



Non-Retinal Complications of Diabetes Mellitus

Shaheen P Shah MRCOphth MSc



Diabetes mellitus (DM) is a chronic disease that occurs when a) the pancreas does not produce enough insulin (insulin dependent diabetes or Type 1 diabetes), which is typically of early onset or b) when the body cannot effectively use the insulin it produces (maturity onset or non-insulin dependent diabetes). Type 1 diabetes can be regarded as an autoimmune disorder in which there is destruction of the pancreatic islet cells in a genetically susceptible host.¹

The world is currently experiencing a global epidemic of this incurable disease and the human and economic costs are enormous. Predictions estimate a doubling of the number affected from 180 million reaching an estimated 366 million by 2030.² The global increase in diabetes is attributed to increased life expectancy, urbanisation and changes in lifestyle and diet. Although previously recognised as a disease of the developed world, diabetes is becoming increasingly prevalent in the Developing World. In India alone, the number affected is expected to reach 80 million by 2030. Unfortunately, the majority of the burden from this disease is also now predicted to lie in low income countries and in those of working age.³

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Hyperglycaemia, or raised blood sugar, is a common effect of uncontrolled DM and over time, it leads to serious damage to many of the body's systems. The eyes, often indicators of systemic disease, are particularly vulnerable to the pathophysiological changes that occur as a result of DM and clinical trials have demonstrated that tight control of blood glucose, HbA1c and blood pressure can reduce the risk of blindness.⁴

Diabetic retinopathy, which is the leading cause of blindness in people under the age of 65 years in high income countries, is the most well-known ocular complication of DM.⁵ In addition to diabetic retinopathy, there are a number of other complications associated with this metabolic disorder that can be devastating to vision and ocular health.⁶ This review aims to describe these other, less well-known, complications.

Cataract

Cataract, opacification of the lens, is one of the most common causes of loss of vision, with an estimated 16 million people worldwide bilaterally blind as a result (Figure 1).⁷ DM influences the function and morphology of the lens⁸ and advanced glycation end products have been postulated as a possible pathogenic mechanism for diabetic cataract.⁹

Although the main cause of a visually significant cataract is ageing, both clinical epidemiological studies and basic science studies have documented an association between diabetes and cataract formation.^{4, 5, 10-20} The classic "diabetic snowflake cataract" that occurs in acute diabetes is nowadays rarely seen. In fact, the subtype of cataract most associated with DM is the posterior sub-capsular form and less frequently, cortical and nuclear cataract. The risk of cataract increases with

diabetes duration and severity of hyperglycemia.²¹ In addition, in patients with diabetes, cataract occurs at a younger age and is thought to progress more rapidly.²²

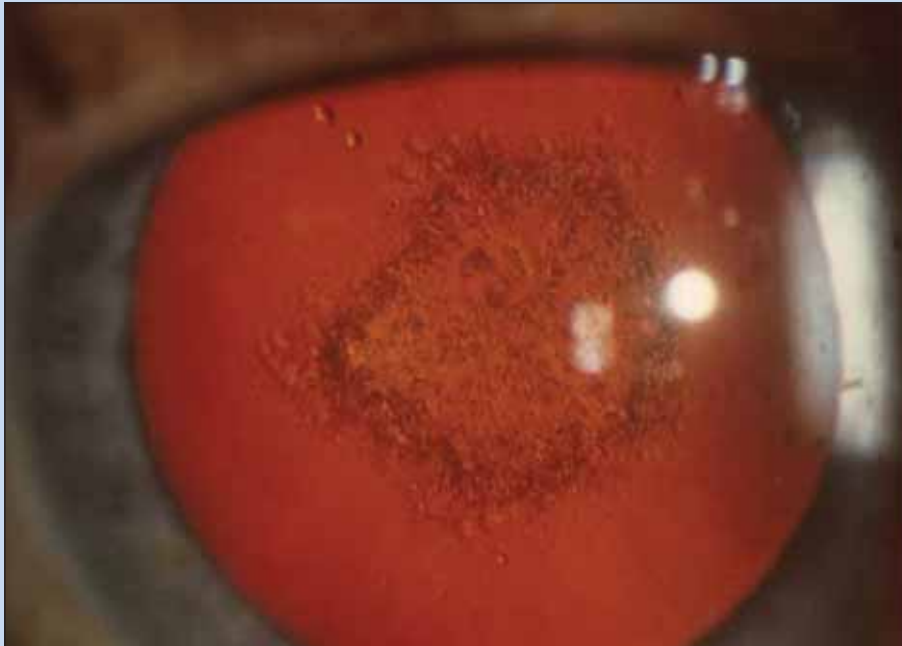
The Blue Mountains Eye Study showed impaired fasting glucose, in the absence of clinical diabetes, has also been noted as a risk factor for the development of cataract.¹⁷

The treatment for cataract is surgical, a highly cost-effective intervention, with excellent prognosis for sight restoration and it is now estimated that globally there are about 15 million cataract operations performed annually.²³ In fact, cataract surgery is now the most commonly performed surgical procedure in the UK.²⁴ In people with diabetes, the ten-year cumulative incidence of cataract surgery was found to be 8% in those with Type 1 diabetes and 25% in those with Type 2 diabetes in the Wisconsin

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➔ **Figure 1**
Posterior sub-capsular cataract

Epidemiologic Study.

Outcomes of cataract surgery in individuals with DM are known to be poorer, most commonly as a result of progression of diabetic retinopathy. However, better control of diabetic retinopathy with laser treatment prior to cataract surgery can improve surgical outcomes.^{25, 26} Some case control studies have also demonstrated that rates of posterior capsular opacification following cataract extraction are higher in individuals affected by DM.^{27, 28}

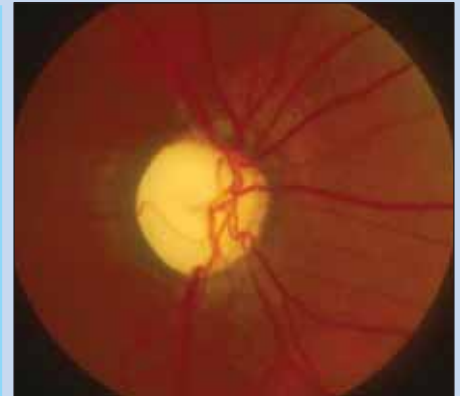
Infection

Patients with diabetes are predisposed to infections. Although not fully understood, the increased rate of infection seen in patients with DM is thought to relate to an immunosuppressive condition brought about by impaired innate and acquired immunity. Higher levels of hyperglycaemia are believed to correspondingly increase this level of immunosuppression.^{29, 30}

One of the most devastating complications following intraocular surgery is one of acute infectious exogenous endophthalmitis. This must be suspected in any patient that presents a few days after intraocular

surgery with decreasing vision, severe pain, redness, discharge, conjunctival injection, anterior chamber inflammation and vitritis. The estimated prevalence of presumed infectious endophthalmitis in the UK is estimated at 1.4 to 1.65 per 1,000 operations^{31, 32} and several studies have shown that patients with diabetes have an increased risk of developing this complication.³³⁻³⁶ The standard treatment of endophthalmitis is with intravitreal antibiotics but in resistant cases, pars plana vitrectomy is occasionally required. When affected with endophthalmitis, patients with DM tend to have a worse outcome following treatment and usually require more aggressive management.³⁷

Endogenous endophthalmitis (defined as an intraocular infection resulting from spread from a remote primary source) is also more common in diabetic patients. Gram-positive organisms are the most common bacterial pathogens, especially the streptococcal species, including *Streptococcus pneumoniae*. Although relatively rare overall (accounting for approximately 10% of all endophthalmitis cases³⁸ in one series), nearly a third of cases occurred in diabetic patients.³⁹



➔ **Figure 2**
Optic nerve changes associated with glaucoma

Orbital infections account for the majority of primary intraorbital disease processes. Sinusitis is the most common aetiology⁴⁰ and diabetes is a known risk factor for orbital cellulitis.⁴¹ Clinical signs and symptoms include erythema and inflammatory changes, as well as proptosis, injection, limitation of extraocular motility, and visual loss secondary to optic nerve compression.

One serious head and neck infection, seen almost exclusively in diabetic patients, is rhino-orbito-cerebral mucormycosis. Mucormycosis refers to a group of fungal infections^{42, 43} that can initially present with ophthalmic signs. Presentation may be as an orbital cellulitis, an ophthalmoplegia or as an orbital apex syndrome.⁴⁴ An examination of the nose may reveal necrosis of the nasal septum and turbinates, and the patient may complain of a blood stained discharge. The condition is an emergency and must be suspected in individuals with diabetes as the condition rapidly progresses and is associated with a high rate of mortality.⁴⁵ The treatment typically consists of a combination of surgical debridement and an intravenous anti-fungal agent.

Glaucoma

Glaucoma is a progressive optic neuropathy associated with typical optic disc changes (Figure 2) and visual field defects. Patients with DM are at risk of two major types of glaucoma: primary glaucoma and neovascular glaucoma (NVG).



➤ **Figure 3**
Acute uveitis

A number of recent epidemiological studies have found a positive association between diabetes and primary open angle glaucoma (POAG).⁴⁶⁻⁴⁹ The risk of glaucoma has been reported to be 1.6–4.7 times higher in individuals with diabetes compared with non-diabetic individuals. In fact, one case control study estimated the risk at nearly three times higher compared to normal controls.⁵⁰ The pattern of visual field loss may also be slightly different with significantly more changes in the inferior half of the field in diabetic patients with POAG⁵¹ than in non-diabetic patients. This difference was attributed to a possible vascular sub-component, the plausible biological mechanism being microvascular damage impairing blood flow to the anterior optic nerve.^{52, 53} Diabetes also impairs the autoregulation of posterior ciliary circulation, which may exacerbate glaucomatous optic neuropathy.⁵⁴ An alternative explanation of the higher prevalence in diabetics has been suggested by authors analysing a UK cohort.⁵⁵ The authors suggested that detection bias may be the reason – patients with DM have regular eye examinations and so this may lead to the increased detection of glaucoma.

Some reports suggest that DM may be associated with primary angle closure glaucoma (PACG). The mechanism is thought to be either via systemic autonomic dysfunction or increased lens thickness as a result of sorbitol overload.⁵⁶⁻⁵⁸ Patients with PACG usually present with an acute attack, which is associated with severe ocular

pain, blurred vision, redness, headaches and nausea. The symptoms, being brought about by an acute rise in intraocular pressure (IOP), require urgent referral to an ophthalmologist.

NVG, a severely blinding, intractable disease, occurs when new fibrovascular tissue proliferates into the chamber angle, obstructs the trabecular meshwork, and produces peripheral anterior synechiae and progressive angle closure.⁵⁹ In most cases, retinal ischaemia is the causal factor for NVG. Ischaemia releases factors that promote abnormal angiogenesis. One such factor, found in elevated concentration in the aqueous humour of patients with rubeosis and NVG, is vascular endothelial growth factor (VEGF).⁶⁰ Neovascularisation of the iris and angle, an early precursor of NVG, is commonly seen in patients with progressive diabetic retinopathy and a non-dilated slit-lamp examination including gonioscopy is vitally important to detect both iris and angle neovascularisation.

Proliferative diabetic retinopathy has been consistently demonstrated to be one of the leading causes of ischaemia and thus NVG⁶¹, and in one large series, nearly one third of patients with NVG had DM.⁶²

The management of NVG is two-fold: treatment of the underlying condition and treatment of the raised IOP. Panretinal photocoagulation is the standard treatment of ischaemic retinal disease, but more recently reports of regression of rubeosis with the use of anti-VEGF agents have been published.^{63, 64}

Raised Intraocular Pressure

Ocular hypertension is also significantly associated with DM.⁶⁵ Among eyes without glaucoma, suspected glaucoma or other disorders which could affect IOP measurement, DM has been significantly correlated with higher IOP ($p = 0.0019$).^{66, 67} Possible reasons to explain why individuals affected by DM tend to have higher IOP readings in large population-based studies include thicker central corneas and glucose-mediated increased corneal stiffening

due to collagen cross-linking. These reasons may also explain why those with ocular hypertension appear to have a reduced risk for glaucoma progression.^{68, 69}

Refractive error

Data from the National Health and Nutrition Examination Survey (NHANES) in the United States indicates that among adults aged >20 years with diabetes, 11.0% had visual impairment (i.e. presenting visual acuity worse than 20/40 in their better-seeing eye wearing glasses or contact lenses, if applicable) and approximately 65.5% of these cases of visual impairment were correctable. This finding underscores the importance of public awareness and public health intervention in reducing the prevalence of refractive error, especially among people with diabetes.⁷⁰ The prevalence of myopia in diabetic patients is also higher than in the non-diabetic population^{71, 72} and this may be related to a myopic shift due to nuclear sclerosis of the lens.

Corneal abnormalities

Patients with DM have been found to exhibit abnormalities of the corneal epithelium and endothelium.⁷³

Microscopic examination of the diabetic cornea has shown that the epithelium consists of more enlarged, pleomorphic and irregularly arranged cells with fewer microvilli, compared to the non-diabetic population. These findings suggest that the diabetic epithelium has an impaired ability to heal.⁷⁴

Diabetic patients can experience a variety of corneal complications, including superficial punctate keratopathy, persistent epithelial defects or trophic ulceration.⁷⁵⁻⁷⁷ In one study, corneal abnormalities eg gerontoxon (arcus senilis), limbal vascularisation, punctate keratopathy, endothelial dystrophy, recurrent corneal erosion (RCE) and ulcers were detected in up to 73.6% of patients with diabetes.⁷⁸ RCE syndrome is characterised by a disturbance at the level of the corneal epithelial basement membrane (related to adherence to Bowman's layer), resulting in recurrent



breakdown of the corneal epithelium. DM is a recognised risk factor for RCE and typical symptoms include pain, photophobia, blurred vision and hyperaemia.

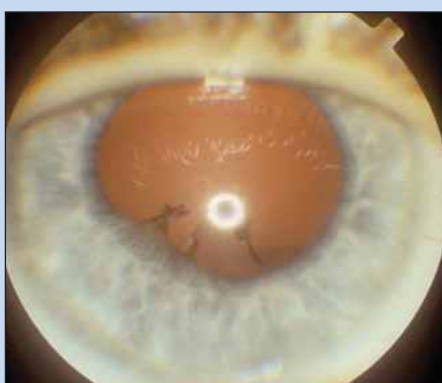
A decrease in corneal sensitivity has been demonstrated in individuals with DM^{79, 80} and therefore, neurotrophic keratopathy must be considered when a patient with diabetes develops otherwise unexplained corneal epithelial disease. Equally, DM must be considered when a patient presents with an unexplained neurotrophic corneal ulcer.⁷⁵⁻⁸¹ Diabetic peripheral neuropathy has also been found to be related to the presence of diabetic keratopathy.⁸²

Contact lens wearers that suffer from DM should be advised to take extra care with contact lens hygiene and should be advised to seek a medical opinion early if any symptoms of infection develop. Early intervention helps to prevent vision loss from microbial keratitis, the treatment of which is intensive topical antibiotic therapy (usually hourly administration for the first 48 hours).

Slower healing of the cornea after LASIK surgery has been cited as a possible reason for the increased rate of complications seen in patients with diabetes undergoing this refractive procedure.

Uveitis

Associations between anterior uveitis (Figures 3 and 4) and DM have been reported.⁸³⁻⁸⁵ Some but not all studies found that patients who suffered from diabetic autonomic neuropathy



➔ **Figure 4**
Posterior synechiae in uveitis

appeared to have a higher rate of uveitis, suggesting a possible common autoimmune process.⁸⁶⁻⁸⁸

Ocular Ischaemic Syndrome

The patient with ocular ischaemic syndrome (OIS) is typically an elderly male. OIS results from chronic vascular insufficiency and common findings include cataract, anterior segment inflammation, dilated retinal veins (but not tortuous), midperipheral large retinal haemorrhages, cotton-wool spots, and neovascularisation (iris, optic nerve, retinal). Ocular hypotony may even occur due to low arterial perfusion of the ciliary body.

OIS should be suspected in elderly patients with a history of neovascularisation of the anterior segment. Symptoms include ocular pain and visual loss including amaurosis fugax.^{89, 90} DM is a major risk factor for carotid artery stenosis and plaque formation.⁹¹ Stenosis of the carotid artery reduces perfusion pressure to the eye, resulting in the ischaemic phenomena. The prevalence of diabetes in patients with OIS is therefore significantly higher than in the general population.⁹⁰ Carotid doppler ultrasonography is useful to delineate the presence and severity of carotid artery stenosis and carotid endarterectomy has been shown to slow or prevent the progress of chronic ocular ischaemia caused by internal carotid artery stenosis.⁹² OIS has a poor visual prognosis and is also associated with a high mortality rate (five year mortality of 40%). The leading cause of death is cardiac disease, followed by stroke and cancer.^{93, 94}

Retinal Vascular Occlusion

The central retinal artery originates from the ophthalmic artery and its artery branches feed the inner retinal layers. Central retinal artery occlusion (CRAO) deprives the entire inner retina of its blood supply unless a cilioretinal artery is present (15–30% of eyes). Patients with CRAO usually present with a sudden significant loss of vision, an afferent pupillary defect, diffuse retinal whitening, and the resultant

classic 'cherry spot' on the macula. CRAO is associated with a poor final acuity of counting fingers or worse in approximately two-thirds of cases.⁹⁵

Patients with branch retinal artery occlusion (BRAO), usually have a focal wedge-shaped area of retinal whitening. Visible emboli in retinal arteries are observed more frequently in patients with BRAO.⁹⁶ DM is a recognised risk factor for retinal artery occlusion and patients should be referred immediately to an ophthalmologist for management, in particular to rule out the possibility of Giant Cell Arteritis.

Retinal vein occlusion (RVO) is an acute vascular condition characterised by dilated tortuous retinal veins with retinal haemorrhages, cotton wool spots, and macular oedema. Central RVO (CRVO) occurs at the optic disc, whereas branch RVO (BRVO) occurs at retinal branches, usually at the site of arterio-venous crossings (Figure 5). CRVO may be subdivided further into non-ischaemic and ischaemic types, the latter is associated with a poorer visual prognosis. Many but not all studies have found an association with DM.⁹⁷⁻⁹⁹ When a patient with DM presents with acute vision loss and asymmetric signs of retinopathy, RVO should be considered.

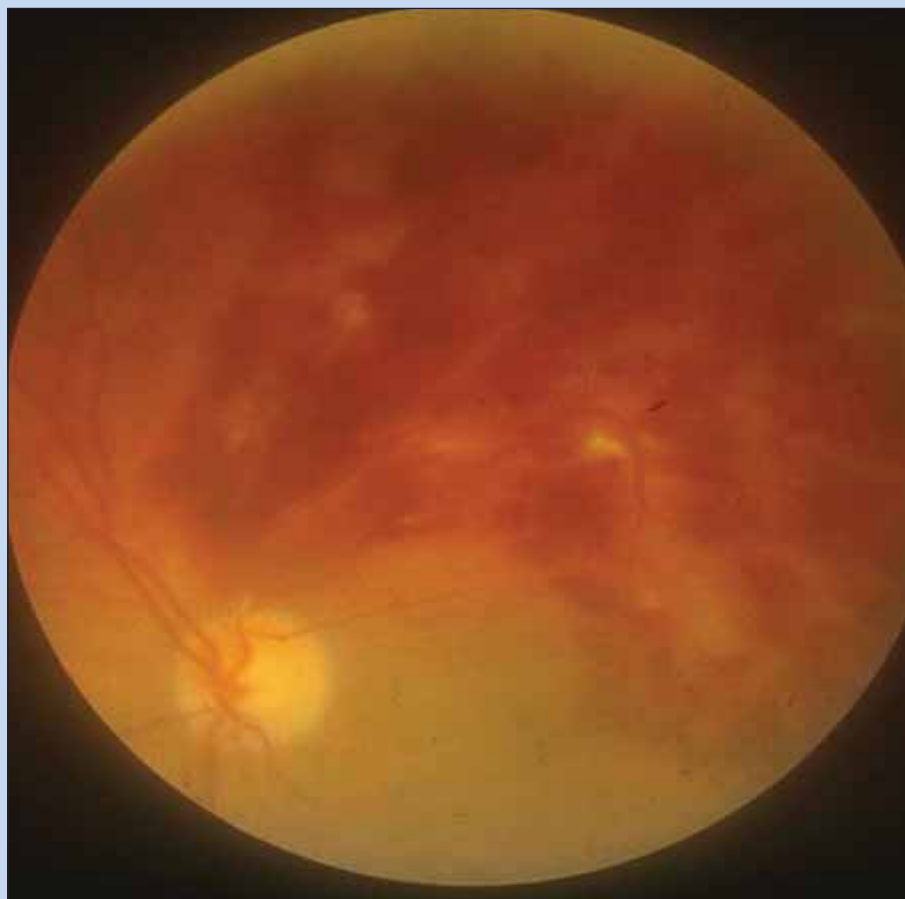
Neuro-ophthalmic complications

A range of neuro-ophthalmic manifestations occur secondary to diabetic eye disease and although relatively rare, some may be severely visually disabling and are therefore important to understand. Defects may be related to the vascular, neuropathic, or metabolic changes induced by DM. DM can affect the afferent visual system, the pupillary and accommodative reflexes, and the efferent system.¹⁰⁰

Facial Nerve Palsy

Diabetic peripheral neuropathy, a microvascular disease, is characterised by loss of myelinated nerve fibres, degeneration, and blunted nerve fibre reproduction.¹⁰¹

Peripheral facial nerve palsies are relatively common and most frequently



➔ **Figure 5**
Branch retinal vein occlusion

are diagnosed as Bell's Palsy. This is an acute onset paralysis or weakness without any known cause and is therefore a diagnosis of exclusion. DM is commonly associated.¹⁰²⁻¹⁰⁴ Corneal exposure results from poor lid closure (lagophthalmos) and inadequate blinking. Impaired lacrimation can also cause dry eye. However, epiphora from malposition of the lower lid is a more frequent symptom.

Ocular Motor Disorders

The most common ocular manifestations are in the form of diplopia (from misalignment of the visual axes) which is typically of the third (oculomotor), fourth (trochlear) and sixth (abducens) cranial nerves. Of 146 patients presenting to a London eye emergency department with diplopia, two-thirds had a cranial nerve palsy of which 42% had DM.¹⁰⁵ The cause is believed to be related to ischaemia in the nerve trunk resulting

from insufficiency of the vasa nervosa or small vessels that supply the relevant nerve.¹⁰⁶ Sometimes these patients have ipsilateral pain in the eye or orbit, the pathogenesis of which is not fully understood.

With an oculomotor (third) nerve palsy, the eye is typically infraducted and abducted (down and out), and ptosis can cover the pupil. In diabetic patients, the key finding is relative sparing of the pupillary sphincter. Loss of parasympathetic function results in pupil dilation and loss of accommodation. As the pupillary light-reflex fibres lie outside the oculomotor nerve, pupil involvement secondary to diabetic neuropathy rarely occurs. Pupil involvement usually means a compressive cause (aneurysmal or tumour involvement) and an anisocoria of 2mm or more requires urgent investigation.¹⁰⁷ If the ptosis is significant, the patient may not complain of diplopia, however

when diplopia is from a large-angle divergence of the visual axes, patching one eye is the only practical short-term solution. When the angle is smaller, fusion in the primary position can be achieved using a horizontal or vertical prism, or both.

Most fourth nerve palsies are of undetermined origin (other causes include trauma and microvascular disease) and usually present with a diplopia which has a horizontal and vertical component. Superior oblique palsy causes an ipsilateral hypertropia and excyclotorsion. Frequently, the patient will compensate with a contralateral head tilt. The presence of a superior oblique palsy can be confirmed using the Parks-Bielschowsky three-step test:

Step 1: Which eye is hypertropic?

Paralysis of the superior oblique is one cause of hypertropia.

Step 2: Is the hypertropia greater in left or right gaze?

Hypertropia due to superior oblique paralysis is greater on gaze to the contralateral side.

Step 3: Is the hypertropia greater in left or right head tilt?

Hypertropia due to superior oblique paralysis is greater in a head tilt to the ipsilateral side.

This test determines the paretic muscle by performing alternate cover testing in different head positions. It only works in cases of a single paretic muscle and since the superior oblique is the vertical muscle most commonly affected in isolation, this test is basically a test for dysfunction of the superior oblique.

DM is the most significant risk factor for an isolated sixth nerve palsy.¹⁰⁸ The diplopia in primary position is due to ipsilateral esotropia and becomes worse with gaze into the field of the weakened lateral rectus. Slow abducting saccades are another feature. Other conditions such as Duane's retraction syndrome or spasm of near reflex must be excluded.

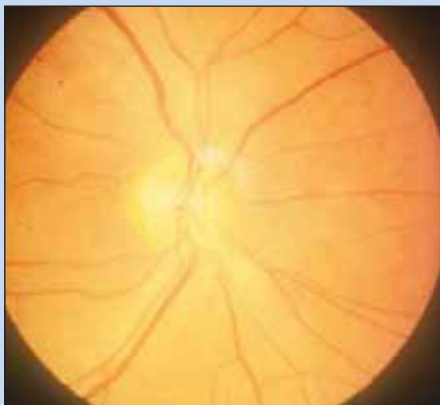
Management of these nerve palsies is challenging. The most important issue in diagnosing a microvascular cranial nerve palsy is whether a) it fits an expected pattern and b) whether it is isolated. In general, if a microvascular

aetiology is determined, then resolution is expected to occur in approximately two to four months. In an acute pupil sparing third nerve palsy, the patient must be watched closely over the following days to monitor for pupil involvement. Secondary aberrant regeneration (upper lid elevation on attempted downgaze) never occurs in microvascular cranial nerve palsy and neuro-radiological imaging must be obtained. If the nerve palsy is not isolated, or progressive, or other palsies are sequentially involved, then microvascular aetiology is unlikely. The age of the patient is also important and if the palsy occurs in a young age group, a microvascular aetiology is again unlikely. Lastly, diplopia is a common symptom of Giant Cell Arteritis and must be considered in anyone over the age of 50 years.

Accommodation and Pupils

Reduced amplitude of accommodation can be a result of diabetic eye disease. This can occur as a complication of panretinal photocoagulation for proliferative diabetic retinopathy. This can either be temporary or permanent. The prevalence is unknown but it is likely to be under reported. The complication is thought to arise as a result of thermal injury to the short ciliary nerve, causing parasympathetic denervation of the ciliary muscle.¹⁰⁹

Pupillary involvement in DM has similarities with Adie's tonic pupil. A tonic pupil responds to both light and near stimuli, but constriction and re-dilation is very slow. This is thought to



➔ **Figure 6**
Anterior ischaemic optic neuropathy

be due to denervation hypersensitivity. The segmental denervation (vermiform movements) observed in Adie's pupil, is not present in tonic pupils secondary to diabetes.

Anterior Ischaemic Optic Neuropathy (AION)

AION is an ischaemic infarction of the anterior portion of the optic nerve, secondary to occlusion of the short posterior ciliary arteries (Figure 6). There are two forms – arteritic (A-AION) and non-arteritic (NA-AION). The former is caused by Giant Cell Arteritis, an inflammatory vascular condition that requires prompt recognition and treatment if visual loss is to be prevented. The latter is thought to occur either because of transient non or hypoperfusion of the optic nerve head or an embolic lesion of the arteries/arterioles feeding the nerve head. Studies suggest that up to 25% of patients with NA-AION have a history of diabetes.¹¹⁰ In a person with a predisposing risk factor such as diabetes, the precipitating element is typically nocturnal arterial hypotension (these patients usually wake in the morning to discover their visual loss).¹¹¹ Ocular risk factors include an absent or small cup in the optic disc, angle closure glaucoma or other causes of markedly raised IOP. In contrast to visual acuity, which can sometimes be within normal limits in almost half of the eyes with NA-AION, visual field defects are universal, the most common being an altitudinal defect. Fundoscopy usually reveals swelling of the optic disc (occasionally just in one segment), prominent, dilated vessels over the disc and peripapillary haemorrhages. The fellow eye may become involved in a significant number of cases.

There is no proven treatment for NA-AION. In one study, patients were seen within two weeks of the onset of visual loss, with initial visual acuity of 20/70 or worse and an improvement was reported in 41–43% and a worsening in 15–19% at six months.¹¹² Diabetic papillopathy is sometimes considered a form of atypical NA-AION. Typical findings include a swollen, hyperaemic optic disc and

peripapillary capillary dilatation. An important differentiation to make is firstly, papilloedema and secondly, neovascularisation of the optic disc.

Syndromes

Wolfram syndrome, a genetic condition possibly of autosomal recessive inheritance, is associated with juvenile onset DM and optic atrophy. The syndrome is also known as DIDMOAD (Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy, and Deafness).¹¹³ In most cases, DM and optic nerve atrophy are seen in the first and second decades, between the ages of 2–15 and 4–18 years, respectively.¹¹⁴ In 12 UK families with Wolfram syndrome, genetic linkage to the short arm of chromosome 4 was confirmed.¹¹⁵ Optic atrophy which results from a permanent loss of ganglion cells has also been reported as a direct consequence of diabetic ketoacidosis.¹¹⁶

Alstrom syndrome, another rare genetic condition should be suspected in a child with a retinal dystrophy, particularly if their weight is above the 90th percentile. The condition is progressive with no perception of light by the age of 20 years. The diagnosis of DM often occurs in the second or even third decade of life.¹¹⁷

Conclusion

This article is not a comprehensive review but, apart from the obvious diabetic retinopathy, provides an overview of the range of ocular pathologies that are associated with DM. Optometrists are advised to be familiar with the possible presentations, a number of which are ocular emergencies that require urgent medical investigation and treatment.

About the author

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Figures courtesy of Professor Susan Lightman

References

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- Which one of the following is correct? Type 1 diabetes is:
 - of late age onset
 - occurs when there is an excess of insulin
 - occurs when there is destruction of the pancreatic islet cells
 - occurs when there is an excess of growth hormone
- Which one of the following is correct regarding diabetes and infection?
 - there is an increased risk of post-operative endophthalmitis
 - gram-negative organisms are the most common bacterial pathogens
 - the immunosuppressive effect does not vary with level of hyperglycaemia
 - outcomes of the treatment of endophthalmitis are similar to individuals not affected by diabetes
- Which one of the following is correct? Rhino-orbito-cerebral mucormycosis:
 - refers to a group of fungal infections
 - is a relatively benign condition
 - does not require urgent attention
 - presents with uveitis
- Which one of the following is incorrect regarding diabetes and glaucoma?
 - there is a positive association between diabetes and POAG
 - the risk of neovascular glaucoma is increased in patients with diabetes
 - microvascular damage affecting the anterior optic nerve is thought to be involved in the pathogenesis
 - diabetic patients with open angle glaucoma are non responsive to prostaglandin analog hypotensive agents
- Which one of the following is correct? Neovascular glaucoma:
 - is easily treatable
 - is associated with good prognosis
 - occurs due to excessive uveoscleral outflow
 - is caused in most cases by retinal ischaemia
- Which one of the following is incorrect? The corneal epithelial cells of a diabetic:
 - are enlarged
 - are regularly arranged
 - have fewer microvilli
 - are pleomorphic
- All of the following are characteristics of ocular ischaemic syndrome except:
 - cataract
 - stenosis of the carotid artery
 - ocular pain
 - corneal decompensation
- Which one of the following is incorrect regarding facial nerve palsy?
 - corneal exposure results from poor lid closure
 - corneal sensation is always reduced
 - the blink reflex is impaired
 - epiphora results from malposition of the lid
- Which one of the following is correct regarding a third nerve palsy caused by an aneurysm compared to a third nerve palsy due to diabetes?
 - it affects the pupillary response to light
 - it does not affect accommodation
 - it never causes upper lid elevation on attempted downgaze
 - secondary aberrant regeneration occurs in microvascular cranial nerve palsy
- Which one of the following is incorrect regarding non-arteritic anterior ischaemic optic neuropathy (NA-AION)?
 - it may occur due to hypoperfusion of the optic nerve head
 - it may occur due to an embolic lesion
 - ocular risk factors include a large optic disc cup
 - it is associated with diabetes
- Which one of the following is incorrect regarding NA-AION?
 - it is associated with optic disc oedema
 - visual acuity may be within normal limits
 - the fellow eye may become involved
 - the condition is fully treatable
- Which one of the following is correct? Wolfram Syndrome:
 - is a common condition
 - is a condition that has no genetic tendency
 - includes hearing loss
 - is typical in individuals with Type 2 diabetes

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