Intravitreal Triamcinolone Acetonide for Patients with Macular Edema due to Branch Retinal Vein Occlusion

Kai-Chun Cheng and Wen-Chuan Wu
Department of Ophthalmology, Kaohsiung Medical University Hospital, Kaohsiung, Taiwan.

We designed a case series study to evaluate the outcome of intravitreal triamcinolone acetonide for the treatment of macular edema due to branch retinal vein occlusion (BRVO). The prospective comparative nonrandomized clinical interventional study included 27 patients (27 eyes) with macular edema due to BRVO. The study group consisted of 16 patients who had accepted an intravitreal injection (IVI) of 4 mg triamcinolone acetonide. The control group included 11 patients without IVI of triamcinolone acetonide. The mean follow-up was 103.00 ± 36.24 days in the study group and 94.55 ± 36.31 days in the control group. In the study group, visual acuity measurements improved significantly ($p < 0.001$) from 0.77 ± 0.43 logarithm of minimal angle of resolution (logMAR) preoperatively to a best postoperative visual acuity of 0.44 ± 0.43 logMAR. Fourteen eyes (87.5%) gained improvement in visual acuity, with 10 eyes (62.5%) showing an increase in visual acuity of at least two Snellen lines. All 16 patients showed significant macular edema resolution in optical coherence tomography examination ($p < 0.001$) and perivascular leakage decrease in fluorescein angiography post-IVI. In the control group, baseline best-corrected visual acuity and best-corrected visual acuity during the follow-up did not vary significantly ($p = 0.294$). In conclusion, IVI of triamcinolone acetonide can lead to an increase in visual acuity and a resolution of macular edema in patients with BRVO.

Key Words: branch retinal vein occlusion, intravitreal triamcinolone acetonide, macular edema
(*Kaohsiung J Med Sci* 2006;22:321–30)

Branch retinal vein occlusion (BRVO) is a common cause of retinal vascular disease [1]. It affects males and females equally. This disease occurs most frequently between 60 and 70 years of age, and most commonly occurs at arteriovenous crossings, where the artery and vein share a common adventitial sheath [2–4]. Patients often complain of sudden onset of blurred vision or visual field defect. Fundus reveals intraretinal hemorrhage, retinal edema and, often, cotton-wool spots in a sector of retina drained by the affected vein. The most common site is at the superotemporal quadrant [5–8]. The vision-limiting complications include macular edema, macular nonperfusion, and vitreous hemorrhage from neovascularization [9–12]. The degree of macular involvement determines the level of visual impairment. Previous treatment for this condition included anticoagulant therapy [13], sheathotomy [14,15], or grid argon laser photocoagulation [16]. Anticoagulant therapy in the management of BRVO only produced limited effects and many complications during systemic administration. The therapeutic effect of sheathotomy for BRVO is still controversial and under investigation. Currently, the

Received: December 28, 2005 Accepted: April 11, 2006
Address correspondence and reprint requests to: Dr Wen-Chuan Wu, Department of Ophthalmology, Kaohsiung Medical University Chung-Ho Memorial Hospital, 100 Tzyou 1st Road, Kaohsiung 807, Taiwan.
E-mail: ufping64@ms5.hinet.net

only proven therapy for macular edema due to BRVO is macular grid argon laser photocoagulation, but the clinical outcomes are often disappointing (average improvement in vision of 1.33 Snellen lines) [16]. A recent addition to the options for treatment of macular edema due to BRVO has been intraocular injection of corticosteroids. We tried intravitreal triamcinolone acetonide to treat BRVO with associated macular edema and evaluated its effect.

Materials and Methods

Patient selection

The comparative nonrandomized clinical interventional study included 27 patients (27 eyes) with macular edema due to BRVO (only one quadrant involvement). There were 16 patients (13 men and 3 women; 16 eyes; 8 right eyes) in the study group and 11 patients (4 men and 7 women; 11 eyes; 7 right eyes) in control group. No cataract surgery was performed either before, in combination with, or after, intravitreal injection (IVI). Those patients enrolled in the study group agreed to accept an IVI of 4 mg triamcinolone acetonide. Patients who refused IVI of triamcinolone acetonide were assigned to the control group. In three eyes (27%) in the control group (cases 1, 3 and 10), retinal argon laser photocoagulation was performed for treating BRVO during follow-up, whereas others accepted only regular follow-up visit. The mean age of the study group was 55.75 ± 10.00 years and the mean age of the control group was 54.91 ± 9.26 years. The mean follow-up duration was 103.00 ± 36.24 days (range, 54–186 days) in the study group and 94.55 ± 36.31 days (range, 60–154 days) in the control group (Table 1).

We performed a series of ophthalmic examinations at study baseline and at repeated intervals afterwards as visual acuity [best-corrected visual acuity was determined from Snellen chart and calculated as the logarithm of minimal angle of resolution (logMAR)], slit-lamp biomicroscopy, pneumotonometry (Full auto Tonometer TX-F; Canon, New York, USA), ophthalmoscopy and optical coherence tomography (Stratus OCT™ III Model 3000; Zeiss Humphrey, New York, USA). For the study group, we performed the above examinations during the 1st week postinjection, twice at 2-weekly intervals, and then at routine monthly intervals. For the control group, the same examinations were performed once a month. The main outcome measures were best-corrected visual acuity, macular thickness assessed with optical coherence tomography (OCT), and postoperative complications.

Table 1. Baseline clinical characteristics

	Study group	Control group
Number	16 patients (16 eyes; 8 right eyes)	11 patients (11 eyes; 7 right eyes)
Male : Female	13 : 3	4 : 7
Mean age (yr)	55.75 ± 10.00	54.91 ± 9.26
Mean follow-up (d)	103.00 ± 36.24	94.55 ± 36.31

Surgical procedure

IVI of triamcinolone acetonide was performed under sterile conditions in the operation theater with an operation microscope. Povidone-iodine (Saint-iodine®; Patron, Gangshan, Taiwan) was applied before IVI. Then, the patient was completely draped. We used a lid speculum to open the eye. Paracentesis into the anterior chamber was performed and some aqueous fluid was aspirated by using a 26-gauge needle with a 1.0-mL tuberculin syringe to decrease the volume of the eye. The injection of 4 mg (0.1 mL) crystalline triamcinolone acetonide (Kenacort™-A; Bristol-Myers Squibb, Taipei, Taiwan) into the vitreous cavity was performed using a sharp 27-gauge needle through the pars plana, 4 mm from the limbus. After that, an antibiotic eyedrop (Tobramycin–Tobrex®; Alcon, Belgium) was applied.

Statistical analysis

Statistical calculations were performed using standard software (MS Excel 2003; Microsoft Co., Redmond, WA, USA). Data were compared using paired *t*-test. A p value <0.05 was considered to be significant.

Results

In the study group, visual acuity measurements improved significantly ($p<0.001$) from 0.77 ± 0.43 logMAR preoperatively to a best postoperative visual acuity of 0.44 ± 0.43 logMAR. Fourteen eyes (87.5%) showed visual acuity improvement and two eyes

(12.5%) remained the same during follow-up compared with study baseline. Measured in Snellen lines, 10 eyes (62.5%) showed an improvement by at least 2 Snellen lines or more (Table 2). In the control group, after argon laser photocoagulation or just continuous observation, only three eyes (27.27%) showed visual acuity improvement. Four eyes (36.36%) had the same visual acuity during follow-up compared with baseline. For four eyes (36.36%), visual acuity measurements after the baseline measurement were worse than those at the start of the study (Table 3). In the study group, the visual acuity was significantly increased postoperatively ($p < 0.001$). On the other hand, the visual acuity of the control group did not vary significantly ($p = 0.294$) between baseline best-corrected visual acuity and best-corrected visual acuity during follow-up.

In all the cases in the study group, the decline of cotton wool spots, retinal hemorrhage, and macular edema were noted during fundus examination after IVI of triamcinolone acetonide (Figure 1), and flourescein angiography showed vascular leakage decrease postoperatively (Figure 2).

OCT examination of the study group demonstrated clinical improvement in macular edema ($p < 0.001$) postoperatively (Figure 3). Preinjection foveal thickness ranged from 253 to 782 μm (mean, 525 ± 173.34 μm). Final foveal thickness ranged from 170 to 421 μm (mean, 261.50 ± 79.02 μm) with an average decrease of 46.90% in foveal thickness (Table 2). In the control group, the foveal thickness measured by OCT between baseline and final measurements did not show significant resolution ($p = 0.170$). Baseline foveal thickness ranged from 215 to 717 μm (mean, 428.27 ± 167.65 μm). Final foveal thickness ranged from 192 to 725 μm (mean, 396.91 ± 175.28 μm) with an average decrease of 6.48% in foveal thickness (Table 3).

Recurrence of macular edema and a decrease in visual acuity occurred in one case of the study group 3 months postoperatively. Subsequently, the patient accepted a second injection of triamcinolone acetonide. After that, the macular edema subsided and visual acuity improved again.

In the study group, no obvious complication was noted postoperatively, but three patients (18.75%) had ocular hypertension (intraocular pressure ≥22 mmHg) which could be controlled to a normal range by antiglaucomatous medication. No pre-existing cataracts progressed and no cataract formation was noted in the study group during follow-up and no eye required cataract surgery. No serious side effects, such as postoperative endophthalmitis or retinal detachment, occurred.

Discussion

Intravitreal triamcinolone acetonide was first used by Antcliff et al to treat uveitic cystoid macular edema [17]; consequently, many researchers tried intravitreal triamcinolone acetonide to treat macular edema caused by different diseases such as diabetic macular edema [18,19] and proliferative diabetic retinopathy [20]. Intravitreal triamcinolone acetonide has also been used to treat retinal vasculitis caused by retinal vascular diseases such as Vogt-Koyanagi-Harada syndrome [21], sympathetic ophthalmia [22], central retinal vein occlusion [23] and diabetic papillopathy [24], but studies mentioning BRVO are still rare.

Jonas et al injected 20–25 mg triamcinolone acetonide into 10 eyes of 10 patients with BRVO in the study group while 18 patients with BRVO in the control group did not receive IVI [25]. They found that visual acuity increased significantly in the study *vs*. control group during the follow-up period. There were no significant complications except that seven eyes (70%) in the study group had elevation of intraocular pressure (>21 mmHg) which was readily normalized by topical antiglaucomatous medication.

Lee and Shah examined the effect of triamcinolone acetonide IVI in six patients with cystoid macular edema secondary to BRVO [26]. Three of six eyes showed improvement in vision. All three patients without vision improvement accepted a second injection. All six eyes had an increase in visual acuity at the final follow-up visit. Five (83.3%) of six eyes showed an improvement of at least two lines of vision. OCT of five eyes (one patient was seen at a satellite office without OCT capabilities) showed a decrease of cystoid macular edema in four eyes (80%). One patient had a postoperative rise in intraocular pressure, requiring a trabeculectomy. In a retrospective study of 13 eyes of 13 patients (mean age, 68 years) who underwent IVI with 4 mg triamcinolone acetonide for macular edema due to BRVO, Cekic et al observed a significant decrease in retinal thickness in all cases with OCT, accompanied by a gain in final visual acuity in half the patients [27].

Table 2. Clinical data of study group before and after intravitreal injection of triamcinolone acetonide

Case	Age	Gender	Lens status	Preoperative data		Postoperative data					
				BCVA (logMAR)	Foveal thickness (μm)	Final BCVA (logMAR)	Final foveal thickness (μm)	Foveal thickness change (%)	Follow-up (d)	Visual acuity improvement	Visual acuity improvement ≥2 Snellen lines
1	64	F	Clear	0.7	511	0.4	198	−61.25	118	Yes	Yes
2	67	M	Mild cortical opacity	1.0	602	0.7	421	−30.07	160	Yes	No
3	52	M	Mild nuclear sclerosis + mild cortical opacity	0.7	543	0.2	195	−64.09	115	Yes	Yes
4	73	M	Mild nuclear sclerosis + mild cortical opacity	1.0	671	0.5	292	−56.48	69	Yes	Yes
5	52	M	Mild cortical opacity	0.7	478	0.7	402	−15.90	60	No	No
6	63	F	Mild nuclear sclerosis + mild cortical opacity	1.5	782	1.1	327	−58.18	115	Yes	No
7	45	M	Clear	1.0	523	0.7	312	−39.77	60	Yes	No
8	54	M	Mild cortical opacity	1.3	657	0.5	222	−66.21	127	Yes	Yes
9	44	M	Clear	0.2	293	0.1	189	−35.50	54	Yes	Yes
10	47	M	Clear	0.2	267	0.0	210	−21.35	54	Yes	Yes
11	68	F	Mild cortical opacity	1.5	748	1.5	283	−62.17	63	No	No
12	53	M	Clear	0.7	656	0.2	259	−60.52	60	Yes	Yes
13	67	M	Clear	0.4	286	0.2	177	−38.11	186	Yes	No
14	51	M	Clear	0.7	475	0.3	205	−56.84	87	Yes	Yes
15	53	M	Clear	0.1	253	0.0	170	−32.81	119	Yes	Yes
16	39	M	Clear	0.5	653	−0.1	319	−51.15	98	Yes	Yes

BCVA = best-corrected visual acuity; logMAR = logarithm of minimal angle of resolution; M = male; F = female.

Table 3. Clinical data of control group during follow-up

Case	Age	Gender	Lens status	Baseline data		Final OPD visit data					
				BCVA (logMAR)	Foveal thickness (μm)	Final BCVA (logMAR)	Final foveal thickness (μm)	Foveal thickness change (%)	Follow-up (d)	Visual acuity improvement	Visual acuity improvement ⩾2 Snellen lines
1	48	F	Clear	1.3	717	1.2	725	1.11	120	Yes	No
2	52	F	Clear	1.1	634	1.2	677	6.78	66	No	No
3	56	F	Clear	−0.1	215	0.0	227	5.58	60	No	No
4	59	M	Mild nuclear sclerosis	0.5	253	0.5	192	−24.11	65	No	No
5	56	F	Mild cortical opacity	0.7	483	0.7	352	−27.12	60	No	No
6	52	F	Mild cortical opacity	0.7	407	0.7	313	−23.10	150	No	No
7	39	M	Clear	0.2	296	0.1	232	−21.62	61	Yes	No
8	61	F	Mild cortical opacity	1.0	497	1.4	433	−12.88	121	No	No
9	75	M	Mild cortical opacity	0.1	251	0.4	367	46.22	90	No	No
10	59	M	Mild nuclear sclerosis + mild cortical opacity	1.0	571	0.1	496	−13.14	93	No	No
11	47	F	Clear	0.3	387	0.6	352	−9.04	154	Yes	No

BCVA = best-corrected visual acuity; logMAR = logarithm of minimal angle of resolution; M = male; F = female; OPD = outpatient department.

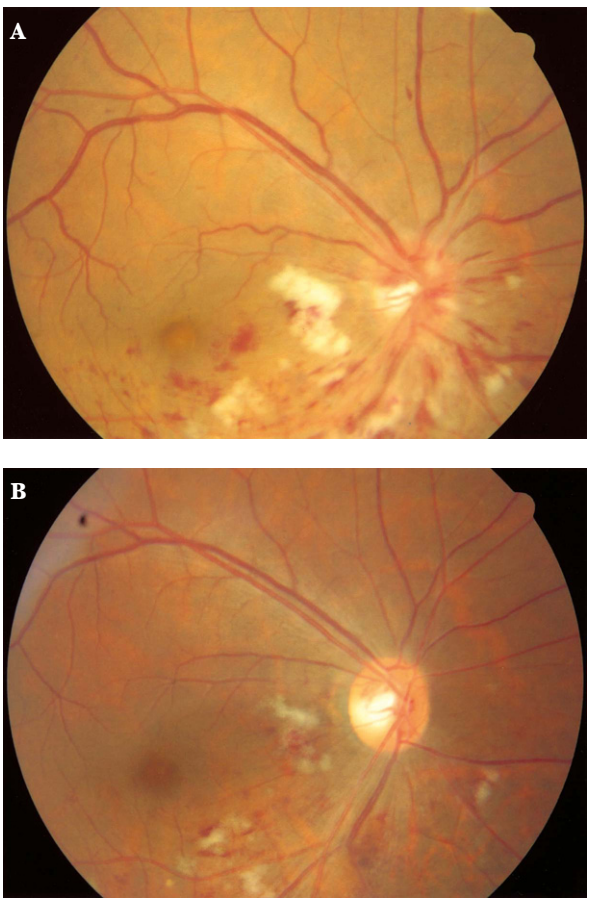

Figure 1. *Case 5 in study group: (A) Preinjection color fundus photograph of the right eye (53-year-old male) with inferiotemporal branch vein occlusion demonstrating the segmental pattern of intraretinal hemorrhage with multiple cotton-wool spots. The veins of the involved area are dilated and tortuous. (B) After 1 week of intravitreal triamcinolone injection, intraretinal hemorrhage with cotton-wool spots has subsided.*

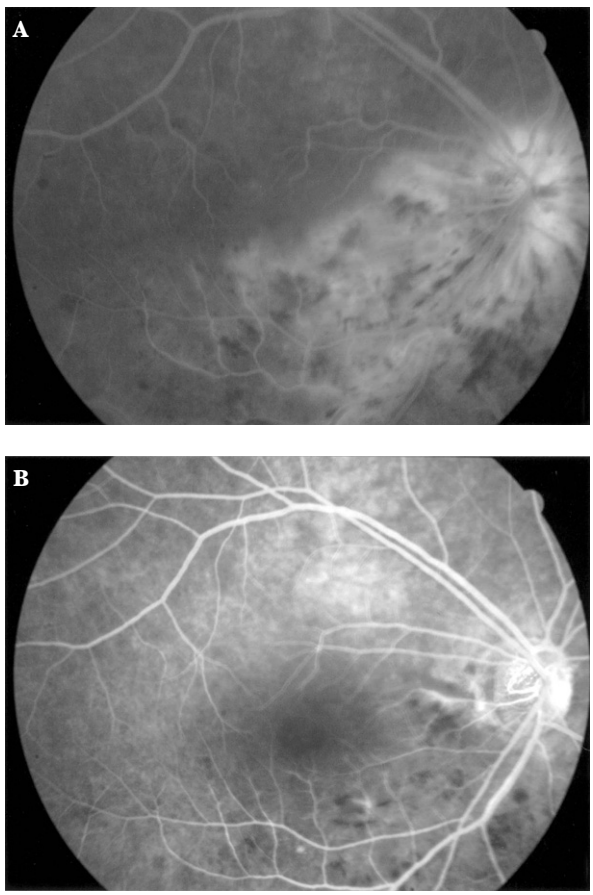

Figure 2. *Case 5 in study group: (A) Preinjection late-phase fluorescein angiography of the right eye shows a branch retinal vein occlusion (temporal lower quadrant) with macular edema. It also demonstrated severe fluorescein leakage and blockage secondary to retinal hemorrhage. (B) Resolution of macular edema with restoration of normal foveal contour were noted 1 week after intravitreal triamcinolone injection. Fluorescein leakage also decreased.*

From the results of the present study, we found that intravitreal triamcinolone acetonide may be effective in the treatment of macular edema and increase visual acuity in patients with branch retinal vein occlusion. It also appears effective in decreasing vascular leakage. All 16 patients in the study group showed a decrease in central foveal thickness in OCT examination. The patients in the study group experienced a significant increase in visual acuity ($p < 0.001$), while those in the control group did not ($p = 0.294$).

The anti-inflammatory reaction hypotheses of corticosteroid are as follows. First, it may reduce capillary permeability by increasing the activity and density of the tight junctions in the retinal capillary endothelium [28]. Second, it may inhibit the metabolic pathway of vascular endothelial growth factor, a major vascular permeability-increasing factor [29,30]. Third, it may also attenuate leukostasis and blood–retinal barrier breakdown [31]. Our hypothesis for why intravitreal triamcinolone acetonide is more effective for early BRVO is as follows.

Intraretinal intercellular structures are disrupted due to chronic macular edema. Although numerous retinal cells are damaged due to capillary nonperfusion, partial retinal cell function and structure may be reversible if early resolution of macular edema can be reached by treating with intravitreal triamcinolone acetonide.

One patient in the study group suffered from recurrence of macular edema and a decrease in visual

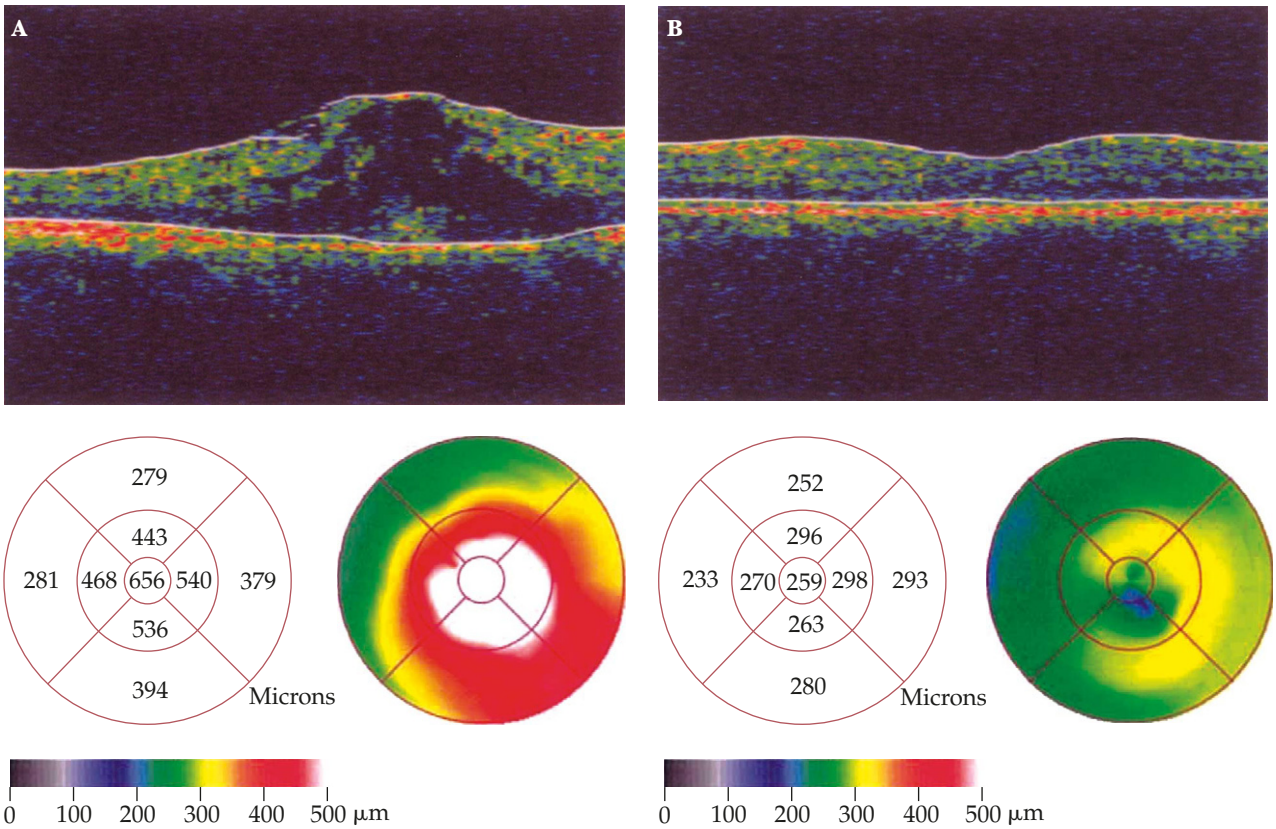

Figure 3. *Case 12 in study group: (A) Preinjection cross-sectional optical coherence tomography shows cystoid macular edema in the right eye. The retinal thickness map demonstrates marked retinal thickening. The mean central (1 mm diameter) thickness is 656 μm (normal mean central thickness is <210 μm). (B) One month postinjection, optical coherence tomography demonstrates decline of the cystoid macular edema. The mean central thickness is 259 μm.*

acuity 3 months postoperatively. Reinjection of triamcinolone acetonide led to a visual acuity improvement and a reduction in macular thickness. This observation is similar to the study reported by Salinas-Alaman et al, whose patient received a second injection after 3 months [32].

In our case series, no toxic effects of triamcinolone acetonide on the retina and optic nerve were noted in the study group. Previous clinical and experimental studies also demonstrated the safety of using intraocular steroid [33–37].

The most common side effect reported after IVI of triamcinolone acetonide is a rise in intraocular pressure. In our study, ocular hypertension (intraocular pressure ⩾22 mmHg) occurred in three eyes (18.75%). This problem could be controlled by topical antiglaucomatous treatment. Other studies also revealed similar observations [38–41].

An increase in cataract formation has been described in patients after IVI of triamcinolone acetonide. Progression of cataract was observed in six of 26 eyes in a study by Challa et al [42] and in four of seven eyes in a study by Danis et al [43]. Jonas et al observed a progression in cataract in one of their patients, although it was not a significant change [44]. Park et al found a progression in one of 10 eyes [45], and Martidis et al noted a similar finding in one of eight eyes [46]. However, in our study, we did not observe any cataract formation or deterioration in pre-existing cataracts in the study group.

Intraocular infection is one of the most severe complications after triamcinolone acetonide IVI. Moshfeghi et al [47] and Nelson et al [48] observed infectious endophthalmitis in their cases after intravitreal triamcinolone acetonide injection. Non-infectious endophthalmitis has also been reported by Nelson et al [48], Moshfeghi et al [49], Roth et al [50], Chiu et al [51] and Sutter and Gillies [52]. In none of the eyes in our study group were postoperative infectious or non-infectious endophthalmitis observed.

The advantages of intravitreal triamcinolone acetonide include avoiding systemic adverse effects, having simultaneously high concentrations of drug at the site of action, and no toxic effect on intraocular tissue.

The major limitations of our study are nonrandomized selection between study and control groups, small number of patients in both study and control groups, and the relatively short follow-up period. Further study with longer follow-up and a larger series is warranted to assess the long-term efficacy and safety of intravitreal triamcinolone acetonide.

Conclusion

The application of triamcinolone acetonide IVI showed a significant increase in visual acuity with a decrease in macular edema and vascular leakage in patients with BRVO. A side effect observed in our study was an elevation of intraocular pressure, which can be readily resolved by topical antiglaucomatous medication. The results of this study suggest that intravitreal triamcinolone acetonide may be an alternative therapeutic option for patients with macular edema due to BRVO.

References

1. Branch Vein Occlusion Study Group. Argon laser scatter photocoagulation for prevention of neovascularization and vitreous hemorrhage in branch vein occlusion. *Arch Ophthalmol* 1986;104:34–41.
2. Jensen VA. Clinical studies of tributary thrombosis in central retinal vein. *Acta Ophthalmol Suppl* 1936;10: 1–193.
3. Duker JS, Brown GC. Anterior location of the crossing artery in branch retinal vein obstruction. *Arch Ophthalmol* 1989;107:998–1000.
4. Weinberg D, Dodwell DG, Fern SA. Anatomy of arteriovenous crossings in branch retinal vein occlusion. *Am J Ophthalmol* 1990;109:298–302.
5. Christoffersen NL, Larsen M. Pathophysiology and hemodynamics of branch retinal vein occlusion. *Ophthalmology* 1999;106:2054–62.
6. Becker B, Post LT Jr. Retinal vein occlusion. Clinical and experimental observations. *Am J Ophthalmol* 1951; 34:677–86.
7. Blankenship GW, Okun E. Retinal tributary vein occlusion. History and management by photocoagulation. *Arch Ophthalmol* 1973;89:363–8.
8. Gutman FA, Zegarra H. The natural course of temporal retinal branch vein occlusion. *Trans Am Acad Ophthalmol Otolaryngol* 1974;78:OP178–92.
9. Fekrat S, Goldberg MF, Finkelstein D. Laser-induced chorioretinal venous anastomosis for nonischemic central or branch retinal vein occlusion. *Arch Ophthalmol* 1998;116:43–52.
10. Finkelstein D. Ischemic macular edema: recognition and favorable natural history in branch vein occlusion. *Arch Ophthalmol* 1992;110:1427–34.
11. Finkelstein D, Clarkson JG and The Branch Vein Occlusion Study Group. *Branch and Central Retinal Vein Occlusions. Focal Points 1987: Clinical Modules for Ophthalmologists 5 (module 12)*, no 3, American Academy of Ophthalmology.
12. Glitinan FA, Zegarra H. Macular edema secondary to occlusion of the retinal veins. *Surv Ophthalmol* 1984; 28:462–70.
13. Hattenbach LO, Steinkamp G, Scharrer I, et al. Fibrinolytic therapy with low-dose recombinant tissue plasminogen activator in retinal vein occlusion. *Ophthalmologica* 1998;212:394–8.
14. Opremcak EM, Bruce RA. Surgical decompression of branch retinal vein occlusion via arteriovenous crossing sheathotomy: a prospective review of 15 cases. *Retina* 1999;19:1–5.
15. Cahill MT, Kaiser PK, Sears JE, et al. The effect of arteriovenous sheathotomy on cystoid macular oedema secondary to branch retinal vein occlusion. *Br J Ophthalmol* 2003;87:1329–32.
16. Branch Vein Occlusion Study Group. Argon laser photocoagulation for macular edema in branch vein occlusion. *Am J Ophthalmol* 1984;98:271–82.
17. Antcliff RJ, Spalton DJ, Stanford MR, et al. Intravitreal triamcinolone for uveitic cystoid macular edema: an optical coherence tomography study. *Ophthalmology* 2001;108:765–72.
18. Jonas JB, Sofker A. Intraocular injection of crystalline cortisone as adjunctive treatment of diabetic macular edema. *Am J Ophthalmol* 2001;132:425–7.
19. Martidis A, Duker JS, Greenberg PB, et al. Intravitreal triamcinolone for refractory diabetic macular edema. *Ophthalmology* 2002;109:920–7.
20. Jonas JB, Sofker A. Intravitreal injection of crystalline cortisone as adjunctive treatment of proliferative diabetic retinopathy. *Am J Ophthalmol* 2001;131:468–71.
21. Andrade RE, Muccioli C, Farah ME, et al. Intravitreal triamcinolone in the treatment of serous retinal detachment in Vogt-Koyanagi-Harada syndrome. *Am J Ophthalmol* 2004;137:572–4.
22. Jonas JB. Intravitreal triamcinolone acetonide for treatment of sympathetic ophthalmia. *Am J Ophthalmol* 2004;137:367–8.
23. Greenberg PB, Martidis A, Rogers AH, et al. Intravitreal triamcinolone acetonide for macular oedema due to central retinal vein occlusion. *Br J Ophthalmol* 2002;86:247–8.

24. Al-Haddad CE, Jurdi FA, Bashshur ZF. Intravitreal triamcinolone acetonide for the management of diabetic papillopathy. *Am J Ophthalmol* 2004;137:1151–3.
25. Jonas JB, Akkoyun I, Kamppeter B, et al. Branch retinal vein occlusion treated by intravitreal triamcinolone acetonide. *Eye* 2005;19:65–71.
26. Lee H, Shah GK. Intravitreal triamcinolone as primary treatment of cystoid macular edema secondary to branch retinal vein occlusion. *Retina* 2005;25:551–5.
27. Cekic O, Chang S, Tseng JJ, et al. Intravitreal triamcinolone injection for treatment of macular edema secondary to branch retinal vein occlusion. *Retina* 2005; 25:851–5.
28. Antonetti DA, Wolpert EB, DeMaio L, et al. Hydrocortisone decrease retinal endothelial cell water and solute flux coincident with increased content and decreased phosphorylation of occludin. *J Neurochem* 2002;80: 667–77.
29. Kompella UB, Bandi N, Ayalasomayajula SP. Subconjunctival nano- and microparticles sustain retinal delivery of budesonide, a corticosteroid capable of inhibiting VEGF expression. *Invest Ophthalmol Vis Sci* 2003;44:1192–201.
30. Fischer S, Renz D, Schaper W, et al. *In vitro* effects of dexamethasone on hypoxia-induced hyperpermeability and expression of vascular endothelial growth factor. *Eur J Pharmacol* 2001;411:231–43.
31. Tamura H, Miyamoto K, Kiryu J, et al. Intravitreal injection of corticosteroid attenuates leukostasis and vascular leakage in experimental diabetic retina. *Invest Ophthalmol Vis Sci* 2005;46:1440–4.
32. Salinas-Alaman A, Garcia-Layana A, Sadaba-Echarri LM, et al. Branch retinal vein occlusion treated by intravitreal triamcinolone. *Arch Soc Esp Oftalmol* 2005;80:463–5.
33. Young S, Larkin G, Branley M, et al. Safety and efficacy of intravitreal triamcinolone for cystoid macular oedema in uveitis. *Clin Exp Ophthalmol* 2001;29:2–6.
34. McCuen II BW, Bessler M, Tano Y, et al. The lack of toxicity of intravitreally administered triamcinolone acetonide. *Am J Ophthalmol* 1981;91:785–8.
35. Hida T, Chandler D, Arena JE, et al. Experimental and clinical observations of the intraocular toxicity of commercial corticosteroid preparations. *Am J Ophthalmol* 1986;101:190–5.
36. Kwak HW, D'Amico DJ. Evaluation of the retinal toxicity and pharmacokinetics of dexamethasone after intravitreal injection. *Arch Ophthalmol* 1992;110: 259–66.
37. Kivilcim M, Peyman GA, EI-Dessouky ES, et al. Retinal toxicity of triamcinolone acetonide in silicone-filled eyes. *Ophthalmic Surg Lasers* 2000;31:474–8.
38. Martidis A, Duker JS, Greenberg PB, et al. Intravitreal triamcinolone for refractory diabetic macular oedema. *Ophthalmology* 2002;109:920–7.
39. Wingate RJ, Beaumont PE. Intravitreal triamcinolone and elevated intraocular pressure. *Aust N Z J Ophthalmol* 1999;27:431–2.
40. Jonas JB, Kreissig I, Degenring R. Intraocular pressure after intravitreal injection of triamcinolone acetonide. *Br J Ophthalmol* 2003;87:24–7.
41. Bakri SJ, Beer PM. The effect of intravitreal triamcinolone acetonide on intraocular pressure. *Ophthalmic Surg Lasers Imaging* 2003;34:386–90.
42. Challa JK, Gillies MC, Penfold PL, et al. Exudative macular degeneration and intravitreal triamcinolone: 18-month follow-up. *Aust N Z J Ophthalmol* 1998;26: 277–81.
43. Danis RP, Ciulla TA, Pratt LM, et al. Intravitreal triamcinolone acetonide in exudative age-related macular degeneration. *Retina* 2000;20:244–50.
44. Jonas JB, Kreissig I, Sofker A, et al. Intravitreal injection of triamcinolone for diffuse diabetic macular edema. *Arch Ophthalmol* 2003;121:57–61.
45. Park CH, Jaffe GJ, Fejrat S. Intravitreal triamcinolone acetonide in eyes with cystoid macular oedema associated with central retinal vein occlusion. *Am J Ophthalmol* 2003;136:419–25.
46. Martidis A, Duker JS, Greenberg PB, et al. Intravitreal triamcinolone for refractory diabetic macular oedema. *Ophthalmology* 2002;109:920–7.
47. Moshfeghi DM, Kaiser PK, Scott IU, et al. Acute endophthalmitis following intravitreal triamcinolone acetonide injection. *Am J Ophthalmol* 2003;136:791–6.
48. Nelson ML, Tennant MTS, Sivalingam A, et al. Infectious and presumed non-infectious endophthalmitis after intravitreal triamcinolone acetonide injection. *Retina* 2003;23:686–91.
49. Moshfeghi AA, Scott IU, Flynn HW, et al. Pseudohypopyon after intravitreal triamcinolone acetonide injection for cystoid macular edema. *Am J Ophthalmol* 2004;138:489–92.
50. Roth DB, Chieh J, Spirn MJ, et al. Non-infectious endophthalmitis associated with intravitreal triamcinolone injection. *Arch Ophthalmol* 2003;121:1279–82.
51. Chiu YT, Tsai YY, Lin JM. Noninfectious hypopyon after intravitreal triamcinolone acetonide injection for central retinal vein occlusion: a case report. *Kaohsiung J Med Sci* 2005;21:466–9.
52. Sutter FKP, Gillies MC. Pseudoendophthalmitis after intravitreal injection of triamcinolone. *Br J Ophthalmol* 2003;87:972–4.

利用玻璃體內注射 Triamcinolone Acetonide 治療因視網膜分枝靜脈阻塞而導致的黃斑部水腫

鄭凱駿　吳文權

高雄醫學大學附設中和紀念醫院 眼科

為了評估利用玻璃體內注射 triamcinolone acetonide 來治療因視網膜分枝靜脈阻塞而導致的黃斑部水腫，我們設計一個前瞻性、比較性、非隨機性的臨床實驗。本實驗包含了 27 個有視網膜分枝靜脈阻塞合併黃斑部水腫的病人 (共 27 顆眼睛)，實驗組有 16 個病人，每位病人都接受了 4 mg triamcinolone acetonide 的玻璃體內注射，對照組有 11 個病人，每位病人都拒絕接受 triamcinolone acetonide 的玻璃體內注射。實驗組的病人其平均追蹤天數為 103.00 ± 36.24 天，對照組的病人其平均追蹤天數為 94.55 ± 36.31 天。實驗組的病人其平均視力從術前的 0.77 ± 0.43 logMAR 進步到術後的 0.44 ± 0.43 logMAR，其視力有明顯的改善 ($p < 0.001$)，14 顆眼睛 (87.5%) 其視力有進步，其中有 10 顆眼睛 (62.5%) 其視力以 Snellen 視力表測量甚至進步至少 2 行以上。實驗組中的 16 個病人其黃斑部水腫及螢光眼底攝影的螢光劑滲漏情形在術後都有明顯改善。對照組的病人其原本的最佳矯正視力與追蹤期間的最佳矯正視力相比並沒有明顯的差異 ($p = 0.294$)。利用玻璃體內注射 triamcinolone acetonide 來治療視網膜分枝靜脈阻塞的病人，可以改善病人的視力，使黃斑部水腫消退。

關鍵詞：視網膜分枝靜脈阻塞，玻璃體內注射triamcinolone acetonide，黃斑部水腫

(高雄醫誌 2006;22:321－30)

收文日期：94 年 12 月 28 日

接受刊載：95 年 4 月 11 日

通訊作者：吳文權醫師

高雄醫學大學附設中和紀念醫院眼科

高雄市三民區自由一路100號