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SHORT COMMUNICATION

Intravitreal bevacizumab for neovascular glaucoma following central retinal artery occlusion

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PURPOSE. *To report a case of neovascular glaucoma due to central retinal artery occlusion treated with a single intravitreal injection of bevacizumab.*
METHODS. *A 68-year-old patient with a 10-week history of central retinal artery occlusion presented with neovascularization of the iris and the angle and intraocular pressure of 30 mmHg. The patient received a single injection of 1.25 mg bevacizumab in 0.1 mL intravitreally.*
RESULTS. *Iris and angle neovascularization regressed within 48 hours of the injection. Intraocular pressure dropped from 30 to 15 mmHg, and there was marked improvement in patient comfort. Panretinal photocoagulation was applied 4 weeks after the injection.*
CONCLUSIONS. *Bevacizumab seems to be a useful adjunct to panretinal photocoagulation in the treatment of neovascular glaucoma. (Eur J Ophthalmol 2007; 17: 269-71)*

KEY WORDS. *Bevacizumab, Intravitreal injection, Neovascular glaucoma, Retinal artery occlusion*

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INTRODUCTION

Bevacizumab (Avastin, Genentech Inc., San Francisco, CA, USA) is a humanized monoclonal antibody which blocks all isoforms of vascular endothelial growth factor. Vascular endothelial growth factor was implicated to have a role in the development of abnormal vessels in a variety of ocular diseases, including wet age-related macular degeneration, proliferative diabetic retinopathy, and central retinal vein occlusion (1). The off-label use of bevacizumab in the treatment of those diseases has recently been reported in several articles (2-5). Management of neovascular glaucoma is one of the most challenging tasks for the ophthalmologist. We describe a case of neovascular glaucoma due to central retinal artery occlusion treated with a single intravitreal injection of bevacizumab.

Case report

A 68-year-old woman with a 10-week history of central retinal artery occlusion of the left eye was referred for panretinal photocoagulation due to neovascularization of the iris and the angle.

The visual acuity was amaurosis and intraocular pressure was 30 mmHg. Anterior segment evaluation revealed neovascularization of the pupil margin from 1 o'clock to 6 o'clock. Gonioscopy revealed neovascularization in the angle with some peripheral anterior synechiae at 6 o'clock. Fundus examination showed pale retina with attenuated arteries and cherry red spot at the fovea. Fluorescein angiography of the iris showed leakage from new iris vessels.

Despite maximal medical therapy to control intraocular pressure, the patient experienced marked discomfort. Treatment options were discussed in detail, and the use of bevacizumab was explained as an alternative, experimental medication. After opting for intravitreal bevacizumab, the patient signed a consent form.

Intravitreal bevacizumab was applied in the operating theater after placing a lid speculum and instilling 5% povidone iodine on the eye. A total dose of 1.25 mg bevacizumab in 0.1 mL was injected into the vitreous cavity

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through the inferotemporal pars plana. Topical tobramycin was prescribed three times daily for 3 days.

One day after the injection the iris vessels began to regress, and on the next day there was only a small fibrous line at the 6 o'clock position. Intraocular pressure dropped down from 30 mmHg to 15 mmHg, and the patient reported a marked improvement in comfort. Fluorescein angiography performed on the next day revealed no leakage from the iris.

Four weeks after the injection there was no sign of recurrent iris neovascularization, intraocular pressure was 14 mmHg, with no topical antiglaucoma medication, and the patient received panretinal photocoagulation.

DISCUSSION

The use of intravitreal bevacizumab for neovascular glaucoma has recently been reported by Kahook et al (6). They treated a patient who developed neovascular glaucoma after central retinal vein occlusion. Despite the treatment with panretinal photocoagulation and cyclophotocoagulation, intraocular pressure remained elevated and neovascularization of the iris and the angle persisted. The injection of bevacizumab led to a dramatic regression of new iris and angle vessels, as well as to a decrease of intraocular pressure.

In another report by Davidorf et al (7), intravitreal bevacizumab was injected in a patient with neovascular glaucoma of the left eye, who had a history of proliferative diabetic retinopathy treated with panretinal photocoagulation. Again, after receiving bevacizumab, there was a dramatic regression of iris and angle new vessels. However, intraocular pressure remained elevated despite maximal medical therapy, possibly due to peripheral anterior synechiae.

In our patient neovascular glaucoma developed as a result of central retinal artery occlusion.

The reported incidence of rubeosis iridis after central retinal artery occlusion is nearly 20% (8-10). The mean time for the development of iris new vessels is 4 to 5 weeks (8, 9), unlike in eyes with central retinal vein occlusion, in which the new vessels develop at a mean time of 5 months (11).

We treated the patient with a single intravitreal injection of bevacizumab which led to a dramatic regression of iris and angle new vessels, and to a sharp drop of intraocular pressure. There were no complications regarding the procedure or the action of bevacizumab itself, and panretinal photocoagulation was performed 4 weeks after the injection.

Intravitreal bevacizumab could be a useful adjunct to panretinal photocoagulation in the treatment of neovascular glaucoma, due to its remarkable biologic effect. Further studies are necessary to determine its optimal dosage and timing in the treatment of proliferative vascular diseases.

The authors have no financial or proprietary interest in any material or method mentioned.

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