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Module 12 Part 1

COURSE CODE: C-10413/0

The Time bomb of Diabetes



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This article highlights the need for appropriate management of diabetic patients, in order to improve both their ophthalmic and their systemic health. The microvascular and macrovascular complications of diabetes are discussed as well as some of the newer treatments. As the incidence of diabetes is rising, it is important that an optometrist has a good working knowledge of this systemic disease. It is important that there is good communication between the optometrist and the patient's GP as the management of diabetes calls for a multidisciplinary approach.

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Diabetes is a chronic disease that occurs when the pancreas does not produce enough insulin. It may also occur when the body cannot effectively use the insulin it produces. Insulin is an essential hormone, responsible for the regulation of blood sugar. Insulin is synthesised and released by the beta cells in the islets of Langerhans of the pancreas. Insulin acts to reduce the concentration of circulating glucose. Diabetics are susceptible to the long-term complications of hyperglycaemia due to insufficient insulin. Insulin lowers blood glucose levels by suppressing hepatic glucose production, stimulating glucose uptake by skeletal muscle and fat, stimulating lipogenesis and inhibiting lipolysis in adipose tissue.

The number of patients with diabetes in the UK is steadily rising. In the UK, diabetes affects approximately 2.3 million people, and it is thought there are at least half a million more people who have the condition but are not aware of it. The World Health Organization (WHO) estimates that more than 180 million people worldwide currently have diabetes.



➔ **Figure 1**
Small atrophic pancreas

This number is likely to more than double by 2030.

In 2005, an estimated 1.1 million people died from diabetes. Almost 80% of diabetes deaths occur in low and middle-income countries. Half of diabetes deaths occur in people under the age of 70 years. This death rate is higher in diabetic females compared to diabetic males. The WHO projects that deaths secondary to diabetes will increase by more than 50% in the next 10 years. Most notably diabetes deaths are projected to increase by over 80% in upper-middle income countries between 2006 and 2015.

Classification of diabetes

Diabetes may be classified into Type 1 or Type 2 diabetes. Type 1 diabetes accounts for approximately 10% of all cases of diabetes. Type 1 diabetes (previously known as insulin-dependent or childhood-onset) is characterised by a lack of insulin production. The onset of Type 1 diabetes is generally before the age of forty and occurs predominantly in the teenage years. Type 1 diabetes occurs due to a T cell mediated autoimmune attack against the insulin producing beta cells in the islets of Langerhans in the pancreas¹. Both CD4 and CD8 T cells are

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involved in this process. Postulated candidate autoantigens include insulin, glutamic acid decarboxylase and protein tyrosine phosphatase.

Both genetic and environmental risk factors have been postulated to cause diabetes. Various viruses such as Coxsackie, Rubella, Cytomegalovirus and Epstein Barr Virus have been implicated. These viruses may be directly toxic to the beta cell or cross react acting as beta cell autoantigens. Dietary factors have been suggested to increase the risk of developing diabetes. Breastfed infants have a lower risk for Type 1 diabetes compared to babies weaned on cow milk. Some cow's milk proteins, eg bovine serum albumin, have antigenic similarities to a beta islet cell antigen.

There is a familial risk associated with diabetes. There is a 60% lifetime concordance for developing Type 1 diabetes in monozygotic twins. Only 30% of monozygotic twins develop diabetes within 10 years after their twin has been diagnosed. Dizygotic twins have an 8% risk of concordance, which is similar to the risk among other siblings. The frequency of diabetes developing in children with a diabetic mother is 2-3% and 5-6% if the father has Type 1 diabetes. The risk to children rises to almost 30% if both parents are diabetic. HLA class II molecules DR3 and DR4 are strongly associated with Type 1 diabetes. More than 90% of Caucasian patients with Type 1 diabetes express one or both of these molecules, compared to 50-60% in the general population. Patients expressing DR3 are at a higher risk of developing other autoimmune endocrinopathies as well as coeliac disease. Patients expressing DR4 are usually younger at diagnosis and more likely to have positive insulin antibodies, yet they are unlikely to have other autoimmune endocrinopathies. If patients express both DR3 and DR4 they run a very high risk of developing diabetes.

Diabetes occurs as a consequence of pancreatic damage (figure 1). Pancreatitis is a complication of alcohol abuse. The pancreas can also become damaged in patients with cystic fibrosis or haemochromatosis. Thiazide diuretics have been reported to be toxic to the islet cells. Corticosteroids elevate

blood glucose and can induce an iatrogenic diabetic state.

With Prader-Willi, Down's, Progeria, Klinefelter and Turner's syndromes hyperglycaemia is a recognised complication. If this hyperglycaemia is prolonged, this may result in the development of permanent diabetes. Patients with myotonic dystrophy and Friedreich's ataxia also have a tendency to develop diabetes mellitus. Wolfram's syndrome or DIDMOAD comprises Diabetes Insipidus Diabetes Mellitus Optic Atrophy and Deafness. It has an autosomal dominance mode of inheritance. Diabetes insipidus is also characterised by excretion of large amounts of severely diluted urine. Diabetes insipidus is caused by a deficiency of antidiuretic hormone and is unrelated to diabetes mellitus.

The symptoms of Type 1 diabetes may occur suddenly. Symptoms include thirst, excessive excretion of urine, constant hunger, weight loss, recurrent infections, and fluctuations in vision as well as fatigue. When the blood glucose concentration is pathologically elevated there is loss of glucose in the urine. The sugary urine draws out water from the body by osmosis and the diabetic patient

becomes very dehydrated. Diabetic ketoacidosis is a life threatening complication of diabetes. On presentation with this clinical condition, the patient has a smell of acetone on their breath. The ketotic patient may be very confused and even appear drunk. Without prompt treatment with insulin, the condition can quickly progress towards coma or death.

The diagnosis of Type 1 diabetes can be made by measuring a random blood glucose (15.0mM), a fasting venous plasma glucose (7.0mM) or a plasma glucose (11.1mM) 2 hours following an oral glucose challenge.

Type 2 diabetes (formerly called non-insulin-dependent or adult-onset) results from the body's ineffective use of insulin. There is impaired insulin secretion by beta cells combined with insulin resistance in muscles and liver. Circulating insulin levels may be within the normal range but there is insulin resistance in the target tissues.

Type 2 diabetes comprises 90% of people with diabetes around the world. It affects 1-2% of the total UK Caucasian population. It is five-times more common than Type 1 diabetes. It is more common among Asian (11%) and



➔ **Figure 2**
Diabetic nephropathy

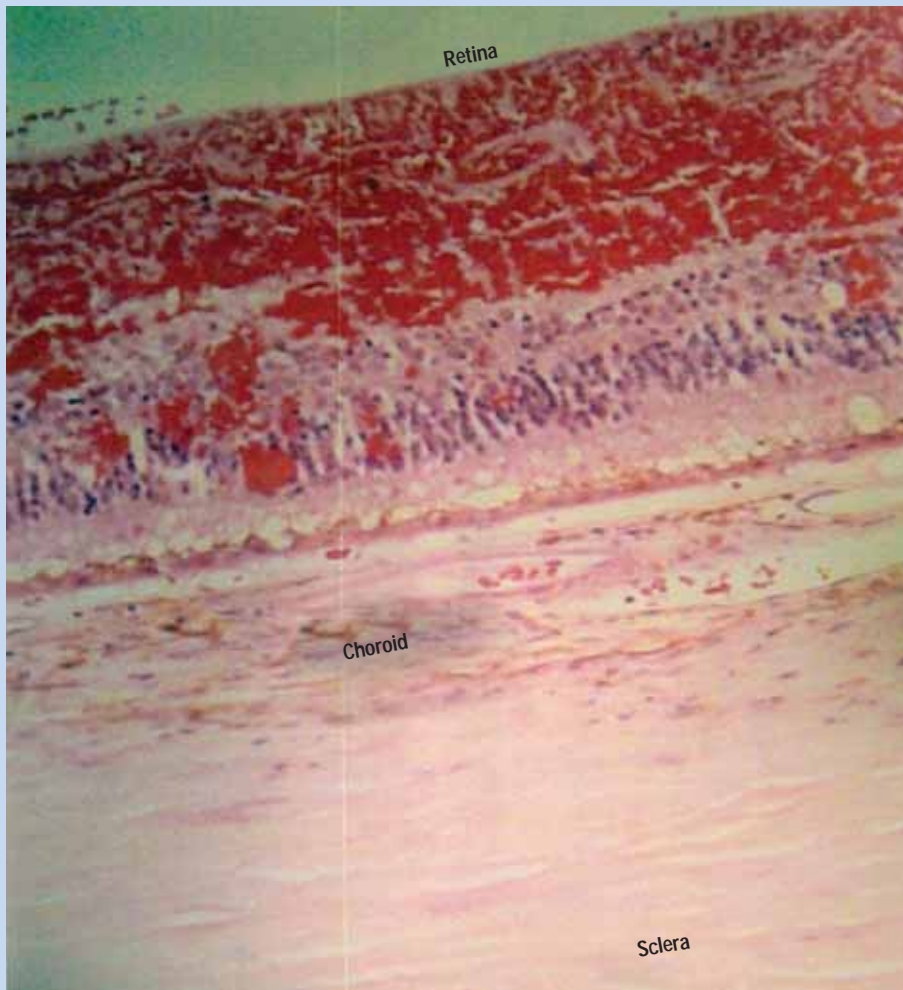


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➔ Figure 3

Intra and preretinal haemorrhages seen as a consequence of proliferative diabetic eye disease

African-Caribbean (9%) populations. In the over-70 age group, Type 2 diabetes affects 5-7% of people.

Type 2 diabetes has been reported to occur in obese children. There is an established autosomal dominant inheritance pattern seen in the condition of MODY - Maturity Onset Diabetes of the Young.

Type 2 diabetes is insidious in onset in that patients are unaware of having the condition and may have advanced complications at presentation. It is therefore useful to screen for Type 2 diabetes as part of health checks. There is a strong association between Type 2 diabetes and obesity. Up to 70% of diabetics are obese and there is a high correlation with increased abdominal fat and girth. Excessive energy intake and insufficient exercise potentiates the

development of this condition. This is not a new concept. As early as 6BC, Sushruta identified diabetes and classified it as "Medhumeha". He advised that exercises would cure the condition. There is an established connection with malnutrition both in utero and also in early life, and the subsequent development of adult diabetes.

Up to 40% of Type 2 diabetics have a positive family history. There is a 60-90% concordance observed in identical twins. If a first degree relative is affected there is a five-fold increased lifetime risk of developing diabetes. TCF7L2 is a genetic defect associated with an increased risk of developing Type 2 diabetes.

Maternally inherited diabetes is linked with a mutation in RNA.

Maternally Inherited Diabetes and Deafness (MIDD) accounts for less than 3% of diabetic patients. Patients with MIDD have a characteristic fundal dystrophy.

Hyperglycaemia which is first recognised during pregnancy is termed gestational diabetes. Like Type 2 diabetes it is identified through routine antenatal screening rather than presenting with symptoms. Untreated gestational diabetes can damage the health of both the foetus and the mother. Mothers with gestational diabetes have a higher risk of delivering babies with a high birth weight, congenital cardiac and central nervous system abnormalities and skeletal muscle malformations. Patients with gestational diabetes have an increased risk of developing Type 2 diabetes in later life.

Patients with an Impaired Glucose Tolerance are also at higher risk of progressing to Type 2 diabetes. The passage from a prediabetic state to Type 2 diabetes is characterised by three steps. The first is a reduction in the beta cells of the pancreas and of compensatory insulin secretion. The second step occurs as there is an overproduction of glucose by the liver, as a result of oversecretion of glucagon. Finally, there is an increase of resistance to insulin in the skeletal muscles.

Systemic complications of diabetes (Type 1 and 2)

As a consequence of prolonged hyperglycaemia, patients can develop both microvascular and macrovascular complications of diabetes. This occurs as a combination of the accumulation of sorbitol, abnormal growth of vascular smooth muscle and the glycation of proteins. Basement membranes grow both thicker and weaker. In addition, there is a reduction in the levels of circulating nitric oxide in diabetic patients. Nitric oxide is important in maintaining good endothelial cell function and reduces atherogenesis. Platelet function is also abnormal in diabetes. There is a propensity for platelet aggregation and activation and this is coupled with a tendency towards coagulation. Microvascular complications cause neural renal,



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lenticular and retinal dysfunction. Impairment of the microvascular blood supply results in chronic end organ damage.

Diabetic neuropathy affects up to 50% of people with diabetes. Symptoms secondary to sensory nerve damage include early pain followed by paraesthesiae and numbness in the extremities of the hands and feet. Motor neural damage results in distal limb weakness, termed diabetic amyotrophy. The autonomic nerves may also be affected as a consequence of hyperglycaemia. Autonomic failure can result in bladder dysfunction and impotence.

Gastrointestinal complications following autonomic failure include diabetic gastroparesis, vomiting and severe diarrhoea. Orthostatic hypotension is a complication encountered in diabetic patients with autonomic dysfunction.

Single nerve damage or mononeuropathies may be observed in diabetic patients. Diabetic patients presenting to an optometrist with diplopia may have a mononeuropathy of the oculomotor (3rd), trochlear (4th) or abducens (6th) cranial nerve. The third nerve palsy or "medical 3rd nerve palsy" secondary to diabetes can be differentiated from a "surgical 3rd nerve palsy" in that there is no pupillary involvement. This is because the parasympathetic nerve which supplies the pupil has a superficial course with the 3rd nerve. This parasympathetic nerve is vulnerable to external compression from tumours but its location ensures it has a good blood supply. Its superficial position also allows it to draw on nutrients from the surrounding cerebrospinal fluid. Pain is not a discriminating factor between a medical and a surgical 3rd nerve palsy. An acute mononeuropathy may be very painful. It is important that patients are fully medically investigated when presenting with an isolated nerve palsy. Giant cell arteritis may present with a mononeuropathy. Signs of aberrant regeneration indicate a more sinister underlying cause. In general, most patients with a diabetic cranial nerve palsy make a good recovery within six months. Further investigations are required if there is evidence of

progression or if the palsy does not resolve.

The diabetic foot is at risk of injury because of its reduced sensation and blood supply. Ulcers may occur secondary to pressure necrosis. These ulcers may quickly progress into cellulitis or osteomyelitis. Diabetic patients are more likely to develop intermittent claudication and critical limb ischaemia. Excluding trauma, diabetic distal amputations account for the majority of amputations of the lower limb.

Diabetes is among the leading causes of kidney failure. In total 10-20% of people with diabetes die secondary to nephropathy (figure 2). Features of kidney disease include a decrease in glomerular filtration rate, an increase in glomerular capillary pressure, proteinuria and eventually progression to end-stage renal failure.

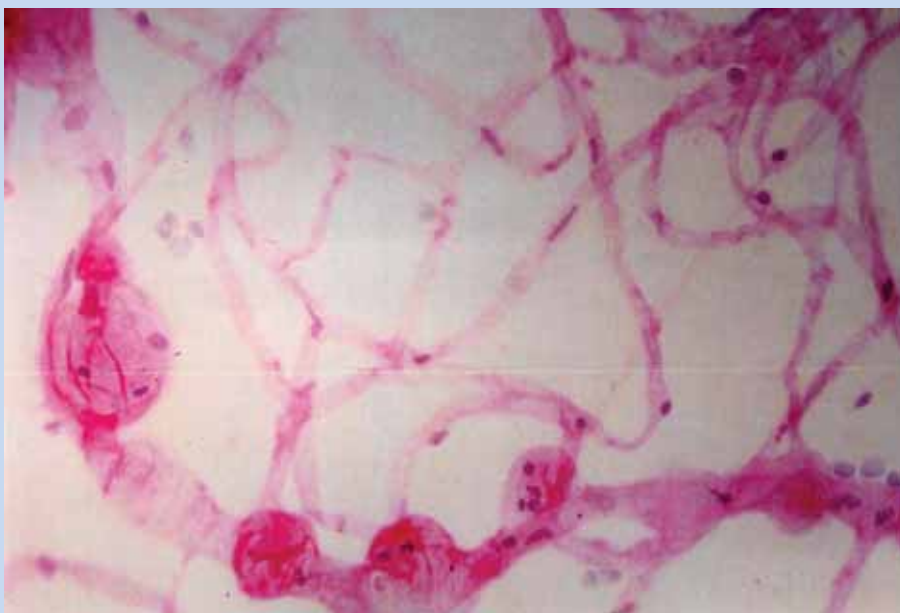
Because chronically elevated blood pressure contributes to the decline in renal function, hypertensive patients with diabetes must be referred for long-term management of their blood pressure. Potentially nephrotoxic drugs including contrast dye should be avoided whenever possible in diabetic patients. Renally excreted or potentially nephrotoxic drugs should be given at reduced dosage as appropriate, titrated to the patient's serum creatinine level.

There is an increased risk of systemic infections in diabetic patients. Mucormycosis is a fungal disease which may be fatal and is peculiar to the diabetic patient. Malignant otitis externa is almost exclusive to diabetic patients. Diabetic patients have an increased risk of reactivation of tuberculosis and of staphylococcal infections.

Macrovascular disease is the leading cause of death in patients with diabetes, causing 75% of diabetic deaths. Diabetes markedly increases the risk of coronary, cerebral and peripheral atherosclerosis and the clinical consequences of myocardial infarction, stroke, limb ischaemia and death.

Diabetes doubles the risk of myocardial infarction in male patients and quadruples it in female patients. The risk of a myocardial infarction in patients with diabetes and no evidence of coronary heart disease matches that of patients without diabetes who have had a previous myocardial infarction. Approximately 50% of diabetic patients die five years after a myocardial infarction, double the rate observed in nondiabetic patients. In patients with known coronary artery disease and diabetes, the rates of death approach 45% over seven years and 75% over 10 years.

The risk of stroke in patients with



➔ **Figure 4**

Abnormal aneurysmal outpouchings in diabetic vasculature



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diabetes is significantly increased. Stroke risk increases by almost three fold among men taking hypoglycaemic medication. Diabetes also increases stroke related mortality, doubles the rate of recurrent stroke and trebles the frequency of stroke-related dementia.

Atherosclerosis tends to develop earlier and be more marked in diabetic patients. The effect of diabetes on atherosclerosis is so pronounced that the benefit of female gender is eliminated in female patients with diabetes. Atherosclerosis normally has a lower prevalence in women compared to men but in diabetes there is an equal risk of atherosclerosis in both genders. Because of their high atherosclerotic risk, diabetic patients with lipid abnormalities must be treated aggressively to lower the risk of serious atherosclerosis. It has been suggested that statin therapy should be started in all patients with Type 2 diabetes, regardless of their lipid levels, to lower their risk of cardiovascular disease. Retrospective analyses of the Scandinavian Simvastatin Survival Survey and the Cholesterol and Recurrent Events (CARE) trial have demonstrated that statin therapy reduced the risk of cardiovascular events in diabetic patients with

coronary artery disease and elevated LDL cholesterol by 55% and 24% respectively. In addition to increasing the risk of cardiovascular events, atherosclerosis is also a risk for cerebrovascular accidents.

Hypertension, which also increases the risk of atherosclerosis, is twice as common in patients with Type 2 diabetes as in persons without diabetes. Angiotensin-converting enzyme (ACE) inhibitors reduce the risk of progressive nephropathy.

Ocular complications of diabetes

Diabetic retinopathy remains the leading cause of blindness in people of working age². In total 8% of those registered legally blind (vision <3/60 in the better eye) have diabetes. Diabetic patients are twenty-nine times more likely to be blind than non-diabetics of a similar age³. The longer the patients have diabetes, the higher the prevalence of diabetic retinopathy. A total of 20% of Type 2 diabetics have retinopathy at presentation. It is unusual to detect retinopathy for the first five years after a Type 1 diabetic has been diagnosed. Pregnancy may cause acceleration to proliferative retinopathy (figure 3). Diabetic macular oedema is a leading

cause of legal blindness in patients with Type 2 diabetes.

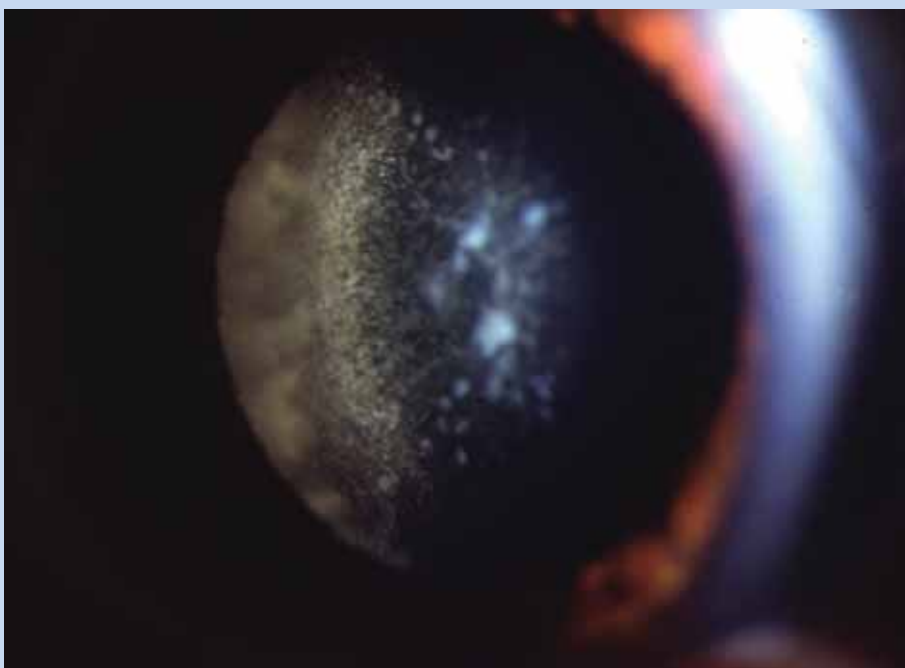
The vascular disruptions of diabetic retinopathy are characterised by abnormal vascular flow, disruptions in permeability and capillary non-perfusion. It is the endothelial cell which is responsible for maintaining the blood-retinal barrier and damage to the endothelial cell in diabetes results in the accumulation of extracellular fluid at the macula.

The regulation of retinal capillary perfusion is by pericytes. Loss of retinal pericytes correlates with the formation of microaneurysms (figure 4). Damage to the pericytes as well as thickening of the capillary basement membrane in diabetes leads to altered retinal haemodynamics, including abnormal autoregulation of retinal blood flow.

White blood cells play a role in the pathogenesis of diabetic retinopathy. It is thought that these cells are less deformable in diabetics and may be involved in capillary non-perfusion and dropout. They may be activated in the diabetic patient and release their toxic superoxide radicals, damaging the vascular endothelium.

As a result of microangiographic occlusions, the retina becomes ischaemic. It then releases factors such as VEGF – Vascular Endothelial Growth Factor. VEGF promotes neovascularisation within the retina. Neovascular fronds may bleed and fibrose with consequent retinal detachment. In addition, the retinal ischaemia drives the development of rubeosis iridis. Argon laser pan retinal photocoagulation has been the traditional treatment modality used to treat proliferative diabetic retinopathy. Anti-VEGF drugs given intravitreally reduce the neovascular drive but must be given repeatedly for the effect to be sustained. Retinal ischaemia and consequent neovascularisation is a feature of both Type 1 and Type 2 diabetes.

Diabetics are more prone to the development of chalazia. When there is an associated hyperlipidaemia, xanthelasmata may occur. The microvascular neuropathy associated with diabetes can affect the corneal



➔ **Figure 5**
Snowflake cataract



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nerves and result in reduced corneal sensation. This predisposes patients to developing corneal ulcers as the early warning symptoms of epithelial erosion are lost. When this risk is combined with reduced wound healing, it is clear that diabetics must be vigilant in their contact lens hygiene.

The intraocular pressures recorded in diabetics are higher than the normal population. The Blue Mountains study showed diabetics to be at a higher risk of primary open angle glaucoma. It has been shown that visual field loss occurs in diabetic patients with glaucoma at lower pressures than glaucoma patients who do not have diabetes. Many patients with glaucoma are managed with topical B blocker medication. These drops can mask the symptoms of hypoglycaemia.

The lens in diabetic patients is larger than that observed in the general population. This predisposes patients towards the development of narrow-angle glaucoma. It has been demonstrated that a significant proportion of patients with narrow-angle glaucoma also have Type 2 diabetes.

The iris of diabetic patients may demonstrate a moth-eaten appearance as a consequence of chronic pigment fallout. These patients will exhibit speckled transillumination. Diabetic patients tend to dilate less well with mydriatic agents than their non-diabetic counterparts.

If a patient has persistent hyperglycaemia, this will lead to an increase in the sorbitol concentration within the lens. This causes the lens to imbibe water and swell, the consequence being an acute myopic shift. In patients who have unstable diabetes it is inappropriate to prescribe new spectacles until their sugars have normalised.

There is a three-fold increased prevalence of cataract in diabetics under the age of 65. This risk is increased by duration of disease and poor blood sugar control. A snowflake cataract is particular to Type 1 diabetes (figure 5). This cataract develops rapidly and if the blood sugar is quickly normalised it may spontaneously resolve.

Acute optic disc swelling in young diabetics is termed diabetic papillopathy and generally resolves without sequelae. In elderly diabetic patients, there may be swelling of the optic disc secondary to non-arteritic ischaemic optic neuropathy. This is typically associated with hemifield defect. Acute optic disc swelling in diabetic patients may also be secondary to a central retinal vein occlusion.



➔ **Figure 6**
Insulin syringe

Treatment of diabetes

The Diabetes Control and Complications Trial (DCCT), showed that intensive therapy was employed to maintain normal blood glucose levels, there was a great reduction in the development and progression of retinopathy, microalbuminuria, and neuropathy over seven years. In the United Kingdom Prospective Disease Study (UKPDS), more than 5,000 patients with Type 2 diabetes were followed up for up to 15 years. Those in the intensely treated group had a significantly lower rate of progression of microvascular complications than that of those receiving standard care. These important trials show how vital it is that diabetic patients have good glycaemic control. Hypertension and hyperlipidaemia should be aggressively treated in diabetic patients in order to reduce the risk of a cerebrovascular accident. Antiplatelet therapies such as aspirin or clopidogrel are prescribed to patients with diabetes and atherosclerosis unless contraindicated. Hyperlipidaemia occurs in both Type 1 and Type 2 diabetes.

Treatment of Type 1 diabetes is by

supplying the deficient insulin. Because insulin is a peptide, it is generally administered by injection as it is degraded when taken orally. The first patient to be treated with insulin injections was in 1922 and the inventor, Banting, received the Nobel Prize in Medicine as a consequence of this important intervention (figure 6). Insulin pumps regularise the delivery of insulin to patients. In India, some patients use an aerosol sprayed into the mouth which is then absorbed into the bloodstream via the buccal mucosa.

An appropriate diet, weight reduction and increased physical activity are the cornerstones of the treatment of Type 2 diabetics. However, antidiabetic drugs are often also required. None of the pharmacological treatments correct all the anomalies involved in the pathogenesis of Type 2 diabetes. Treatments which may initially be quite successful tend to fail after some years of therapy. Combination treatments and insulin in order to regulate the blood glucose are often required.

The sulfonylureas are the oldest class of diabetic drug but are no longer used as a primary agent. They are of use in the later stages of the disease. Sulfonylureas potentiate the physiological effect of glucose and are able to stimulate insulin secretion at low glucose concentrations. Failure of these drugs occurs when there is a reduction in beta cell mass and function. The major adverse effect of sulfonylureas is hypoglycaemia.

Metformin was shown to have a beneficial effect in the UKPDS on reducing the number of associated cardiovascular events. Metformin is generally recommended as a first line drug therapy in the majority of Type 2 diabetic patients. The major effect of metformin is the reduction of hepatic glucose production and to a lesser extent extends improvement of peripheral insulin sensitivity. A metallic taste and digestive side effects such as anorexia and diarrhoea are the most commonly reported adverse events. These lead to discontinuation of Metformin in 5% of patients. Lactic acidosis, although rare is the most severe side effect of this drug.

The insulin sensitising agents



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 ➔ **Figure 7**

Amputation secondary to diabetes

thiazolidinediones act mainly at a peripheral level. The most frequent side effects observed are related to fluid retention which can lead to heart failure in the case of preexisting cardiac disease. Several cases of macular oedema have been reported during treatment with this class of drug and seem to correlate with fluid retention and weight gain.

Dipeptidyl peptidase IV inhibitors (DPP IV) induce insulin secretion in a glucose dependent manner. They do not demonstrate any hypoglycaemic effects as their action on the beta cell ends when blood glucose normalises. They are orally bioavailable. Future developments in the treatment of diabetes would include pancreatic beta cell transplants and stem cell therapy.

Hypoglycaemia is an acute complication of diabetes treatments. Symptoms include increased agitation and sweating. The patient may lose consciousness and collapse. Seizures with consequent brain damage and death may then occur. It is important to treat any diabetic patient suspected of suffering a hypoglycaemic attack immediately by raising their blood sugar. In most cases a sugary drink will revive the patient. This sugary drink will only temporarily raise the blood glucose and so patients should be given some food in order for blood glucose to be maintained. In cases of severe hypoglycaemia intravenous fluids or an injection of glucagon is required to elevate the blood sugar concentration.

Conclusion

The total cost to the NHS of diabetes mellitus and its complications (figure 7) has been estimated to be £9bn, totalling almost 10% of the total yearly NHS budget. Diabetic patients have a higher rate of both hospital admission and length of stay. Prevention is always better than cure and it is vital that diabetic patients are well educated about their disease and its potential complications. Diabetic patients should be advised and helped to stop smoking. They should also be encouraged to take regular exercise. The prevalence of this disease is dramatically increasing. Not only is it a potentially blinding condition if poorly managed, it will also lead to premature deaths. The development of new pharmaceutical agents to better manage diabetic disease and its complications have had a significant impact on improving patients' mortality and morbidity.

About the author

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Further reading

<http://www.who.int/mediacentre/factsheets/fs312/en/index.html>

<http://www.nhs.uk/Conditions/Diabetes>

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Acknowledgements

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- 1) Photographic Department, Royal Victoria Eye and Ear Hospital, Dublin Figures 1-4
- 2) Photographic Department, Moorfields Eye Hospital Figure 5
- 3) Ronan McCall, Medical Photographer, Portlaoise General Hospital Figures 6-7



Module questions

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

An answer return form is included in this issue. It should be completed and returned to CET initiatives (c-9568) OT, Ten Alps plc, 9 Savoy Street, London WC2E 7HR by January XX 2009

- 1) Which one of the following is incorrect? Hypoglycaemia:
- includes thirst as a recognised symptom
 - is treated with glucagon
 - is not treated with insulin
 - can be treated by intravenous fluids
- 2) Which one of the following is incorrect? In uncontrolled gestational diabetes:
- babies may be born with cardiac abnormalities
 - babies tend to be of low birth weight
 - mothers have an increased incidence of developing Type 2 diabetes
 - it affects both mother and baby
- 3) Which one of the following is incorrect regarding diabetes?
- diabetes Insipidus is a variant of diabetes mellitus
 - diabetes Insipidus is part of Wolfram's syndrome
 - patients with Friedrich's ataxia have a higher risk of developing diabetes mellitus
 - diabetes may be associated with deafness
- 4) Which one of the following is incorrect? Diabetic foot ulcer:
- occurs secondary to reduced sensation
 - occurs as diabetics are more prone to infection
 - occurs as a consequence of hyperperfusion
 - may be complicated by cellulitis and osteomyelitis
- 5) Which one of the following is incorrect? Autonomic dysfunction can manifest as:
- orthostatic hypotension
 - urinary incontinence
 - vomiting
 - reduced knee jerks
- 6) Which one of the following is incorrect? Atherosclerosis:
- occurs earlier in diabetic patients
 - is more severe in diabetic patients
 - is a risk for cerebrovascular accidents
 - is reversed by Aspirin
- 7) Which one of the following is incorrect? Acute optic disc swelling in diabetic patients:
- may represent a diabetic papillopathy
 - may be secondary to a central retinal vein occlusion
 - may be secondary to a non-arteritic ischaemic optic neuropathy
 - is a complication of Metformin
- 8) Which one of the following is incorrect? Retinal ischaemia:
- is associated with increased levels of VEGF
 - is treated by pan retinal photocoagulation
 - is not a feature of Type 2 diabetes
 - drives the development of rubeosis iridis
- 9) Which one of the following is incorrect? Hyperlipidaemia:
- should be aggressively treated in diabetic patients
 - is associated with the development of xanthelasmata
 - increases the risk of stroke
 - is not a feature of Type 1 diabetes
- 10) Presenting features of Type 1 diabetes include all of the following except:
- thirst
 - fatigue
 - collapse
 - weight gain
- 11) Which one of the following is incorrect? In Type 2 diabetes:
- insulin levels may be normal
 - there is resistance to insulin in the target tissues
 - it does not respond to exogenous insulin
 - patients may have advanced complications at presentation
- 12) Which one of the following is incorrect? Diabetic deaths:
- occur acutely in diabetic ketoacidosis
 - are a recognised complication of Mucormycosis
 - may be precipitated by severe sustained hypoglycaemia
 - are generally caused by diabetic retinopathy

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