

Intravitreal triamcinolone acetonide in refractory pseudophakic cystoid macular edema: Functional and anatomic results

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PURPOSE. To evaluate safety and efficacy of intravitreal triamcinolone acetonide (TAAC) injections in the treatment of refractory pseudophakic cystoid macular edema (CME).

METHODS. Seven eyes of six patients (age range: 50–74) with pseudophakic CME resistant to standard treatment received intravitreal injections of 4 mg of TAAC with all vehicle. Mean preinjection duration of CME was 18.3 months. A mean of 2.1 ± 1.2 (range 1 to 4) treatments were performed in four eyes (57.1%) when visual acuity deteriorated towards baseline levels. Visual acuity assessment, optical coherence tomography (OCT), and fluorescein angiography (FFA) were performed pre- and postoperatively to evaluate results of TAAC injections. Intraocular pressure (IOP) and complications related to treatment were assessed.

RESULTS. After 11.1 ± 3.9 months, mean best-corrected visual acuity (BCVA) increased ($p=0.019$) from 20/132 to a best value of 20/38. Mean macular thickness decreased from 517.29 ± 146.98 μm to a best value of 263.71 ± 83.13 μm ($p=0.0018$). Area of fluorescein leakage decreased ($p<0.0001$) from 11.84 ± 0.93 mm^2 at baseline to a minimal value of 3.86 ± 0.98 mm^2 . The anatomic and functional improvement appeared after 1 month from the intravitreal injection and persisted through at least 3 months of follow-up. At the end of follow-up BCVA, macular thickness, and area of fluorescein leakage did not differ from baseline. Four eyes (57.1%) developed IOP values higher than 21 mmHg, controlled by topical treatment. Two patients developed an endophthalmitis-like reaction.

CONCLUSIONS. Intravitreal TAAC was relatively safe and effective in resistant cases of pseudophakic CME with a temporary beneficial effect on visual acuity and macular edema. (*Eur J Ophthalmol* 2005; 15: 89-95)

KEY WORDS. Triamcinolone acetonide, Pseudophakic cystoid macular edema

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INTRODUCTION

While acute pseudophakic cystoid macular edema (CME) may resolve spontaneously, chronic visually significant pseudophakic CME remains difficult to treat and cannot respond to conventional medical therapies. Several treatments have been proposed to control severe postoperative CME (1-6). Nevertheless, in spite of all therapeutic options, resistant cases of CME have not been eliminated.

A new therapeutic approach to persistent recalcitrant CME in pseudophakic eye is the intravitreal injection of triamcinolone acetonide (TAAC) (6-8), a potent and relatively insoluble steroid reported to be effective in the prevention or treatment of macular edema refractory to conventional therapy (9-11).

The purpose of this pilot study was to evaluate safety and functional and anatomic efficacy of multiple intravitreal injection of TAAC for the treatment of refractory pseudophakic chronic CME.

METHODS

The patients' demographics are shown in Table I. Seven pseudophakic eyes of six consecutive patients (all men) were included in this study. The mean age of patients was 60±10.1 years (range: 50–74). They met the following criteria: 1) diagnosis of chronic CME with intraretinal fluorescein angiographic cystoid spaces and optic nerve head staining; 2) at least a 6-month history of decreased best-corrected visual acuity (BCVA) after ophthalmic surgery; 3) unresponsiveness to local and systemic nonsteroidal anti-inflammatory drugs and corticosteroids for at least 6 months.

Mean duration of CME before intravitreal injection was 18.4 months (range 6 to 36 months). General health of all patients was good except for three patients, one with non-insulin dependent diabetes mellitus, and the other two with hypertension and heart arrhythmia, and both receiving cardiologic therapy. One patient with complicated cataract surgery had the anterior vitreous

incarcerated in the corneoscleral surgical wound and had already undergone a pars plana vitrectomy. Both eyes of Patient 3 had received pars plana vitrectomy to repair a retinal detachment. No eye had a history of ocular hypertension or glaucoma. The patients were fully informed about the experimental character of the therapy and had signed an informed consent.

After aseptic preparation with povidone iodine 5% and topical anesthesia (oxybuprocaine), all eyes received an intravitreal injection of 4 mg (0.1 ml) of commercially available TAAC (Kenacort 40 mg/ml; Bristol-Myers Squibb), through the inferotemporal pars plana 3.5 mm from the corneal limbus, using a 27-gauge needle. After slow injection, indirect ophthalmoscopy assessed proper intravitreal localization of the suspension and perfusion of the optic nerve. In all cases an anterior chamber paracentesis was performed. Post-operatively, all patients continued to use prednisolone acetate drops 1% four times daily for 1 week.

BCVA using the ETDRS chart, intraocular pressure

TABLE I - PATIENT CHARACTERISTICS

Case	Age, yr	Eye	Lens	Other ocular surgeries*	Duration of CME, mos	Other ocular pathology†	Treatment history before intravitreal TAAC	Systemic diseases
1	74	OD	PC IOL	Macular grid	6	NPDR	PF	DM
2	71	OD	Scleral fixation; open PC	PPV	18	Vitreous incarcerated in surgical wound	PF	HTN
3‡	55	OD	PC IOL	PPV	34		Systemic steroids	HTN
		OS	PC IOL	PPV	36		Systemic steroids	
4	50	OS	PC IOL; open PC	Nd:YAG capsulotomy	9		PF	
5	58	OS	PC IOL		7		PF	
6	52	OS	PC IOL			20		PF

All subjects were male

*Other than cataract surgery

†Other than CME

‡Patient 3 underwent TAAC injection in both eyes.

CME = Cystoid macular edema; TAAC = Triamcinolone acetonide; OD = Right eye; PC IOL = Posterior chamber intraocular lens; NPDR = Nonproliferative diabetic retinopathy; PF = Prednisolone acetate 1%; DM = Diabetes mellitus; PC = Posterior capsulotomy; PPV = Pars plana vitrectomy; HTN = Hypertension; OS = Left eye.

(IOP), indirect ophthalmoscopy, color fundus photography, fluorescein angiography (FA), and optical coherence tomography (OCT) were evaluated at each visit. Eyes were routinely examined the first postoperative day and the response to treatment was monitored functionally and anatomically weekly for the first 4 weeks after injection, and at monthly intervals thereafter. All seven eyes completed 6 months of follow-up, and three eyes had a follow-up of 12 months or more. Patients were treated with topical antiglaucoma medications if the IOP became elevated over 25 mmHg.

FAs were obtained with a digital scanning laser ophthalmoscope (Heidelberg Retina Angiograph, Heidelberg Engineering GmbH, Heidelberg, Germany). The size of the area with fluorescein cystoid pooling was circled and measured in the late phase of the angiography.

OCT scanning (OCT 2000 scanner, Humphrey Instruments, San Leandro, CA) was performed by the same operator in all cases (C.F.). At each visit, six linear scans 5.00 mm in length were taken at 0, 30, 60, 90, 120, and 150 degrees centered on fixation.

RESULTS

The mean follow-up time was 11.1±3.9 months (range 6 to 17). Treatment characteristics of the enrolled eyes are summarized in. Mean BCVA increased significantly (t-test; p=0.019) from 20/132 (0.82 ± 0.48 logMAR; range 0.2–1.48) to 20/38 (0.28 ± 0.24 logMAR; range 0–0.7). Compared with the initial BCVA, VA at

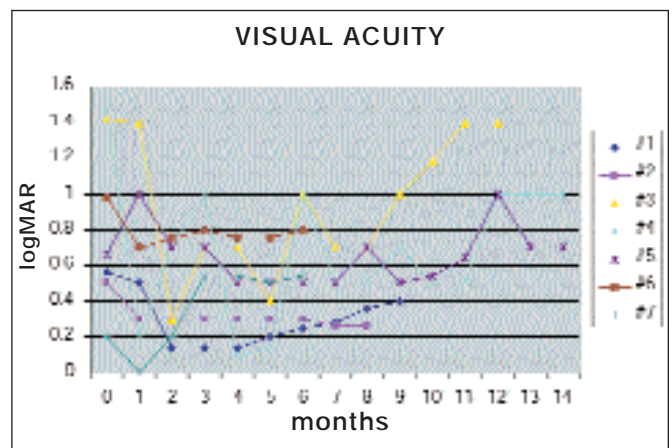


Fig. 1 - Diagram showing the difference between preinjection and postinjection visual acuity after a mean follow-up of 11.1 months.

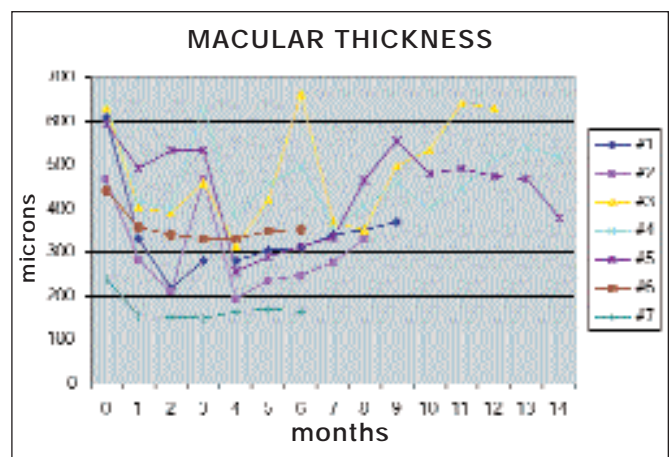


Fig. 2 - Diagram showing the difference between preinjection and postinjection macular thickness measured by optical coherence tomography after a mean follow-up of 11.1 months.

TABLE II - RESULTS OF INTRAVITREAL TRIAMCINOLONE INJECTION FOR REFRACTORY PSEUDOPHAKIC MACULAR EDEMA

Eye n.	Follow-up (mt)	No. inject	VA (Snellen)		FA edema (mm ²)		OCT thickness (mm)		Intraocular pressure (mmHg)			
			Pre	Post	Pre	Post	Pre	Post	Pre	1 Mo	3 Mo	6 Mo
1	9	1	20/73	20/50	11.08	9.1	609	368	14	14	22	15
2	9	2	20/63	20/36	10.78	9.8	466	330	12	14	14	17
3	12	3	20/526	20/502	13.27	13.3	632	628	12	13	15	22
4	14	4	20/604	20/200	12.25	12.6	641	517	16	14	20	28
5	17	3	20/91	20/100	11.11	10.3	594	379	18	20	22	14
6	6	1	20/191	20/126	11.68	5.3	442	331	11	15	15	15
7	6	1	20/32	20/69	12.69	4.1	237	150	13	16	16	16
	11.1±3.9	2.1±1.2	20/132	20/105 (p=0.67)	11.8±0.9	9.21±3.44 (p=0.0756)	517.2±146.9	386.4±15.6 (p=0.126)	13.7±2.5	15.1±2.3 (p=0.29)	17.7±3.5 (p=0.029)	18.1±5 (p=0.06)

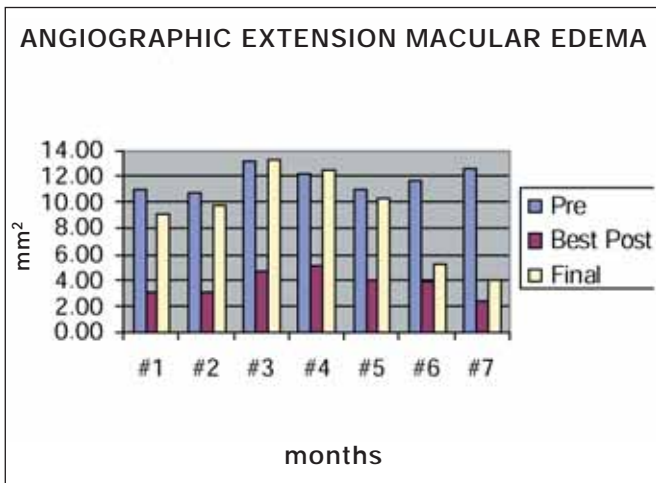


Fig. 3 - Diagram showing the difference between preinjection and postinjection fluorescein leakage after a mean follow-up of 11.1 months.

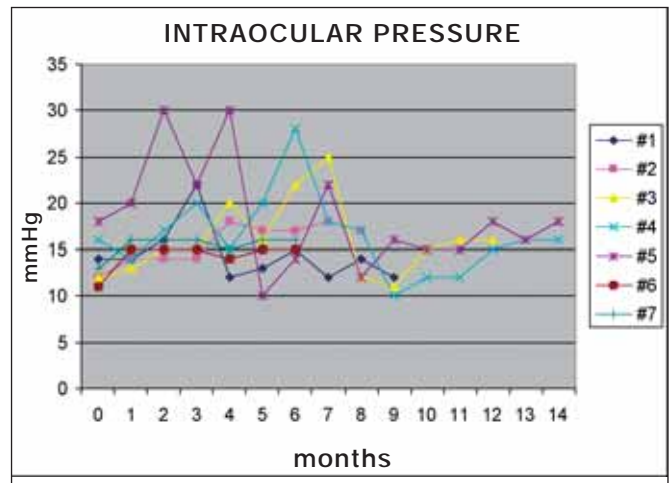


Fig. 4 - Diagram showing the fluctuation of the intraocular pressure in each patient after intravitreal injection of 4 mg of triamcinolone acetonide during a mean follow-up of 11.1 months.

the end of the follow-up ((20/105 (0.72 ± 0.38 logMAR; range 0.26–1.)) did not improve significantly (t-test, p=0.676). Mean increase in VA at the end of follow-up was 0.10 logMAR (Fig. 1). Mean number of reinjections was 2.1 ± 1.2 (range 1 to 4). In four eyes of three patients, within 12 ± 0.8 weeks (range 11–13), VA deteriorated toward the baseline level due to macular edema recurrence requiring reinjection. These eyes were given repeated injections two to four times.

The mean interval between the second and the third injection was 20 weeks (three eyes); the interval between the third and the fourth injection was 12 weeks (one eye). The interval was similar in vitrectomized and nonvitrectomized patients.

Macular thickness decreased significantly, in less than 4 weeks (range 2–8), from a mean pretreatment value of 517.3 ± 146.9 μm to a mean best post-treatment value of 236.7 ± 83.1 μm (t-test, p=0.0018) (Fig. 2), corresponding to a 54.2% reduction. Complete resolution of the subretinal fluid in macular area was observed in four eyes, and complete resolution of the cysts was achieved in three eyes. Compared with the initial macular thickness, foveal height at the end of the follow-up (391.14 ± 146.7 μm; range 150–628) did not improve significantly (t-test, p=0.134). At the end of the follow-up period, complete resolution of the subretinal fluid in macular area and complete resolution of the cysts were achieved in one eye.

Area of fluorescein leakage on the angiograms decreased significantly (p < 0.0001) from 11.84 ± 0.93

mm² (range 10.78–13.27) at baseline to a minimal value of 3.86 ± 0.98 mm² (range 2.5–5.20 mm²), but it did not significantly decrease (p=0.0756) at the end of follow-up (9.21 ± 3.44; range 4.1–13.3) (Fig. 3).

Mean IOP increased significantly (t-test, p=0.0043) from 13.7 ± 2.5 mmHg at baseline to a maximal value of 29.3 ± 11.47 mmHg (range 16–48) during the first month of follow-up. In all eyes, IOP was controlled by topical antiglaucomatous medication decreasing to 15.1 ± 2.3 mmHg (p=0.29) at the end of the first month. Mean IOP measurements taken at the end of the follow-up period were 15.71 ± 1.89 and were not significantly different (p=0.1169) from those measured at baseline (Fig. 4). During the study period, IOP was higher than 21 mmHg in 4 eyes (57.1%).

In the right eye of Patient 3 2 days after the injection, and in eye 4 the day after two of the injections received, crystals of TAAC appeared in the anterior chamber, with no flare or fibrin. In eye 3, the crystals were stratified on peripheral endothelium inferiorly (Fig. 5); in eye 4 they simulated a hypopyon (Fig. 6). Both patients had mild ocular discomfort and a decrease in VA. Cornea was mildly edematous, and retina examination was impossible due to the presence of white crystals in the anterior vitreous gel. IOP was increased (42 and 48 mmHg, respectively), and VA was at hand motion level. A B-scan ultrasound confirmed the presence of diffuse fine dots in the vitreous cavity. Suspecting an infective process, local and systemic therapy was started.

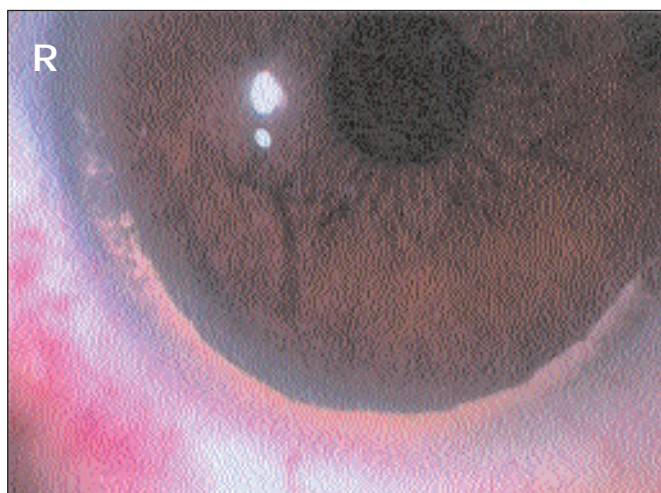


Fig. 5 - Triamcinolone acetonide crystals were stratified on peripheral endothelium inferiorly without evident signs of infections.

Two days later, the corneal clarity returned, IOP was within normal limits, and the eye became quiet. Within 7 days, BVCA improved to preinjection level, and hypopyon disappeared from the anterior chamber; retinal biomicroscopy revealed TAAC deposit and persisting CME. Both the patients had a largely interrupted posterior capsule.

In no eye did we observe TAAC toxic effects or complications such as retinal detachment, vitreous hemorrhages, vitreoretinal fibrovascular proliferation, or endophthalmitis.

All the eyes that had undergone PPV before enrollment in this study required multiple TAAC injections after surgery in order to maintain BCVA and reduce CME. The effect duration of TAAC injections did not differ from eyes that did not undergo PPV. Patient 4 after PPV and internal limiting membrane (ILM) peeling did not require further intervention.

DISCUSSION

Triamcinolone acetonide is a minimally water-soluble steroid injected in a suspension form. Theoretically, the intravitreal route allows better bioavailability than systemic and local administration (12-17), avoiding systemic side effects. Its mean intraocular measurable concentration after intravitreal injection can last for about 3 months because of its long clearance time (18-20).



Fig. 6 - After triamcinolone acetonide intravitreal injection a hypopyon-like condition was observed. Cornea was mildly edematous, anterior chamber flare was present, and retina examination was impossible due to the presence of white crystals in the anterior vitreous gel.

Recently, Conway and coauthors (6) performed intravitreal injection of 1 mg of TAAC for refractory chronic pseudophakic CME in eight eyes of eight patients, followed for 31 weeks (range 12-51). They found it safe and effective in improving VA and in reducing macular edema, as evaluated by means of contact lens biomicroscopy and FA.

Benhamou et al (7) published a report on a series of three eyes of three patients with longstanding pseudophakic CME who were treated with 8 mg of intravitreal TAAC. They noticed a dramatic decrease in macular thickness by OCT in all eyes, by about 50%, with an improvement in VA.

Jonas et al (8) described the results of 25 mg of intravitreal TAAC in five eyes of five patients. After 6.6 ± 4.1 months (range 1.03-11.1) VA improved significantly, without decreasing toward the end of the follow-up period.

All the eyes enrolled in the present study have been followed for a minimum of 6 months, and the dosage of intravitreal triamcinolone was 4 mg, the most commonly used. In order to evaluate more precisely the extension of CME in the present study, we used objective repeatable measures, such as area of fluorescein leakage, and retinal thickness as determined with OCT.

OCT is more sensitive than contact lens biomicroscopy, and as good as FA for detecting macular edema, offering the advantage over FA in evaluating macular thickening, which appears to be more closely related to VA (21), and in demonstrating the distribution of fluid with-

in and underneath the retina (22-24). It has a high degree of reproducibility and has the advantages of being noninvasive, comfortable, safe, and repeatable (25, 26). The anatomic and functional improvement appeared after 1 month from the intravitreal injection and persisted through at least 3 months of follow-up. In our series, all eyes demonstrated a significant functional response to intravitreal injections of TAAC, but BCVA did not change significantly from baseline at the end of the follow-up period. In the same way, retinal thickness and area of fluorescein leakage decreased rapidly and significantly from the baseline, by 54.2% and 67.4%, respectively, returning toward baseline levels at the end of follow-up.

Macular edema recurred in four eyes after a mean of 12 ± 0.8 weeks of previous injection. For this reason, a new injection of intravitreal TAAC was repeated for a mean of 2.1 ± 1.2 times (range 1 to 4). This is consistent with the reported length of measurable concentration of 4 mg of intravitreal TAAC in humans – 93 ± 28 days (27). We did not observe a trend toward resistance in patients who underwent repeated injections.

No adverse effects such as retinal detachment, vitreous hemorrhage, or culture-proven infectious endophthalmitis occurred in our study. In the early postoperative period, two patients with a large interruption in posterior capsule showed a presumed sterile pseudoendophthalmitis. One of these patients had it twice. This condition, more than a true inflammatory reaction (immune or infectious), might have been caused by corticosteroid particles that had passed into the anterior chamber through the interrupted posterior capsule. This is in agreement with the results of vitreous tap in a similar case where large numbers of small TAAC crystals but very few inflammatory cells were found (28). In the total of 15 injections performed in this study it occurred three times (20%). This is far more than the 7 cases in 440 procedures (1.6%) published by Nelson et al (29). The high percentage of pseudoendophthalmitis in our series might be explained by the presence in all the eyes involved of a large communication between the anterior and posterior segment.

We observed IOP elevation in excess of 21 mmHg after about 1 month from intravitreal injection in five eyes, which was controlled by topical antihypertensive therapy, and IOP measurements returned in all eyes to a normotensive level at the end of follow-up.

We injected 4 mg of TAAC without separating it from

its vehicle, without evident retinal toxicity. This is in agreement with animal studies proving TAAC and its vehicle to be nontoxic to the retina (20, 30), and with the lack of toxicity of TAAC in doses of 2 to 20 mg in vitrectomized and nonvitrectomized human eyes (6-11).

In our series, four eyes had undergone a PPV. In two eyes it was necessary to repair a retinal detachment, and in one eye to remove vitreous incarceration to the corneoscleral wound before the TAAC injection. In the remaining eye, PPV was deemed necessary during the study to peel an epiretinal membrane and the ILM, which were thought co-responsible for the CME. Nevertheless, in our and others' experience (31), pars plana vitrectomy, even when associated with ILM peeling, did not lead to the disappearance of CME. Reinjections were still needed postoperatively and the length of the effect of TAAC injections did not differ from eyes that did not undergo PPV.

The current study is limited by the relatively small number of patients and the absence of a control group. Controlled trials are needed to assess the most appropriate dose of intravitreal TAAC, the long-term VA benefit, and the possible complications. Given the usually unfavorable visual outcome, the goal of the management of refractory cases of pseudophakic CME is to maintain better VA for a longer period. Intravitreal TAAC is a promising, low-cost, non-toxic, and relatively safe therapeutic method for chronic pseudophakic CME that fails to respond to conventional medical treatments, preserving the possibility of other forms of treatment. The known potential adverse effects of TAAC and the observed complications, rapidly reversible, may be acceptable given the risk of irreversible visual loss from chronic CME.

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