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SHORT COMMUNICATION

Case report

Intravitreal triamcinolone acetonide as treatment of ischemic ophthalmopathy

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PURPOSE. *To describe the clinical course of a patient receiving repeated intravitreal injections of triamcinolone acetonide (25 mg) as treatment of ischemic ophthalmopathy.*
METHODS. *A 70-year-old patient with Waldenström disease presented with progressive iris neovascularization, vitreous hemorrhage, and ocular hypotony due to ischemic ophthalmopathy. Visual acuity was 0.05. Within 2.5 years, he received three intravitreal injections of 25 mg of triamcinolone acetonide. Additionally, penetrating keratoplasty and synechiolysis were performed during the follow-up.*
RESULTS. *After each intravitreal injection, visual acuity and intraocular pressure increased, iris neovascularization regressed, and vitreous haze cleared up.*
CONCLUSIONS. *Intravitreal triamcinolone acetonide may induce regression of iris neovascularization, increase intraocular pressure, and improve visual acuity in eyes presenting with ocular hypotony, vitreous hemorrhage, and progressive intraocular neovascularization due to ischemic ophthalmopathy. (Eur J Ophthalmol 2003; 13: 575-6)*

KEY WORDS. *Intraocular pressure, Intravitreal triamcinolone, Iris neovascularization, Ischemic ophthalmopathy, Goniosynechiae*

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INTRODUCTION

Ischemic ophthalmopathy can be caused by a variety of conditions, such as carotid artery occlusion, diabetes mellitus, and other systemic diseases. We describe the clinical course of a patient who developed typical symptoms of ischemic ophthalmology as complications of Waldenström disease.

PATIENTS AND METHODS

A 70-year-old patient with Waldenström disease presented with progressive iris neovascularization, vitreous hemorrhage, and ocular hypotony due to ischemic ophthalmopathy in his left eye, which was the only functioning eye. Intracapsular cataract surgery had been performed 25 years ago. Visual acuity was 0.05, and intraocular pressure ranged between 6 and 10 mm Hg. In an attempt to decrease progressive iris neovascularization, to increase intraocular pressure, and to stabilize the eye, an intravitreal injection of 25 mg of triamcinolone acetonide was transconjunctivally applied through the pars plana. The procedure of the intravitreal application has been described in detail (1-5). The patient was fully informed about the experimental character of the treatment and signed an informed consent. The ethics committee of the university approved the study following the tenets of the Declaration of Helsinki.

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RESULTS

Within 3 months after the first injection, visual acuity improved from 0.05 to 0.125, intraocular pressure increased to values ranging between 16 mm Hg and 22 mm Hg, and iris neovascularization regressed. Six months later, a corneal band degeneration that had developed in the meantime was removed. One year after the first injection, visual acuity decreased to hand movements, owing to marked vitreal haze and recurrence of a progressing iris neovascularization. Sixteen months after the first injection, the patient received a second intravitreal injection of 25 mg of triamcinolone acetonide, after which the iris neovascularization regressed again. Four months later, a penetrating keratoplasty was performed owing to progressive corneal endothelial decompensation. After keratoplasty, visual acuity improved to 0.10, and intraocular pressure increased to 27 mm Hg, requiring antiglaucomatous topical therapy. For 6 months, visual acuity remained stable at 0.10 and intraocular pressure was within the normal range. Owing to recurring and progressive iris neovascularization with subsequent complete closure of the anterior chamber angle and development of anterior synechiae toward the corneal graft, a third injection of 25 mg of triamcinolone acetonide in combination with an anterior synechiolysis and a goniosynechiolysis was carried out 7 months after the keratoplasty. At the end of follow-up, 2 months after the third injection, visual acuity was 0.08, intraocular pressure was 19 mm Hg, and the synechiae to the corneal graft had not re-formed. The fundus was clearly visible as there was no longer vitreous haze.

DISCUSSION

The clinical course of the patient suggests that repeated intravitreal injections of 25 mg of triamcinolone acetonide might be useful in the treatment of ischemic ophthalmopathy presenting with ocular hypotony, progressive iris neovascularization, and vitreous hemorrhage. The recurring iris neovascularization regressed after each injection, suggesting an angiostatic effect of intravitreal triamcinolone acetonide. Anterior synechiae that developed during the follow-up have not recurred after synechiolysis combined with a re-injection of triamcinolone acetonide.

In conclusion, repeated intravitreal injections of triamcinolone acetonide might be useful as an additional tool in the armamentarium of treatment modalities for ischemic ophthalmopathy with complex ocular findings.

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