Intravitreal Triamcinolone Acetonide for the Treatment of Diffuse Diabetic Macular Oedema – A Case Report

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Abstract

Introduction: Although laser photocoagulation is the primary treatment for diabetic macular oedema, treatment of eyes with diffuse macular oedema has been disappointing. Intravitreal injection of steroids is being investigated for the treatment of diabetic macular oedema. Preliminary results indicate that steroid injections do improve macular oedema, but it is not clear if they improve visual acuity. Clinical Picture, Treatment, and Outcome: In this report, we describe a patient with a form of diffuse diabetic macular oedema that responded favourably to intravitreal steroid injections, with improvements in both foveal thickness and visual acuity. Conclusion: Intravitreal steroids can be useful for the treatment of diffuse diabetic macular oedema.

Key words: Cataract, Glaucoma, Endophthalmitis, Steroids

Introduction

Focal and grid laser photocoagulation are the primary surgical treatments for diabetic macular oedema.1 However, laser treatment of eyes with diffuse macular oedema has been disappointing.2 Intravitreal injection of steroids is being investigated in the treatment of diabetic macular oedema and prospective, controlled studies are currently underway. Preliminary results indicate that although intravitreal steroid injections do improve macular oedema, it is not clear if there is a beneficial effect on visual acuity.3 In this report, we describe a patient with a diffuse, cystic, diabetic macular oedema that responded favourably to intravitreal steroid injections. This patient experienced improvements in both foveal thickness and visual acuity following intravitreal injection of triamcinolone acetonide.

Case Report

A 38-year-old male presented for treatment of his diabetic eye disease. He had been diabetic since the age of 3 years and had undergone focal laser treatment and panretinal photocoagulation (PRP) OU prior to presentation. Best-corrected visual acuity was 20/100 OD and 20/80 OS. Anterior segments were quiet and intraocular pressures were 11 mm Hg OU. Diffuse, diabetic macular oedema was present OU (Fig. 1). Fundus fluorescein angiography (FFA) revealed a diffuse, petalloid pattern of diabetic macular oedema (Fig. 2). Optical coherence tomography (OCT) revealed multiple, cystoid spaces within the outer plexiform layer (Fig. 3). Foveal thickness was 527 µm OD and 529 µm OS. The patient underwent bilateral sequential intravitreal injections of triamcinolone acetonide (4 mg in 0.1 mL suspension).

Four months later, best-corrected visual acuity had improved to 20/50 OD and 20/60 OS. Foveal thickness had improved to 185 µm OD and 202 µm OS with restoration of foveal contours (Figs. 3C and 3D).

One year after his initial presentation, visual acuity had deteriorated to less than 20/400 OD and 20/100 OS. The patient had developed nuclear sclerotic and posterior subcapsular cataracts. Fundus examination, FFA and OCT all confirmed recurrent, diffuse, cystoid, diabetic macular oedema. Foveal thickness was now 499 µm OD and 568 µm OS.

References

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Fig. 1. Fundus photographs.
A: Right eye. Although there is diffuse macular thickening, there are almost no microaneurysms, haemorrhages, or exudates. Scars from previous laser treatment are present temporally.
B: Left eye. There is diffuse macular thickening, but few other signs of macular oedema. There is a small area of neovascularisation on the disc, as well as a larger area of neovascularisation in the temporal retina. Scars from previous laser treatment are present temporally.

Fig. 2. Fundus fluorescein angiograms.
A: Right eye at 6 minutes post-injection. A diffuse, petalloid pattern of leakage is apparent circumferentially around the central fovea.
B: Left eye at 6 minutes post-injection. There is a diffuse petalloid pattern of leakage around the central fovea. Areas of neovascularisation are leaking temporally.

Fig. 3. Optical coherence tomography.
A: Right eye at original presentation. There are diffuse, cystoid spaces within the outer plexiform layer and marked foveal thickening (527 µm). Beneath the cystic spaces, there is a macular detachment (arrow).
B: Left eye at original presentation. There is a large, central foveal cyst as well as diffuse thickening of the outer plexiform layer. Foveal thickness is 529 µm.
C: Right eye 4 months following intravitreal injection of triamcinolone. Foveal anatomy has returned to normal. Foveal thickness is 185 µm. Visual acuity has improved from 20/100 to 20/50.
D: Left eye 4 months following intravitreal injection of triamcinolone. Foveal anatomy has returned to normal. Foveal thickness is 202 µm. Visual acuity has improved from 20/80 to 20/60.

Discussion
This report documents a patient with a particular form of diabetic macular oedema characterised by diffuse thickening of the macula on clinical exam, diffuse, petalloid leakage on angiography, marked cystoid changes in the outer plexiform layer on OCT, and marked thickening of the fovea on OCT. Treatment with intravitreal steroids induced a dramatic decrease in foveal thickness and improvement in visual acuity. These improvements appear to be more substantial than those observed in case series of patients treated with intravitreal steroids for typical clinically
significant macular oedema. It is not known if the fact that our patient was a Type I diabetic influenced his clinical outcome.

As described in larger series of diabetic patients undergoing intravitreal steroid injections, our patient experienced some of the side effects associated with this treatment. The complications described with intravitreal steroid injections include elevated intraocular pressure, cataracts, and endophthalmitis. Our patient’s intraocular pressures were successfully managed medically, but he did require cataract surgery, due in part to steroid-induced lens opacities. His tractional retinal detachment was due to proliferative diabetic retinopathy and not due to the intravitreal injections.

The ideal timing of re-treatment in patients that respond to intravitreal steroid injection is unknown. Recent evidence indicates that the beneficial effect of larger doses of triamcinolone lasts about 7 to 8 months. Our patient’s macular oedema appeared to relapse after 1 year, suggesting that injections may need to be repeated every 6 to 9 months.

In summary, in this single case, intravitreal steroids were beneficial for the treatment of diffuse diabetic macular oedema. Although we did not directly compare the effects of grid or focal laser treatment to intravitreal triamcinolone in this case, intravitreal triamcinolone appears to be more suitable for this particular form of diffuse, petalloid diabetic macular oedema.

Competing Interests
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REFERENCES