

Intraocular Pressure Elevation after Intravitreal Triamcinolone Acetonide Injection

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Purpose: To report on intraocular pressure (IOP) after intravitreal injections of triamcinolone acetonide.

Design: Meta-analysis of previously reported data and case series studies.

Participants: The study included 272 patients (305 eyes) receiving an intravitreal injection of approximately 20 mg triamcinolone acetonide as treatment for diffuse diabetic macular edema (n = 84 patients), exudative age-related macular degeneration (n = 181 patients), retinal vein occlusions (n = 20 patients), uveitis (n = 9), pseudophakic cystoid macular edema (n = 6), and other reasons (n = 5). Mean follow-up was 10.4 ± 6.7 months (median, 7.9 months; range, 3.0–35.7 months).

Intervention: Intravitreal injection of approximately 20 mg triamcinolone acetonide.

Main Outcome Measure: Intraocular pressure.

Results: Intraocular pressure readings higher than 21 mmHg, 30 mmHg, 35 mmHg, and 40 mmHg, respectively, were measured in 112 (41.2%) patients, 31 (11.4%) patients, 15 (5.5%) patients, and 5 (1.8%) patients, respectively. Triamcinolone-induced IOP elevation was treated by antiglaucoma medication in all but 3 (1.0%) eyes, for which filtering surgery was performed. Mean IOP started to rise 1 week after injection and returned to baseline values approximately 8 to 9 months after injection. Younger age ($P = 0.029$) was significantly associated with triamcinolone-induced ocular hypertension. Triamcinolone responders and triamcinolone non-responders did not vary significantly in gender ($P = 0.42$), refractive error ($P = 0.86$), diabetes mellitus status ($P = 0.74$), and reason for treatment.

Conclusions: These findings may be useful for comparing risks and benefits of intravitreal triamcinolone acetonide therapy. *Ophthalmology* 2005;112:593–598 © 2005 by the American Academy of Ophthalmology.

Intraocular neovascular, inflammatory, and edematous diseases, such as diabetic macular edema, uveitis, and persistent cystoid macular edema, have increasingly been treated by intravitreal triamcinolone acetonide.^{1–29} Previous studies have shown that 1 of the 2 most common side effects of intravitreal triamcinolone acetonide was a steroid-induced elevation of intraocular pressure.^{5,15,30–34} It was the purpose of this study to evaluate how often and when after the injection intraocular pressure rises; which are the predictive factors for a postinjection elevation of intraocular pressure; whether intraocular pressure comes back to baseline and if so, when; and how many patients need lowering of intraocular pressure with topical medication or with surgery. Addressing these questions may enable the clinician to better compare the disadvantages and possible advantages of the new treatment modality and to estimate the risk of intraoc-

ular pressure-related complications after an intravitreal injection of triamcinolone acetonide.

Patients and Methods

The case series study included 272 patients (305 eyes; 164 women; 148 right eyes), who received an intravitreal injection of approximately 20 mg triamcinolone acetonide as treatment for diffuse diabetic macular edema (n = 84 patients), exudative age-related macular degeneration (n = 181 patients), retinal vein occlusions (n = 20 patients), uveitis (n = 9), pseudophakic cystoid macular edema (n = 6), and other reasons (n = 5). Pars-plana vitrectomy had not been performed in any of the eyes included in the study. For those patients who received the intravitreal injection in both eyes, 1 randomly chosen eye was taken for inclusion in the study. Mean age was 73.0 ± 11.1 years (median, 74.9 years), and mean refractive error was 0.61 ± 1.89 diopters (median, 0.50 diopters; range, -7.0 to $+7.0$ diopters). We have reviewed patients previously reported by our group^{7,8,11–13,16,22,26,27,29,31,32,34} with an emphasis on intraocular pressure data and included additional patients and follow-up data to amplify and expand on information from our previous reports. All patients were fully informed about the experimental character of the treatment. All patients had signed an informed consent. The Ethics Committee of the University had approved the study, which followed the tenets of the Declaration of Helsinki.

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Table 1. Number and Frequency of Elevated Intraocular Pressure after

Intraocular Pressure (mmHg)	Baseline	1 Week	1 Month	2 Months	3 Months	4 Months
≤21	268/272 (98.5%)	153/166 (92.2%)	164/201 (81.6%)	129/162 (79.6%)	131/172 (76.2%)	110/137 (79.6%)
>21–≤25	4/272 (0.5%)	10/166 (6.0%)	25/201 (12.5%)	22/162 (13.7%)	26/172 (15.2%)	12/137 (8.8%)
>25–≤30	0	2/166 (1.2%)	10/201 (4.5%)	9/162 (5.5%)	7/172 (4.1%)	9/137 (6.6%)
>30–≤35	0	0	1/201 (0.5%)	1/162 (0.6%)	5/172 (3.0%)	0
>35–≤40	0	1/166 (0.6%)	1/201 (1.0%)	0	3/172 (1.8%)	0
>40–≤45	0	0	0	1/162 (0.6%)	0	0
>45	0	0	0	0	0	0

In the study group, there were 10 (3.7%) patients with primary open-angle glaucoma, 1 (0.4%) patient with secondary chronic open-angle glaucoma, 3 (1.1%) patients with ocular hypertension, and 2 (0.8%) patients who had shown a rise in intraocular pressure when topical or systemic steroids had been applied before inclusion in the study.

All patients of the study group received an intravitreal injection of approximately 20 mg crystalline triamcinolone acetonide in 0.2 ml Ringer's solution as previously described in detail.³¹ The injection was performed through the inferior pars plana, after a paracentesis was carried out and aqueous humor was released from the anterior chamber. The triamcinolone acetonide was prepared as described in detail previously.³⁵ A previous study had shown that because of the filtering process, of the original 25 mg triamcinolone acetonide taken out of the ampoule, approximately 20 to 23 mg remained in the syringe for the eventual intraocular injection.³⁶ None of the eyes received concomitant steroids topically or systemically.

At baseline of the study, visual acuity and intraocular pressure were determined. Intraocular pressure was measured by Goldmann applanation tonometry. After inclusion in the study, patients were usually reevaluated the first day and 1 week after the injection, followed by reexaminations at approximately bimonthly intervals (Table 1).

Mean follow-up was 10.4±6.7 months (median, 7.9 months; range, 3.0–35.7 months). The entire study population was divided into subgroups of patients who had a follow-up examination at least 3 months after the injection (i.e., the entire study population) and of patients (n = 122) with a follow-up of at least 9 months. The subgroup with a follow-up of at least 9 months (n = 122) and the group of patients with a follow-up of less than 9 months (n = 150) did not differ significantly in age (71.6±12.0 years vs. 74.1±10.2 years; *P* = 0.12), refractive error (0.40±1.86 diopters vs. 0.78±1.90 diopters; *P* = 0.09), reason for the injection, preoperative visual acuity (0.88±0.39 logarithm of the minimum angle of resolution units vs. 0.87±0.39 logarithm of the minimum angle of resolution units; *P* = 0.56), and preoperative intraocular pressure (15.3±2.6 mmHg vs. 15.2±3.2 mmHg; *P* = 0.41).

If intraocular pressure exceeded approximately 25 to 27 mmHg, topical antiglaucoma therapy was started using topical β -blockers and prostaglandin derivatives as the medications of first choice. If intraocular pressure continued to be elevated, topical drugs were combined, such as β -blockers with topical carbonic anhydrase inhibitors and prostaglandin derivatives. Filtering surgery was usually indicated if intraocular pressure continued to be higher than 40 mmHg despite maximal medical treatment, including systemic carbonic anhydrase inhibitors, or if development of or progression of preexisting glaucomatous optic neuropathy was detected by assessment of the optic nerve head and visual fields. Confocal laser scanning tomography of the optic disc and perimetric examinations were usually performed in patients with preexisting glaucoma and in patients with intraocular pressure higher than 35 to 40 mmHg.

Statistical analyses were performed by use of a commercially available statistical software package.³⁷ To test the statistical significance of differences between the study group and the control group, the Mann-Whitney test, Wilcoxon test, or Student's *t* test for parameters such as intraocular pressure and visual acuity was used. For parameters such as gender and right or left eye, the chi square test was applied. The level of significance was 0.05 (2-sided) in all statistical testing.

Results

How Often and When after the Injection Did Intraocular Pressure Rise?

In the study group, mean intraocular pressure increased significantly (*P*<0.001; Wilcoxon test) after the first intravitreal injection from 15.3±2.9 mmHg (median, 15 mmHg) at baseline of the study to a mean maximum of 22.3±7.0 mmHg (median, 20 mmHg; range, 11–64 mmHg) during follow-up.

Defining a rise in intraocular pressure as measurements outside the normal range, at least 1 intraocular pressure measurement higher than 21 mmHg during follow-up was measured in 112 (41.2%) patients. A maximal intraocular pressure reading value higher than 30 mmHg was detected in 31 (11.4%) patients, a maximal intraocular pressure reading value higher than 35 mmHg was measured in 15 (5.5%) patients, and 5 (1.8%) patients showed at least 1 intraocular pressure measurement higher than 40 mmHg.

Including only patients (n = 122) with a follow-up longer than 9 months, 54 (44.3%) patients had intraocular pressure measurements higher than 21 mmHg. A maximal intraocular pressure reading value higher than 30 mmHg was detected in 19 (15.6%) patients, a maximal intraocular pressure reading value higher than 35 mmHg was measured in 9 (8.2%) patients, and 3 (2.5%) patients showed at least 1 intraocular pressure measurement higher than 40 mmHg. Because the eyes with a triamcinolone acetonide-related elevation in intraocular pressure usually showed a steady increase in intraocular pressure and received topical antiglaucoma treatment early, the high intraocular pressure measurements were taken on eyes that usually already received topical treatment.

Defining a rise in intraocular pressure as the difference between the baseline value and the measurement obtained during the follow-up, the mean increase in intraocular pressure, defined as difference between maximal postoperative intraocular pressure and baseline intraocular pressure, was 6.9±6.6 mmHg (median, 6 mmHg; range, -5 to 46 mmHg; Fig 1). A rise in intraocular pressure of more than 10 mmHg was found for 61 (22.4%) patients, an intraocular pressure elevation of more than 15 mmHg in 30 (11.09%) patients, and a rise in intraocular pressure of more than 20 mmHg in 15 (5.5%) patients.

an Intravitreal Injection of about 20 mg Triamcinolone Acetonide

5 Months	6 Months	7 Months	8 Months	9 Months	10 Months
79/102 (77.5%)	81/96 (82.7%)	48/67 (79.1%)	47/58 (79.3%)	36/39 (92.3%)	28/31 (90.3%)
14/102 (13.8%)	5/96 (5.1%)	6/67 (9.0%)	6/58 (10.3%)	3/39 (7.8%)	2/31 (6.4%)
6/102 (6.0%)	5/96 (5.1%)	2/67 (3.0%)	2/58 (3.4%)	0	1/31 (3.2%)
1/102 (1.0%)	3/96 (3.0%)	5/67 (7.5%)	1/58 (1.7%)	0	0
1/102 (1.0%)	2/96 (2.%)	0	1/58 (1.7%)	0	0
1/102 (1.0%)	1/96 (1.0%)	0	1/58 (1.7%)	0	0
0	1/96 (1.0%)	1/67 (1.5%)	1/58 (1.7%)	0	0

Which Were Predictive Factors for a Postinjection Elevation of Intraocular Pressure?

Dividing the entire study group into subgroups according to the reason for intravitreal triamcinolone injection showed that the mean rise in intraocular pressure did not vary significantly ($P > 0.10$) between patients with exudative age-related macular degeneration (6.8 ± 6.6 mmHg; median, 6 mmHg), patients with diffuse diabetic macular edema (6.4 ± 6.1 mmHg; median, 5 mmHg; $P = 0.76$), patients with pseudophakic cystoid edema (5.5 ± 6.1 mmHg; median, 6 mmHg; $P = 0.59$), patients with branch retinal vein occlusion (5.6 ± 4.9 mmHg; median, 6 mmHg; $P = 0.60$), patients with central retinal vein occlusion (9.4 ± 9.7 mmHg; median, 7 mmHg; $P = 0.63$), and patients with uveitis (11.7 ± 7.2 mmHg; median, 13 mmHg; $P = 0.06$). There was a tendency toward a higher increase in intraocular pressure in patients with uveitis and patients with central retinal vein occlusion.

Dividing the study group into a subgroup with chronic open-angle glaucoma, ocular hypertension, or a known steroid responsiveness, and a second subgroup without history of elevated intraocular pressure, the rise in intraocular pressure did not vary significantly ($P = 0.87$) between the glaucoma subgroup (6.5 ± 4.8 mmHg; median 7 mmHg; range, -1 –19 mmHg) and the remaining eyes (6.9 ± 6.7 mmHg; median 5 mmHg; range, -5 –46 mmHg). In a similar manner, maximal intraocular pressure during follow-up did not vary significantly ($P = 0.12$) between the glaucoma group (23.8 ± 5.7 mmHg; median, 24 mmHg; range, 13–34 mmHg) and the remaining patients (22.2 ± 7.0 mmHg; median, 20 mmHg; range, 11–64 mmHg). Maximal rise in intraocular pressure during the follow-up was correlated with younger age ($P = 0.029$). It was statistically independent of the length of follow-up ($P = 0.08$), diabetes mellitus ($P = 0.81$), and preoperative visual acuity ($P = 0.99$).

Dividing the study group into triamcinolone responders with at least 1 intraocular pressure measurement higher than 21 mmHg during follow-up and triamcinolone nonresponders showed that both subgroups did not vary significantly in gender ($P = 0.14$), refractive error ($P = 0.13$), and preoperative visual acuity ($P = 0.64$). The triamcinolone responders had a tendency toward a higher frequency of a history of glaucoma ($P = 0.16$; chi square test) and had significantly ($P < 0.001$) higher baseline intraocular pressure values than the nonresponders. Age was significantly ($P = 0.005$) lower in the triamcinolone responding subgroup than in the triamcinolone nonresponding subgroup (age, 71.3 ± 9.9 years vs. 74.1 ± 11.8 years).

A binary logistic regression analysis confirmed that younger age ($P = 0.05$) was associated with triamcinolone-induced ocular hypertension. Baseline diagnosis of glaucoma or history of steroid-induced ocular hypertension showed a tendency ($P = 0.12$) toward an association with a triamcinolone-induced rise in intraocular pressure. Similar results were found if the amount of elevation in intraocular pressure after the intravitreal injection was taken as an outcome measure. Length of follow-up ($P = 0.13$) and diagnosis

of diabetes mellitus ($P = 0.86$) or diagnosis of uveitis ($P = 0.41$) did not show a significant association in the regression analysis.

Does Intraocular Pressure Return to Baseline and, if So, When?

The rise in mean intraocular pressure started (in a statistical sense) during the first week after the intravitreal injection and returned to the preinjection baseline values approximately 8 to 9 months after the injection (Table 1). Intraocular pressure was significantly ($P < 0.001$) lower at the end of follow-up (16.9 ± 5.7 mmHg; median, 16 mmHg; range, 6–64 mmHg) than the peak values during follow-up. Including only patients ($n = 122$) with a follow-up of more than 9 months, intraocular pressure at the end of follow-up (16.0 ± 3.6 mmHg; median, 16 mmHg) did not vary significantly ($P = 0.16$) from the baseline values of these eyes (15.3 ± 2.6 mmHg; median, 16 mmHg). The return of intraocular pressure values from the peak during the follow-up to the baseline level started approximately 5 months after the injection and was usually completed approximately 9 months after the injection.

The percentage of eyes with intraocular pressure readings higher than 21 mmHg increased from 7.8% 1 week after the injection to 18.4% 1 month after the injection to 20.4% 2 months after the injection and 23.8% 3 months after the injection (Table 1). From that time on, the percentage with intraocular pressure readings higher than 21 mmHg stabilized at approximately 20% to 25% for the period of 4 to 8 months after the injection. Nine months after the

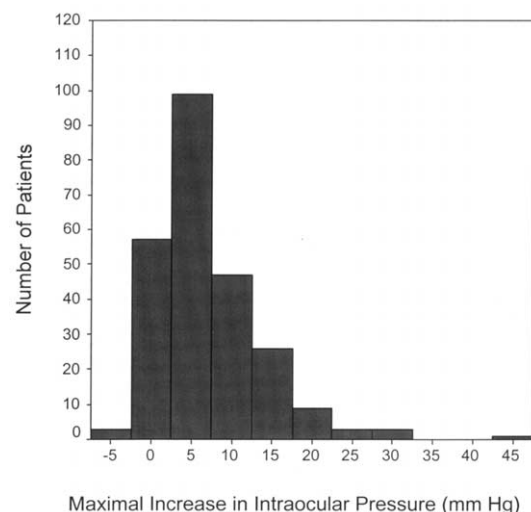


Figure 1. Histogram showing the maximal gain in intraocular pressure during follow-up in 272 patients after intravitreal injection of approximately 20 mg triamcinolone acetonide.

injection, intraocular pressure decreased to 7.3% and to 9.7% at 10 months after the injection (Table 1).

How Many Patients Need Lowering of Intraocular Pressure with Topical Medication or with Surgery?

In all but 3 (3 of 305 or 1.0%) eyes of 3 (3 of 272 or 1.1%) patients, elevation of intraocular pressure was treated by antiglaucoma medication, including topical antiglaucoma drops and systemic carbonic anhydrase inhibitors. Filtering surgery was carried out in 3 (3 of 305 or 1.0%) eyes that showed an elevation of intraocular pressure to values up to 65 mmHg ($n = 2$) or up to 35 mmHg ($n = 1$) despite maximal topical and systemic antiglaucoma therapy. The intravitreal injection had taken place 8.0 ± 0.7 months (range, 7.5–9.0 months) before trabeculectomy. In all eyes, triamcinolone acetonide crystals were ophthalmoscopically visible in the inferior preretinal vitreous cortex at the time of trabeculectomy. One of the patients had primary open-angle glaucoma before the intravitreal triamcinolone injection. The postoperative course after filtering surgery was unremarkable in all patients operated on with functioning filtering blebs and intraocular pressure measurements less than 16 mmHg.

Discussion

Intravitreal triamcinolone acetonide has increasingly been used in previous studies as treatment for intraocular proliferative, edematous, and neovascular diseases, such as central retinal vein occlusion, neovascular glaucoma without or with cataract surgery, chronic preperipheral ocular hypotony, chronic uveitis, persistent pseudophakic cystoid macular edema, exudative age-related macular degeneration, proliferative diabetic retinopathy, ischemic ophthalmopathy, sympathetic ophthalmia, and in other clinical situations.^{1–29} In aqueous humor and in silicone oil, triamcinolone acetonide has been found up to 1.5 years after the intravitreal injection.^{38–42} The ocular pharmacokinetics of intravitreal triamcinolone acetonide was described as nonlinear and by a 2-compartment model with bolus input and first-order output.⁴¹ Systemic and local side effects reported so far include cataract; secondary ocular hypertension leading in some patients to secondary chronic open-angle glaucoma; and postinjection, infectious, or sterile endophthalmitis.^{5,30–35,43} This study addressed various questions.

How Often and When after the Injection Does Intraocular Pressure Rise?

Examining a study population more than 3 times larger with a longer mean and maximal follow-up than in the previous pilot studies, our investigation confirms that approximately 40% of the patients have a secondary ocular hypertension develop. The rise in intraocular pressure started approximately 1 week to 2 months after the injection. The percentage of eyes with intraocular pressure readings higher than 21 mmHg steadily increased from 7.8% at 1 week after the injection to 23.8% at 3 months after the injection. This shows that intraocular pressure in patients after an intravitreal injection of triamcinolone acetonide may have to be controlled at least 3 to 4 months after the injection.

Which Are the Predictive Factors for a Postinjection Elevation of Intraocular Pressure?

In a multiple regression analysis, younger age was a significant factor contributing to the triamcinolone acetonide-induced increase in intraocular pressure. Diagnosis of diabetes mellitus did not influence intraocular pressure after the injection. This may agree with previous randomized clinical trials in which diabetes mellitus was not a major risk factor for glaucoma.⁴⁴ From a clinical point of view, diagnosis of diabetes mellitus may not contradict an intravitreal application of triamcinolone acetonide as previously studies have also demonstrated.^{13–16,45}

Does Intraocular Pressure Come Back to Baseline and, if So, When?

The rise in intraocular pressure started approximately 1 week after the injection, and the measurements returned to the baseline values after approximately 9 months (Table 1). Although not systemically investigated in this study, many eyes included in the investigation showed ophthalmoscopically visible triamcinolone acetonide crystals in the vitreous for a period similar to that for the increase in intraocular pressure. It suggests that when the triamcinolone acetonide crystals have resolved, intraocular pressure may return to its baseline level and that the triamcinolone-induced increase in intraocular pressure is reversible. This concurs with previous studies on the reaction of intraocular pressure after topical application of corticosteroids.⁴⁶

The figures found in this study may be valid for a dose of approximately 20 mg triamcinolone acetonide, which is a considerably higher dose than used in previous studies. In the latter, usually 2 to 8 mg of triamcinolone acetonide was intravitreally injected.^{1–6,9,10,14,15,17–19,21,24,25,28,30} Wingate and Beaumont⁷ examined a total of 113 patients with angiographically proven subretinal neovascularization who received an intravitreal injection of 4 mg triamcinolone acetonide. They found that approximately 30% of the study group had a significant rise (≥ 5 mmHg) in intraocular pressure above baseline develop during the first 3 months. Bakri and Beer³³ examined 43 eyes of 38 consecutive patients who also received an intravitreal injection of 4 mg triamcinolone acetonide and who had a follow-up of 12 weeks. They observed that within 12 weeks after the intravitreal triamcinolone acetonide injection, 21 of 43 eyes (48.8%) demonstrated an increase in intraocular pressure of 5 mmHg or greater, and 12 of 43 eyes (27.9%) had an increase in intraocular pressure of 10 mmHg or greater. The mean time for an increase in intraocular pressure of 5 mmHg or greater to occur was 4.1 weeks, and the mean time to reach maximum intraocular pressure was 6.6 weeks. All eyes responded to topical glaucoma medication. The data on the frequency of a triamcinolone acetonide-induced ocular hypertension in the studies applying a lower dose seem, therefore, not to be markedly different from the figures found in this investigation.^{5,9,14,18,21,47} It has, therefore, remained unclear so far whether the increase in intraocular pressure after the intravitreal triamcinolone acetonide injection is dose dependent or not. When the studies using

different dosages of triamcinolone acetonide are compared with each other, the duration of triamcinolone acetonide-induced ocular hypertension is longer if a dose of approximately 20 mg is used (about 7–9 months vs. 3–5 months).

There are major limitations of this study. The most important flaw in the study is the rather incomplete follow-up of the patients beyond 3 months after the intravitreal injection. The major reason for the lack of data is that a number of patients were followed up by their local ophthalmologists and were usually referred to the hospital only in case of complications such as an elevation of intraocular pressure. As in any study, one cannot exclude the possibility that some patients with increased intraocular pressure as a complication of the intravitreal injection were referred to other hospitals. The flaw in the study of an incomplete data acquisition during the follow-up may, thus, partially increase the percentage of patients with increased intraocular pressure, and it may partially decrease the percentage of patients with a rise in intraocular pressure. Because, however, the rise in intraocular pressure usually occurred within the first 3 months after the injection, the data of this study may be valid for the frequency of triamcinolone acetonide-induced ocular hypertension, and the data of the patients with long follow-up may be useful to estimate the course of intraocular pressure, including the duration of the increase in intraocular pressure.

In conclusion, the data of this prospective study suggest that the intravitreal injection of approximately 20 mg of triamcinolone acetonide can increase intraocular pressure beyond 21 mmHg in up to 40% of patients, and that in most patients, the triamcinolone-induced rise in intraocular pressure can be treated topically, except approximately 1% of patients who must undergo filtering surgery. Intraocular pressure starts to rise during the first week after injection and returns to preinjection values approximately 8 to 9 months after the injection.

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