

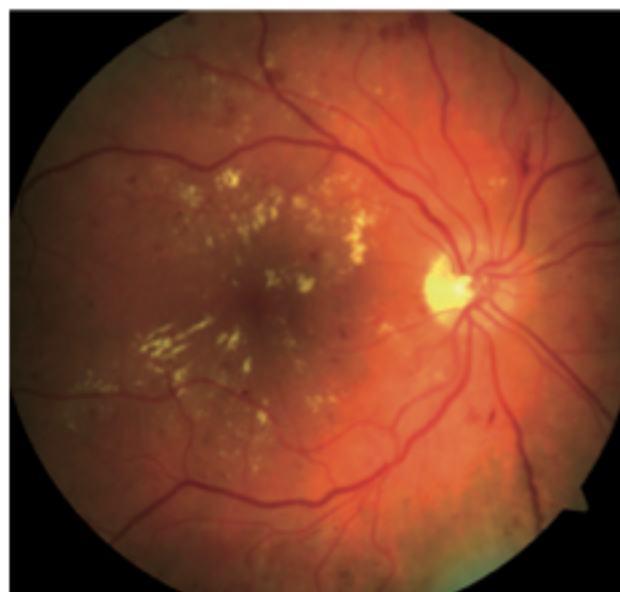
# Diabetic maculopathy

**D**iabetic retinopathy is the most common cause of blindness in working-age Australians. Diabetes can affect the eye in numerous ways but maculopathy is the most common means by which vision deteriorates (fig 1). For many years the primary treatment of this disease was photocoagulation (laser) therapy, however this usually does not result in vision improvement, rather a slowing of vision deterioration. This article looks at what interventions make a difference and includes recent advances in the treatment of diabetic maculopathy.

## Medical therapies remain important

Many studies have shown that strict long-term control of blood sugar levels and blood pressure can slow the progression of both diabetic maculopathy and retinopathy. Managing dyslipidaemia, renal failure and anaemia can also help slow the progression. Smoking increases the risk of progression of diabetic eye disease.

Fenofibrate appears to slow the progression of maculopathy and reduce the amount of laser treatment required. These effects appear to be independent of its lipid lowering ability, and as such, one may consider using



■ Fig 1. Retinal view of diabetic retinopathy and maculopathy.

it in patients with a 'normal' lipid profile, or in conjunction with other lipid lowering medications, if deemed safe.

## Retinal photocoagulation (laser)

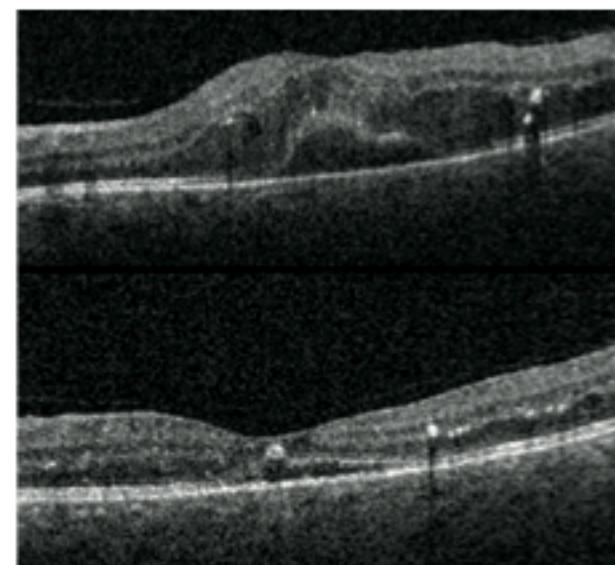
Retinal laser has been used for many years and remains the mainstay of treatment for diabetic macular oedema not involving the central macula. It has been shown to reduce the risk of vision deterioration by 50% compared with sham. Unfortunately, it does not tend to improve vision – rather, the aim of therapy is slowing the decline. It is decreasingly being used for patients with oedema involving the centre of the macula.

## Intravitreal pharmacotherapies

Over the last decade, triamcinolone injected into the vitreous cavity has been used in the management of diabetic maculopathy. It is effective in reducing the oedema associated with diabetic maculopathy, however it has a relatively high complication rate (due to cataract formation and intraocular hypertension) and therefore tends to be used as an adjunctive therapy only.

Anti-vascular endothelial growth factor agents, such as bevacizumab (Avastin<sup>®</sup>) and ranibizumab (Lucentis<sup>®</sup>), have been in common clinical practice for several years. VEGF is released from affected retina and is a major contributor to macular oedema.

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■ Fig 2. OCT scan pre and post treatment, demonstrating reduction in macular oedema following intravitreal bevacizumab.

Recent randomised controlled studies have shown that anti-VEGF agents are superior to photocoagulation in terms of visual acuity gained. They also have less ocular complications than intravitreal triamcinolone.

Late last year, the TGA approved the use of intravitreal ranibizumab for the treatment of centre-involving diabetic maculopathy and the drug is now considered the gold standard for treatment of center-involving diabetic macular oedema. Generally, patients need to have repeated intravitreal injections to maintain visual acuity gains (see Fig 2). ■

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