

Detection and treatment of diabetic

The vital role of OCT

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CASE REPORT

THE PRESENT Optometry Clinic at UNSW Australia, Kensington opened in 2000 with the purpose of teaching Year 4 and 5 students the clinical skills necessary to become competent practitioners. The clinic is open to members of the public, university staff and students. About 4,000 patients present annually, many of whom have vision-threatening pathology.

According to the International Diabetes Federation, diabetes mellitus (DM) affects 366 million people worldwide. That number is predicted to rise to 552 million by 2030.¹ Prevalence and incidence of type 1 and 2 DM varies greatly among different populations and affects male and females equally.²

Diabetic eye disease is rare on initial diagnosis of type 1 DM and about 20 per cent of type 2 DM, rising to 90 per cent and 60 per cent, respectively, after 20 years disease duration.³ It is common for undiagnosed type 2 DM, retinopathy discovered during a routine eye examination to be the first sign of systemic disease.

A 59-year-old Bangladeshi male presented to the UNSW optometry clinic in June 2013. He had been experiencing reduced vision in his right eye for three months and wanted a second opinion. He reported himself to have been a well-controlled type 2 diabetic sufferer for eight years, with average blood sugar levels (BSL) of 6-7 mmol, blood pressure (BP) levels of 110/70 and three-to-four monthly checks with his general practitioner. Ocular history was unremarkable apart from bilateral cataract extraction.

Presenting vision was R 6/12 and L 6/7.5. Best-corrected vision with a low hyperopic script were R 6/7.5 (niph) and L 6/6. Intraocular pressures were 17 mmHg both eyes with Perkins applanation tonometry.

Dilated fundus examination revealed scattered dot-blot haemorrhages at both posterior poles. Scattered exudates and micro-aneurysms were found within the right macula region whilst the left eye had an unusual inner-limiting membrane (ILM) reflex at the macula with dot haemorrhages and microaneurysms. (Figures 1A, 1B, 2A and 2B)

After an urgent assessment at the eye hospital two weeks later, the registrar recommended better BSL control, in conjunction with the general practitioner, as first line treatment. On review in October 2013, the registrar decided to continue to monitor the retinopathy, while maintaining strict glycaemic and blood pressure control.

The patient returned to UNSW optometry clinic February 2014 with a review booked at the eye hospital in April 2014. He reported no visual complaints with well controlled BSL and BP levels. Best-corrected vision was R 6/6-2 and L 6/6-2. Slitlamp examination was unremarkable. Fundus examination (Figures 3A and 3B) revealed an increase in retinal haemorrhages and exudates within the right macula region. In the left eye, there was a cotton wool spot near the

disc and a circinate ring of exudates within one disc diameter of the macula. OCT also reflected these new findings (Figures 4A and 4B).

In light of these findings, the eye hospital was contacted to organise a more urgent assessment. Despite the patient reporting compliance with systemic control, it was evident that this alone was not enough to prevent retinopathy progression.

Discussion

Hyperglycaemia induces a number of biochemical reactions such as vascular endothelial growth factor (VEGF), aldose reductase and angiotensin enzyme expression.^{4,5} These culminate into vessel leakage, haemorrhaging and ischaemia. Inflammatory pathways and leukostasis are also being investigated as pathomechanisms of diabetic eye disease.⁵

Diabetic retinopathy (DR) is the leading cause of preventable blindness of the working age population in developed countries⁴ and is graded based on the clinical features present. The Early Treatment of Diabetic Retinopathy Study (ETDRS) scale is the gold standard from which a number of other grading scales have been adapted.⁶

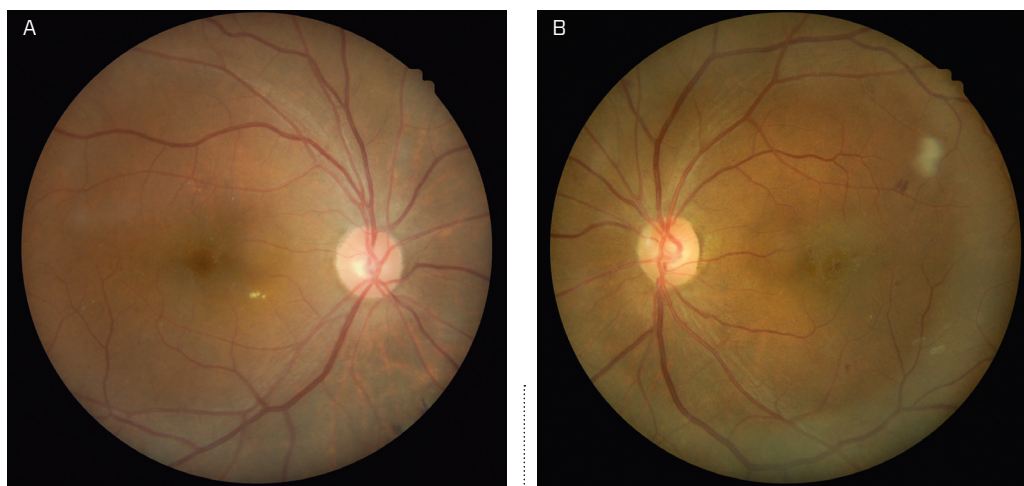
In the case of this patient, on first presentation, he had mild NPDR both eyes and diffuse macula oedema in the right eye. On second presentation, he had moderate NPDR both eyes and CSME in the left eye. This is also classified as vision threatening (VTDR).

Global estimates worldwide found DR present in 34.6 per cent, PDR in 7.0 per cent, DME in 6.8 per cent and VTDR in 10.2 per cent of diabetic patients.^{7,8}

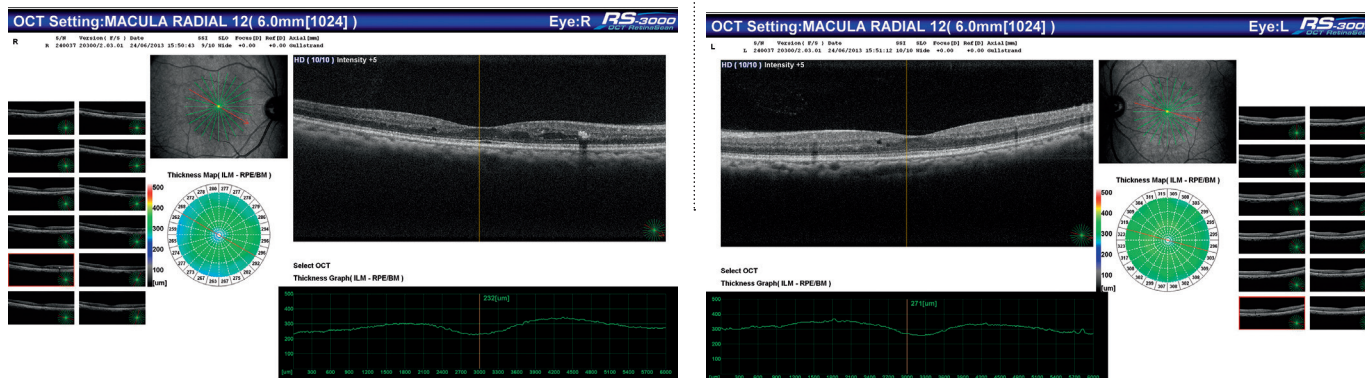
Microaneurysms are small round 'protrusions' that occur locally at capillary walls which have weakened due to capillary closure. These can present de novo or within circinate exudate rings.⁹

macular oedema

in diagnosis and effective comanagement



▲ Figure 1A and Figure 1B. Fundus images of patient (artefact at 2 o'clock)



▲ Figure 2A and Figure 2B. OCT of OD revealed exudates and cystic spaces confirming the presence of macular oedema. This also supported the reduced vision findings in OD.

Intra-retinal haemorrhages occur as a result of blood vessel rupture and are classified based on their location within the retina. Flame haemorrhages lie within the tightly packed nerve fibre layer (NFL). Dot haemorrhages are round, well-defined and found within the outer plexiform layer (OPL). Blot haemorrhages are larger and less defined compared to dots and occur within the less compact inner nuclear layer (INL).⁹

Hard exudates occur at the OPL and represent leakage from increasingly

permeable capillary walls. These can form singularly or as a circinate ring following rupture of a microaneurysm.⁹

In the presence of increasing non-perfusion, venous beading presents as focal dilation and thinning of a venous wall and is highly associated with risk of PDR developing. Cotton wool spots represent localised NFL swelling secondary to obstructed axoplasmic flow.⁹

Intraretinal microvascular abnormalities (IRMA) represent

abnormal communication between arterioles and venules and look similar to neovascularisation (NV). Unlike NV, IRMA lie within the retina, do not leak and do not form abnormal attachments to the vitreous.⁹

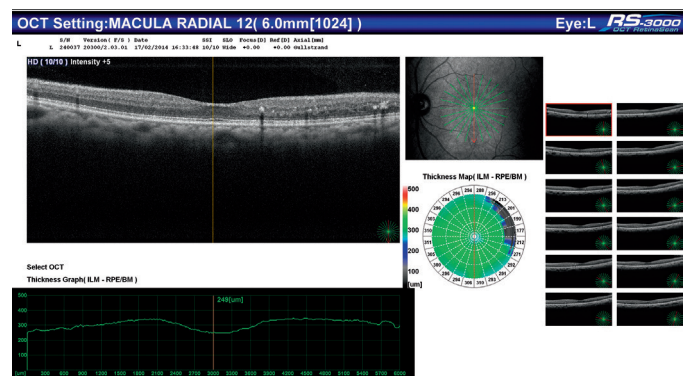
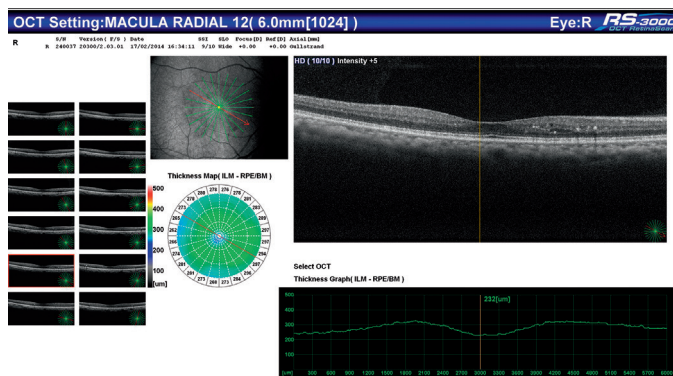
Proliferative changes occur when significant retinal ischemia stimulates angiogenic factors such as VEGF, resulting in new blood vessel formation on the retinal surface.¹⁰



▲ Figure 3A. Increase in retinal haemorrhages and exudates within the right macula region



▲ Figure 3B. Single cotton wool spot adjacent to optic disc and a circinate ring of exudates within 1 disc diameter of the macula in the left eye



▲ Figure 4A and Figure 4B. OCT confirms findings of bilateral macula oedema

Vital role of OCT

From page 5

Diabetic macula oedema (DME) is defined as retinal thickening (build-up of fluid and proteins in the Henle Layer and INL) secondary to a breakdown of the blood retinal barrier.¹¹ DME is the leading cause of vision loss in type 2 diabetes.³ As this case illustrates, urgent ophthalmological assessment is essential.

Treatment

The three major risk factors for DR are duration of diabetes, hyperglycaemic levels and hypertension.² The Diabetes Control and Complication Trial (DCCT) and UK Prospective Diabetes Study (UKPDS) both found that intensive systemic treatment—maintaining blood glucose levels as close to the normal range (HBA1c 7.0 per cent) and strict blood pressure control (< 140/80)—delays the onset and progression of micro-vascular complications of type 1 and 2 diabetes including retinopathy.^{12,13}

It is essential that a multi-disciplinary team of GPs, endocrinologists, dietitians and nurse educators is involved in achieving this alongside eye-care providers.

The ETDRS found that following focal laser photocoagulation, there was 50 per cent reduction in moderate vision-loss in CSME patients¹⁴ and for a number of years focal laser has been mainstay treatment for such patients. Focal laser seals leaking microaneurysms, while grid laser is used in diffuse cases. It is hypothesised that the reduced retinal blood flow, due to increased local oxygenation via photocoagulation, results in reduction of macula fluid.¹⁵

Anti-VEGF therapy, such as bevacizumab is fast replacing laser as first-line treatment for centre involved macular oedema, due to its greater efficacy and increased safety involving the central macula.^{5,15} It is an antibody of VEGF-A and inhibits angiogenesis.^{5,10} For proliferative retinopathy, pan-

retinal photocoagulation is still first-line treatment for fibrous regression of neovascularisation, reduction in vitreous haemorrhage and reduction in tractional retinal detachment.⁵

Conclusion

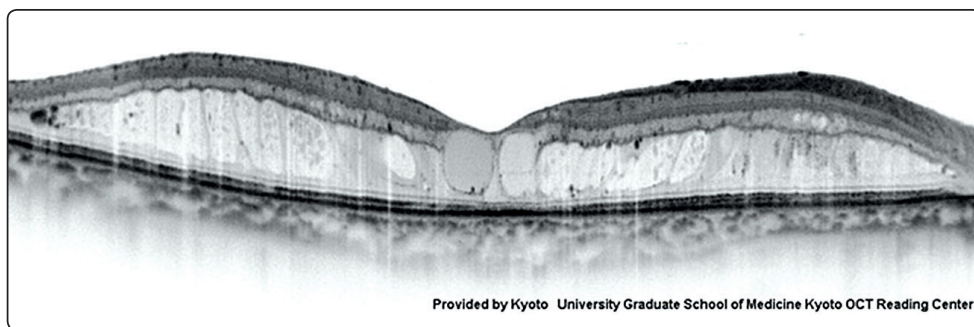
Effective screening programs are essential for early diagnosis of diabetic retinopathy. This case report shows that OCT is a valuable tool that can make a rapid diagnosis of macular oedema as well as monitor for progression. It would be worthwhile for all optometrists to have access to an OCT to use alongside dilated fundus examination and stereo-photos in ensuring timely diagnosis, effective comanagement and appropriate treatment. ▲

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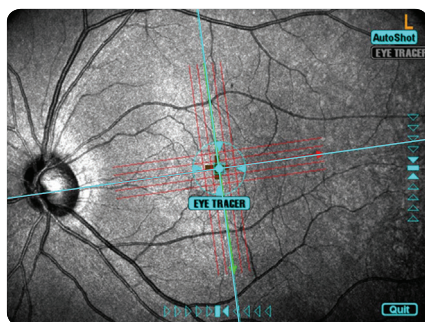
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