

Intravitreal steroid slow-release device replacing repeated intravitreal triamcinolone injections for sympathetic ophthalmia

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PURPOSE. *To report on the intraocular use of a steroid slow-release device in an attempt to avoid multiple intraocular triamcinolone injections in chronic sympathetic ophthalmia.*

METHODS. *A 47-year-old patient with sympathetic ophthalmia had received 17 intravitreal triamcinolone injections to suppress the uveitis, to increase intraocular pressure, and to reduce systemic anti-inflammatory medication. To avoid the frequent reinjections combined with the temporary reduction in vision and potential risk of infection and a recurrence of sympathetic ophthalmia, a slow-release device of 2.1-mg fluocinolone acetonide was intravitreally implanted.*

RESULTS. *During the follow-up of 11 months after the procedure, intraocular pressure stabilized at 12 to 18 mmHg and visual acuity at 0.40 to 0.50. The systemic immunosuppressive therapy was stopped, and consequently, the insulin treatment could be halted.*

CONCLUSIONS. *Despite the limitations of a single case report, the results suggest that an intravitreal slow-release device of fluocinolone may be an alternative to repeatedly administered intravitreal triamcinolone injection for the long-term treatment of sympathetic ophthalmia. The intraocular slow-release application of steroids has enabled patients to live free from diabetic treatment and immunosuppressive medication after 21 years of systemic immunosuppressive therapy with secondary Cushing disease including diabetes mellitus and arterial hypertension. (Eur J Ophthalmol 2008; 18: 834-6)*

KEY WORDS. *Intravitreal triamcinolone, Sympathetic ophthalmia, Immunosuppression, Cushing disease, Intraocular pressure, Fluocinolone, Slow-release device, Retisert*

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INTRODUCTION

Sympathetic ophthalmia is a granulomatous inflammation of the posterior segment of the eye leading to bilateral blindness unless systemic immunosuppressive therapy is initiated in its early stage. Systemic immunosuppressive therapy, including systemic steroid therapy, which usually has to be maintained throughout life, is associated with severe side effects such as bone fragility, susceptibility to infections, obesity, diabetes mellitus, and arterial hypertension. After the intravitreal application of triamcinolone acetonide was introduced into clinical ophthalmology, sym-

pathetic ophthalmia has increasingly been treated by intravitreal triamcinolone, in an attempt to reduce the systemic side effects of steroid treatment (1-3). Since a single intravitreal triamcinolone application is effective for only 2 to 6 months, the intravitreal triamcinolone injections have to be repeated twice to five times a year (4). The risks of intraocular infections and other injection-related complications increase with the number of injections in a cumulative manner. It was, therefore, the purpose of the present study to report on the intraocular use of a steroid slow-release device in an attempt to avoid multiply repeated intraocular triamcinolone injections.

METHODS

For 17 years, a 47-year-old patient with sympathetic ophthalmia had been treated by systemic and local immunosuppressive therapy including cyclosporine A, methotrexate, infliximab, systemic steroids with a daily dosage ranging between 10 and 30 mg prednisolone, and peribulbar steroid injections. The patient had experienced a central retinal vein occlusion in his right eye 18 years ago, initially treated by panretinal argon laser coagulation. Due to secondary angle-closure glaucoma with iris neovascularization, several transscleral retinal cryocoagulations had been performed. After implantation of a glaucoma drainage device 1 year after the central retinal vein occlusion, the patient had developed sympathetic ophthalmia leading to enucleation of his right eye. His left eye eventually underwent intracapsular cataract surgery and pars plana vitrectomy.

Due to the systemic steroid therapy, the patient had developed severe symptoms of Cushing disease with steroid-induced diabetes mellitus, arterial hypertension, and obesity. Despite the intensive treatment, the eye was hypotonic with intraocular pressure measurements ranging between 5 and 8 mmHg. Visual acuity measured 0.30. After an intravitreal injection of about 20 mg of triamcinolone acetonide, visual acuity improved to 0.50, perimetry revealed an enlargement of the constricted visual field, and intraocular pressure increased to values between 12 and 20 mmHg. Since 3 months later, visual acuity regressed to 0.30 and intraocular pressure was reduced to 5 and 10 mmHg, an intravitreal reinjection was carried out, after which visual acuity re-increased to 0.50 and intraocular pressure re-increased to values between 10 and 18 mmHg. Due to the limited duration of the triamcinolone effect, altogether 17 intravitreal triamcinolone injections were performed during the follow-up of now 4.5 years after the first injection. The systemic immunosuppressive treatment was reduced to 2.5 mg prednisolone per day and could not be stopped due to a temporary insufficiency of the adrenal glands. The patient lost 15 kg of body weight; the daily insulin dosage could be reduced from more than 100 units to 0 units. Arterial blood pressure was lowered from about 180/100 mmHg to about 140/90 mmHg, coincidentally with a reduction of systemic antihypertensive treatment from four drugs to a beta blocker as the only medication.

RESULTS

In an attempt to avoid frequent reinjections combined with temporary reduction in vision and potentially increased risk of a recurrence of sympathetic ophthalmia, a slow-release device of 2.1 mg fluocinolone acetonide was intravitreally implanted at a time when triamcinolone crystals were still present in the eye (5-7). The patient was fully informed about the experimental character of the treatment and had signed an informed consent.

During the follow-up of 11 months after the procedure, intraocular pressure has stabilized at 12 to 18 mmHg and visual acuity at 0.30 to 0.40. The anterior chamber was free of flare and cells. The device was located in the temporal inferior region of the vitreous cavity fixed to the scleral wall in the pars plana region. The retina showed pre-existing chorioretinal patchy scars due to the sympathetic ophthalmia. With more time having passed since the systemic steroid was reduced to low levels of 2.5 mg, the systemic immunosuppressive therapy with steroids could eventually be stopped. The arterial blood pressure became almost normalized with only one oral antihypertensive medication taken.

DISCUSSION

Confirming previous studies on the use of the fluocinolone implant for the treatment of noninfectious uveitis (5-7), the results suggest that an intravitreal slow-release device of fluocinolone may be an alternative to repeatedly administered intravitreal triamcinolone injection for the long-term treatment of sympathetic ophthalmia. The major advantage of slow-release devices compared with repeated intravitreal triamcinolone injections is that they provide continuous release of steroid over 2.5 years, so that the patient does not require multiple intraocular triamcinolone injections. It is associated with the other major advantage that one obtains a continuous suppression of the inflammatory intraocular disease and not the intermittent disease remission/relapse which is how intraocular inflammation has been managed, given that one waits until the inflammation relapses before the treatment is reinitiated, either intravitreally or systemically. Since the present investigation is a single case re-

port, one has to take into account that although the study establishes a proof of principle on the use of the intravitreal slow-release device for the treatment of sympathetic ophthalmia, the outcome does not represent incontrovertible evidence to support the safety and efficacy in other patients as well. For the decision of the clinical use of an intravitreal slow-release device one has to consider the potential risks of the device such as a lesion to the retina and secondary induction of a rhegmatogenous retinal detachment, cataract in almost 100% of the patients after a follow-up of a few years, and secondary glaucoma necessitating filtering surgery in a substantial number of patients (5-7). In particular, the present single case report does not allow any conclusions on the safety of the device, particularly in single eyed patients, since one may need the observations of a considerably larger number of patients to deduce statements on the safety of the device.

In conclusion, the intraocular application of the slow-release device for steroids enabled the patient to

live free from steroid-induced diabetes mellitus and arterial hypertension and their treatment after 21 years of systemic immunosuppressive therapy leading to secondary Cushing disease, including diabetes mellitus and arterial hypertension. In addition, the intraocular application of the slow-release device made repeated intravitreal injections of triamcinolone unnecessary.

Proprietary interest: None.

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