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Intravitreal triamcinolone acetonide for refractory diabetic macular oedema

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Diabetic macular oedema is caused by hyperpermeability of the retinal vasculature and results in retinal thickening due to intraretinal fluid accumulation between the inner and outer plexiform layers. The Early Treatment Diabetic Retinopathy Study defined clinically significant macular oedema (CSMO) by the presence of one or more of the following: retinal thickening at or within 500 μm of the centre of the macula; hard exudates at or within 500 μm of the macular centre, if associated with adjacent retinal thickening; retinal thickening greater than or equal to one disc area, any part of which is within one disc diameter of the centre of the macular centre.¹ Focal argon laser photocoagulation is beneficial in non-ischaemic CSMO but less so in diffuse macular oedema.² A treatment now used for macular oedema in various diseases is intravitreal triamcinolone acetonide.

CASE HISTORY

A man of 46 sought advice after two days of bilateral photophobia with red, watery eyes. 8 years before, non-insulin-dependent diabetes had been diagnosed, but he was otherwise well. On examination, unaided visual acuity was 6/18 (improving to 6/6 pinhole) in the right eye and 6/6 in the left. He was diagnosed as having right-sided non-granulomatous uveitis without systemic associations. There were no signs of diabetic retinopathy or cystoid macular oedema. After two weeks of topical steroid treatment the uveitis resolved completely with visual improvement to 6/6 unaided bilaterally. Three months later, right diabetic maculopathy developed. CSMO was absent, so focal laser treatment was not indicated at this stage. Six months later, visual acuity had declined from 6/6 to 6/12 in his right eye

and CSMO was now evident, with macular thickening on ocular coherence tomography. Fluorescein angiography was performed before focal macular argon laser treatment. Three months post-laser there was little angiographic change, and because of the cystoid nature of his macular oedema it was felt that, if his visual acuity decreased, intravitreal triamcinolone should be tried. CSMO then developed in the left eye, and fluorescein angiography indicated that this was suitable for focal laser treatment; however, the right eye showed ischaemic maculopathy (Figure 1) for which laser treatment was thought inappropriate. A month later the patient's unaided visual acuity was 6/24 in the right eye and 6/18 in the left eye, without improvement with a pinhole. He refused laser treatment for the left eye but agreed to intravitreal triamcinolone on the right. 4 mg triamcinolone was injected into the right globe under sterile conditions. After a month visual acuity in this eye had improved to 6/9. On ocular coherence tomography, retinal thickness had lessened from 527 μm to 168 μm (Figure 2). This improvement was still evident six months post-treatment.

COMMENT

Focal macular oedema is characterized by leakage from microaneurysms and dilated capillary segments, whereas in diffuse macular oedema the leakage results from a generalized breakdown of the blood–retinal barrier, with formation of large cystoid spaces. The disappointing results with focal laser photocoagulation in conditions such as ischaemic diabetic maculopathy have prompted evaluation of other approaches. Corticosteroids have been used orally, topically and by periocular injection in the treatment of cystoid macular oedema because of their ability to inhibit the arachidonic acid pathway and

Figure 1 Fluorescein angiogram showing increased size and irregularity (arrows) of the foveal avascular zone

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Figure 2 Ocular coherence tomogram showing (a) diabetic macular oedema with cystoid space formation. (b) Reduction in macular thickness 1 month after intravitreal triamcinolone injection (in colour online)

prostaglandin production. They are also thought to reduce the production of vascular endothelial growth factor, a vascular permeability facilitator. Triamcinolone acetonide has been shown experimentally to reduce breakdown of the blood–retinal barrier experimentally³ and prospective studies point to the value of intravitreal injection in management of diffuse macular oedema caused by diabetes.^{4,5}

This novel mode of administration, giving rapid delivery of triamcinolone to its site of action, seems to reduce cystoid macular oedema and restore visual acuity.⁶ Possible complications are retinal detachment, vitreous haemorrhage, and endophthalmitis, but these risks must be set against the strong likelihood of irreversible visual loss from chronic untreated macular oedema. No directly toxic effects to the retina or optic nerve have been recorded.⁷ Further studies are required to evaluate the long-term efficacy and safety and the possible need for repeated injections or sustained-release devices.

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