to be induced by the same mechanism as PDR, is a very difficult type of glaucoma to treat and it carries a poor prognosis for sight. Microvascular disease of the retina, diabetic retinopathy, remains the most serious, sight-threatening complication of diabetes. Diabetic retinopathy is the leading cause of visual impairment & blindness in Canadians between the ages of 30 and 69 [39]. Individuals with diabetes are 25 times more likely to become blind than persons in the general population [40]. Sight loss associated with diabetic retinopathy occurs due to diabetic macular edema (DME) / maculopathy and due to the sequelae of PDR, namely traction retinal detachment and vitreous haemorrhage. DME usually results in slowly developing visual loss but represents the most common cause of visual impairment & blindness amongst people with diabetes, while PDR results in sudden onset, severe visual loss ( $VA \leq 5 / 200$ ). The vascular changes that occur in the eye are predictive of vascular changes occurring in other parts of the body. [41, 42] A comprehensive eye and vision examination by an optometrist can lead to the detection of the sight-threatening complications of diabetes

and some of the 50% of the population that are living with diabetes but are yet undiagnosed.

## **The Retina**

The retina is a semitransparent nerve tissue membrane that is approximately 200 microns thick; about the thickness of a fingernail. It lines the inside surface of the back of the eye and it is sandwiched between two blood supplies; behind the retina is the choroidal circulation and on the front surface of the retina is the retinal circulation. Both of these blood supplies are derived from the ophthalmic artery which is the first branch of the internal carotid artery. The retina is a non-insulin dependant tissue meaning that glucose readily enters the cells of the retina without the need of insulin and hence if blood glucose is too high it can cause toxicity to the retinal tissue.

## **Pathophysiology of Diabetic Retinopathy**

The “hallmark” of diabetic retinopathy is retinal microvasculature instability, decompensation and collapse [43]. From a morphological perspective, these microvascular changes result in basement membrane thickening of the capillaries, pericyte loss, smooth muscle cell depletion, vascular endothelial cell loss, vascular occlusion & re-canalization and ultimately neovascularisation. This damage to the retinal vessels causes closure of capillary beds leading to edema and ischemia.

Vascular endothelial growth factor (VEGF), a cytokine released by the retinal vascular cells, is thought to govern vascular permeability in response to hyperglycemia and also initiate angiogenesis in response to hypoxia. In terms of DME, VEGF and advanced glycation end-products are proposed to up-regulate the level of intercellular adhesion molecules (ICAM-1) and, in turn, this results in an increased level of leukostasis in the retinal vessels, increased vascular permeability and loss of capillary perfusion. In terms of PDR, as VEGF levels increase in response to hypoxia, pigment epithelial derived factor (i.e. anti-angiogenic) expression is thought to decrease, creating a pro-angiogenic environment.

From a clinical perspective, diabetic retinopathy can be broadly categorized as not present, nonproliferative diabetic retinopathy (NPDR), proliferative diabetic retinopathy (PDR), and / or clinically significant diabetic macular edema (CSDME). NPDR is subdivided into mild-to-moderate, moderate-to-severe, severe and very severe.

## **Pregnant Women with Pre-Existing Diabetes**

In women with pre-existing diabetes, there is a risk of rapid progression of retinopathy. However, this is usually a transient progression and the long-term risk of progression of retinopathy does not appear to be increased by pregnancy [44]; this should be stressed to pregnant female patients with diabetes. Factors that increase the risk for the transient progression of retinopathy during pregnancy include initial retinopathy severity, level of glycemic control (as indicated by glycosylated haemoglobin levels, A1c), duration of

diabetes and concomitant hypertension. In pregnant women with mild-to-moderate non-proliferative diabetic retinopathy (NPDR) this condition is rarely affected permanently unless there is chronic or pregnancy induced hypertension. [45, 46] The mechanism underlying the transient progression of diabetic retinopathy during pregnancy is thought to be related to progesterone induced increased secretion of vascular endothelial growth factor and altered retinal blood flow. If there is a progression of retinopathy to severe NPDR or CSDME, the optometrist should arrange for an urgent consult to a retinal specialist. From a preventative perspective, retinopathy status needs to be monitored carefully during pregnancy, at least every trimester. Women with more severe levels of retinopathy contemplating pregnancy should be considered to undergo pan-retinal photocoagulation prior to conception; approximately 30% of women with moderate NPDR will develop proliferative diabetic retinopathy during pregnancy, as opposed to only 6% in those with mild NPDR [47].

## **Clinical Manifestations of NPDR**

**Mild-to-moderate NPDR** is characterized by the presence of microaneurysms, intraretinal hemorrhages, hard exudates, diabetic macular edema (DME) and foveal avascular zone (FAZ) abnormalities.

Microaneurysms represent either local weakening and bulging of capillary wall (following pericyte loss) or a proliferative cellular response to focal hypoxia. Microaneurysms are derived from retinal capillaries and represent a relatively early sign of diabetic retinopathy. Microaneurysms range in size from 12 to 100 microns, although those less than 30 microns are not visible ophthalmoscopically. Unlike hemorrhages, microaneurysms are round, with smooth margins and exhibit a distinct central light reflex. Occlusion and hyalinization of the microaneurysm will eventually occur.

Intraretinal hemorrhages represent rupture of a microaneurysm, capillary or venule. They adopt a shape that reflects their depth within the retina; hemorrhages in the deeper retinal layers (outer plexiform / inner nuclear) appear as dot (i.e. distinct borders) and blot (i.e. indistinct borders) hemorrhages and if they occur in the superficial retinal nerve fiber layer they appear as flame-shaped hemorrhages. Dot hemorrhages can be distinguished from microaneurysms by fluorescein angiography, although this is not a clinical indication for angiography. Roth's spots present as white centered hemorrhages which are thought to represent auto-occlusion if the source was a microaneurysm or the accumulation of platelets or fibrin. Hemorrhages generally resolve within 3 to 4 months & only affect vision if located at the fovea.

Hard exudates present clinically with a glistening yellow or waxy appearance, are often located within or near the macula and are strongly associated with retinal edema. Hard exudates often demarcate an edematous area of retina and can be centered on a leaking microaneurysm in which case they often form a circinate ring. Hard exudates represent serum lipoproteins that have leaked from abnormally permeable capillaries and are generally reabsorbed (spontaneously or following laser treatment) as a result of mobilization of macrophages.

DME is clinically defined as retinal thickening noted on stereo fundus biomicroscopy and represents the abnormal accumulation of intra-retinal fluid due to breakdown of the blood-retinal barrier. In reality, DME presents a spectrum of signs encompassed by the term diabetic maculopathy, namely edema, hard exudates and ischemic changes. Not surprisingly, the presence of DME is strongly associated with hard exudates and microaneurysms. DME can occur relatively early in the development of diabetic retinopathy.

FAZ abnormalities appear as nonperfused inner retinal capillaries surrounding the fovea, visible using fluorescein angiography. In the physiological situation, fluorescein filled capillaries surround but do not encroach upon the fovea which appears dark; this creates a FAZ of 350 to 1000µm in diameter. FAZ abnormalities associated with diabetic retinopathy include irregular margins, capillary budding into the FAZ, widening of inter-capillary spaces surrounding the FAZ (i.e. capillary closure), bridge vessel formation, increase of the FAZ diameter and dilation of existing capillaries. Ultimately, loss of perfusion to the capillary network surrounding the FAZ will result in vision loss. For this reason, unexplained loss of vision in a patient with diabetes may indicate ischemic diabetic maculopathy which requires prompt referral to a retinal specialist indicating the need for fluorescein angiography.

**Moderate-to-severe NPDR** is characterized by the presence of cotton wool spots, venous beading and intraretinal microvascular abnormalities (IRMA). These retinal signs indicate the later morphological changes associated with diabetic retinopathy and increase in the extent of vascular closure and leakage. Not surprisingly, arteriolar closure rather than capillary closure is thought to cause more severe ischemia & particularly results in cotton wool spots, venous beading and increase in the number of intraretinal hemorrhages.

Cotton wool spots, sometimes referred to as soft exudates, appear clinically as fluffy, yellow-white, striated lesions with feathery borders. They represent localized infarcts (i.e. loss of blood supply) of the nerve fiber layer and result in obstructed axoplasmic flow in ganglion cell axons and swelling of nerve fibers. The ischemic insult will generate a nerve fiber layer defect since the inner retinal nerve fibers are atrophied. Cotton wool spots are often accompanied by dark-blot hemorrhages which are thought to represent partial arteriolar occlusion, or complete occlusion followed by reperfusion. As a result, cotton wool spots and dark-blot hemorrhages are generally indicative of areas of capillary non-perfusion but a few cotton wool spots can occur early in diabetic retinopathy or can occur transiently following initiation of “tight” glycemic control

Venous beading presents clinically as focal narrowing or focal dilation of the retinal venules. The appearance of venous beading is generated by thickening and hyaline degeneration of venular wall. Other venous abnormalities include “loop” formation, re-duplication of segments, sheathing and focal narrowing. Venous beading occurs adjacent to areas of capillary non-perfusion and is often associated with cotton wool spots.

Intraretinal microvascular abnormalities (IRMA) present clinically as irregular, segmented dilatations of tortuous capillary channels between arterioles and venules. IRMA occur adjacent to areas of capillary non-perfusion and may represent intra-retinal neovascularization or dilation of pre-existing capillaries.

Venous beading, IRMA and intraretinal hemorrhages, especially dark-blot hemorrhages with indistinct borders, are strong indicators of retinal ischemia and predict progression to PDR [48]. Cotton wool spots (soft exudates) are thought to represent ischemic changes but the ETDRS found that they did not reliably predict the progression of DR.

### **Causes of Sight-Loss in Diabetic Retinopathy: Clinically Significant Diabetic Macular Edema**

The term clinically significant diabetic macular edema (CSDME) implies that the fovea, and therefore visual acuity, is threatened. CSDME remains the most common cause of decreased vision and blindness among those with diabetes and it can occur at any stage in the development of diabetic retinopathy. The Early Treatment of Diabetic Retinopathy Study [49] defined CSDME based upon clinical stereo fundus biomicroscopy assessment as either: Any retinal thickening within 500 microns of the center of the macula, or; retinal thickening at least 1 disc area in size, any part of which is within 1 disc diameter of the center of the macula, or; hard exudates within 500 microns of the center of the macula with adjacent retinal thickening. Macular edema can be either focal or diffuse. Focal macular edema results from leakage of microaneurysms or IRMA and appears clinically as localized areas of retinal thickening often surrounded by a lipid ring. Diffuse macular edema represents a widespread thickening of the retina resulting from generalized leakage of capillaries at the posterior pole, possibly from beneath the retinal pigment epithelium. Diffuse macular edema is associated with minimal lipid exudates. The results of controlled treatment trials, particularly that of the ETDRS, have established a role for grid laser photocoagulation therapy (in combination with focal laser when appropriate) for clinically significant DME. The ETDRS showed that there was a 50% reduction in the rate of vision loss with laser photocoagulation in the areas of leakage. [49] More recently, use of intravitreal anti-VEGF and steroid based medications may be tried, especially in cases of refractory DME. When the optometrist sees signs of CSDME, an urgent referral to a retinal specialist is indicated.

### **Causes of Sight-Loss in Diabetic Retinopathy: Proliferative Diabetic Retinopathy**

By definition, PDR is seen clinically as the growth of new blood vessels that arise from the retina or optic disc and proliferate along the inner retinal surface (sub-hyaloid space) and into the cortical vitreous, with or without a fibrous component. Typically, neovascularization occurs within 45° of optic disc & especially at the optic disc itself. New vessels within one disc diameter of the optic nerve head are termed neovascularisation of the disc (NVD), while those that arise elsewhere on the retina are termed neovascularisation elsewhere (NVE). Proliferation into the vitreous can lead to subhyaloid, preretinal, or vitreous hemorrhages and to tractional retinal detachment. An

urgent consult with a retinal specialist is indicated as scatter or panretinal photocoagulation of PDR can reduce the risk of severe vision loss by 95%, if treated early. More recently, the use of intravitreal anti-VEGF medications, such as Avastin and Lucentis, has been found clinically to regress the development of PDR.

## **Stages of NPDR and Referral Indications**

NPDR can be staged as not present, mild-to-moderate, moderate-to-severe, severe or very severe. Referral criteria for diabetic retinopathy vary from area to area within Canada but the following represents a reasonable representative guide.

Mild-to-moderate NPDR is characterized by the presence of microaneurysms, intraretinal hemorrhages, hard exudates, diabetic macular edema (DME) and foveal avascular zone (FAZ) abnormalities. Generally, people with mild-to-moderate NPDR can be safely monitored annually. Mild-to-moderate NPDR is often considered to be relatively benign but this can be misleading since clinically significant DME (CSDME) can occur at any stage in the development of retinopathy. From the perspective of an optometrist, CSDME requires an urgent referral to a retinal specialist.

Moderate-to-severe NPDR is characterized by the presence of cotton wool spots, venous beading and intraretinal microvascular abnormalities (IRMA), plus the signs of mild-to-moderate NPDR. People with moderate-to-severe NPDR need to be examined by their optometrist every 6 months depending on the severity of the presentation and the presence of other co-morbidities. Moderate-to-severe NPDR indicates the presence of increased retinal ischemia and should be managed cautiously.

Severe and very severe NPDR is characterized by an abundance of IRMA, cotton wool spots, venous calibre changes especially venous beading. Severe PDR is indicated by any one of the following (“4-2-1 rule”): severe intraretinal hemorrhages in *four* quadrants; venous beading in *two* quadrants; moderately severe IRMA in *one* quadrant. Very severe PDR is indicated by any two of the above criteria. Optometrists should arrange for a prompt consult with a retinal specialist as severe and very severe NPDR signal the probability of imminent PDR and often warrant prophylactic laser surgery.

## Recommendations and Conclusions

Optometrists as primary eye care providers need to take an active role in the management, care and treatment of patients with diabetes. Quite often, these patients are under our care for other eye conditions and often existing patients develop diabetes and need good advice on how to manage any possible complications, especially ocular complications. Optometrists should question patients with diabetes about their glycosylated haemoglobin levels (A1c), blood pressure and cholesterol. The Canadian Diabetes Association recommends people with diabetes should strive to attain A1c levels of 7.0% or below (but recognise this is not always feasible especially for those people susceptible to hypoglycaemic episodes), a blood pressure of 130/80 mmHg or below and a cholesterol level of 2.0 mmol/L or lower for low density lipoproteins and a total cholesterol to high density lipoprotein ratio of below 4.0. As optometrists, we should advise our patients with diabetes on beneficial lifestyle changes such as regular exercise and cessation of smoking. It is important that we communicate our findings to our patients and also to other members of the Diabetic Healthcare team to ensure that they are managed properly and efficiently and referred for care that may be beyond our professional expertise. Based on the 2003 Canadian Diabetes Association Clinical Practice Guidelines, we make the following *additional* recommendations for frequency of examination:

### **For patients with Type 1 diabetes:**

Examination should be performed at the time of diagnosis and then should be performed annually 5 years after the onset of diabetes in individuals 15 years of age or older.

### **For patients with Type 1 diabetes who are planning pregnancy:**

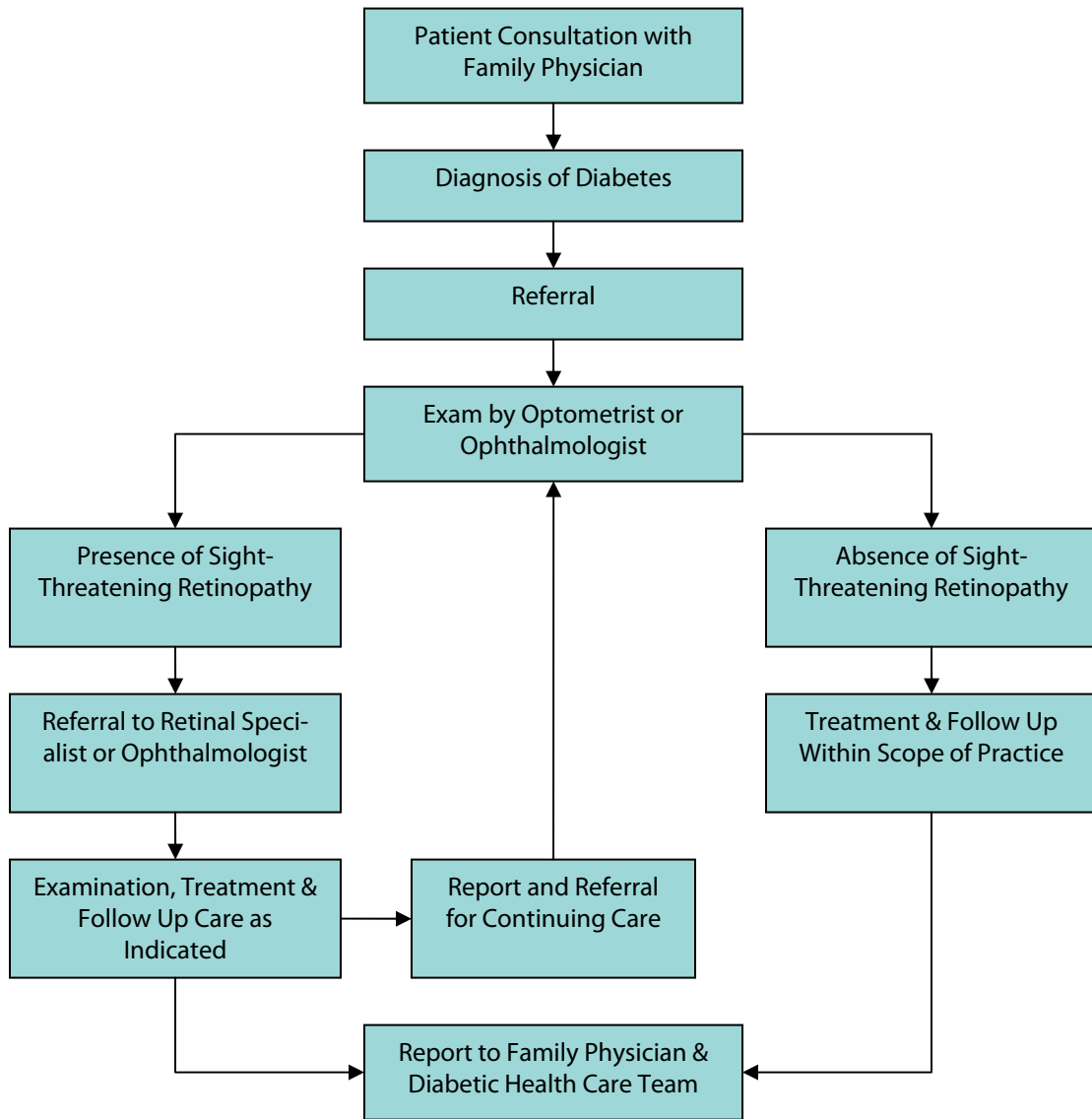
Examination should be performed prior to pregnancy, during the 1<sup>st</sup> and each following trimester, as needed throughout pregnancy and within the 1<sup>st</sup> year post partum.

### **For patients with Type 2 diabetes:**

Examination should be performed at the time of diagnosis. The interval of follow up assessments should be tailored to the severity of the retinopathy. In those with no or minimal retinopathy, the recommended interval is 1 to 2 years.

The following Flow chart should be used by optometrists to manage patients with Diabetes and inform other members of the Diabetic Healthcare Team (DHC Team) of their findings.

## Ocular Management of the Patient with Diabetes



Note : **Diabetic Health Care Team** (DHC Team) refers to the core team which includes the Physician (family physician and/or specialist) and the Diabetic Educators (Nurse and Dietician).

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