AMD: New treatments and future research

Dr Jane Khan,
Ophthalmologist,
Mosman Park. Tel 9385 6665

The devastating effects of age-related macular degeneration (AMD) have, until recently, been largely untreatable. A treatment has become available in the last couple of years that can reverse some of the devastating visual loss occurring in wet AMD.

Anti-VEGF therapy

The most visually threatening lesion of wet AMD is growth of new vessels from the choroid under the macula –vessels that leak, bleed and fibrose, causing permanent scarring and destruction of overlying photoreceptors. Vascular endothelial growth factor (VEGF) is released from damaged tissue and causes new blood vessels to grow.

Monoclonal antibodies to VEGF (e.g. ranibizumab) inhibit this factor and prevent new vessel growth – a treatment that can not only stabilise vision but can reverse some of the visual loss (figure 1). Ranibizumab is now available in Australia as a PBS-listed therapy for wet AMD.

The monthly intraocular injection seems a daunting prospect for new patients but is reported to be less uncomfortable than venepuncture. The procedure can be performed in an outpatient setting as long as sterile techniques are adhered to rigorously. Complication rates are fortunately low: from the injection, lens damage or dislocation, retinal tears or detachment and vitreous haemorrhage; and from the drug itself a possible increased rate of thromoembolic events. For the latter, figures are small and may not be clinically significant given that the treatment population is demographically at increased risk of such events.

Vitamin supplements

Results from the large age-related eye disease (AREDS) study have demonstrated supplementation with a combination of anti-oxidant vitamins and zinc can slow progression of early stages of AMD to the late forms. We now recommend this preventative measure to patients with high-risk features of pigment clumping, intermediate and soft drusen or late AMD in one eye.

Future research

Unfortunately, there is still no effective treatment for 'dry AMD' where photoreceptor loss and RPE atrophy can cause complete loss of central vision to the same extent as wet AMD. The underlying pathologic mechanisms are more obscure and therefore targeted treatments are elusive. A trial is investigating the effects of an implantable device to release ciliary neurotrophic factor. Another trial is currently underway to investigate vitamin and antioxidant supplementation for dry AMD. Further similar trials are now taking place to investigate the effect of supplementation with omega-3 fatty acids for wet AMD and results are expected this year. ■

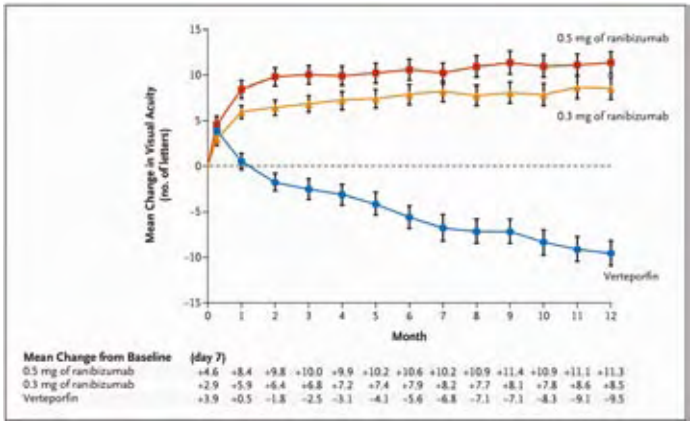

Figure 1 (Reproduced from Brown et al 2006). Patients with predominantly classic subfoveal CNV were treated with ranibizumab monthly injections and compared to patients treated with photodynamic therapy (verteporfin). At month 12, 96% of ranibizumab 0.5 mg treated patients lost <15 letters of visual acuity compared with 64% of photodynamic therapy-treated patients.