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Case Report Open Access

Long-term Intraocular Pressure Control in a Case of Neovascular Glaucoma Treated with Repeated Intravitreal Bevacizumab Injections

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Abstract

Ischemic retinopathies may cause neovascular glaucoma due to the growth of fibrovascular tissues which may close the anterior chamber angle and increase intraocular pressure. Angiogenesis factors, such as vascular endothelial growth factor (VEGF), play a fundamental role in the development and maintenance of these diseases. The purpose of this study was to report a case of bilateral neovascular glaucoma secondary to proliferative diabetic retinopathy treated with 16 intravitreal bevacizumab injections (Avastin®) and followed up for 200 weeks. Adequate intraocular pressure control was observed after bevacizumab injection as well as regression of anterior and posterior segment neovascularization, and maintenance of visual acuity. In the present case, the treatment of neovascular glaucoma with intravitreal bevacizumab was effective for long-term intraocular pressure control although repeated injections were necessary.

Keywords: Neovascular glaucoma; Diabetic retinopathy; Retinal neovascularization; Angiogenesis inhibitors; Case reports

Introduction

Neovascularization of the anterior segment results from growth of fibrovascular tissues (new vessels) over the iris and the anterior chamber angle, which obstructs the trabecular meshwork and may result in peripheral anterior synechiae and angle closure causing neovascular glaucoma with elevation of the intraocular pressure [1,2]. In most cases, neovascularization is associated with diseases that cause retinal ischemia and trigger the increase of angiogenetic factors [3], such as the vascular endothelial growth factor (VEGF), which plays an important role in neovascularization [3,4].

The treatment of neovascular glaucoma remains a challenge to specialists. Panretinal photocoagulation (PRP) seems to be the best initial therapy, but many eyes require antiglaucoma surgeries [5] and have a poor visual prognosis [2].

Intravitreal bevacizumab has been widely used in the treatment of retinal neovascularization diseases [1,4,6-9]. It has also been used in the management of patients with neovascular glaucoma, and short-term results have been positive [1,8,9]. This report describes a case of a patient with proliferative diabetic retinopathy and neovascular glaucoma treated with intravitreal bevacizumab and followed up for 200 weeks.

Case Report

A white 57-year-old man presented with a complaint of poor visual acuity in both eyes for about one year and pain in the left eye (OS) for about one week. The patient had been diagnosed with diabetic retinopathy 14 years before and had previously undergone PRP; he also had a history of glaucoma in both eyes, which was treated with travoprost 0.004%.

At initial examination, best corrected visual acuity was 0.5 in the right eye (OD) and 0.2 in the left eye (OS). Biomicroscopic examination of anterior segment showed absence of iris neovascularization in the pupillary region of both eyes; gonioscopy showed new vessels in the OS anterior chamber angle. Indirect ophthalmoscopy showed a well-performed full scatter PRP in both eyes (OU), and a proliferative diabetic retinopathy OU with discrete fibrosis over the disc in OD and with optic disc neovascularization in OS. IOP was 22 mmHg in OD and 40 mmHg in OS.

The patient was offered treatment for the neovascularization of the anterior segment and optic disc with intravitreal injections of 1.5mg (0.06mL) bevacizumab (Avastin® 100mg/4mL; Genentech Inc, South San Francisco, CA, EUA) (IVB) in OS, after all the risks related with intravitreal injections has been explained. The patient had been treated with IVB in the OS in another ophthalmology service three months before our first evaluation, however no previous ophthalmological details could be retrieved.

After the first application of IVB in OS, the patient was followed up for 200 weeks. Fifteen other intraocular injections were applied, and the control of IOP and neovascularization of anterior and posterior segments was satisfactory (Table 1). On two separate occasions PRP was proposed to patient, who refused the procedure due to pain felt in previous sessions, even after retrobulbar anesthesia has been offered.

The criteria for injection repetition were the appearance of new vessels in the anterior segment, through clinical examination exclusively or retina based on clinical and angiographic examinations, or elevation of IOP over 22 mmHg even if the most intensive treatment regimen was being used. Depending on the treatment time point,

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Time point – T (weeks)	Intravitreal Injection	Retinal neovascularization	Angle neovascularization	OD/OS IOP (mmHg)	OD/OS VA (Snellen notation)
T (0)	--	OD+/OS+	OD-/OS+	22˚/ 40˚	0,5 / 0,2
T (1) [treatment 1]	***OS***				
T (3)	--	OD+//OS-	OD-/OS-	19˚/ 20˚	0,5 / 0,2
T (4) [treatment 2]	***OD***				
T (7)	--	OD-/OS-	OD-/OS-	14˚/ 15˚	0,8 / 0,2
T (33)	--	OD+/OS+	OD+/OS+	29#/ 30#	0,8 / 0,3
T (35) [treatment 3]	***OS***				
T (37) [treatment 4]	***OD***				
T (39)	--	OD-/OS-	OD-/OS-	19¤/ 19¤	0,7 / 0,2
T (61)	--	OD+/OS+	OD+/OS+	15#/ 21#	1,0 / 0,3
T (63) [treatment 5]	***OD***				
T (65) [treatment 6]	***OS***				
T (77)	--	OD-/OS-	OD-/OS-	12#/ 12#	0,5 / 0,2
T (84)	--	OD+/OS+	OD+/OS+	17#/18#	0,7 / 0,3
T (87) [treatment 7]	***OD***				
T (89) [treatment 8]	***OS***				
T (100)	--	OD-/OS-	OD-/OS-	11#/12#	0,8 / 0,3
T (110)	--	OD+/OS+	OD+/OS+	17#/18#	0,6 / 0,3
T (111) [treatment 9]	***OD***				
T (112) [treatment 10]	***OS***				
T (122)	--	OD-/OS-	OD-/OS-	9#/12#	0,5 / 0,2
T (145)	--	OD+/OS+	OD-/OS+	15#/24#	0,4 / 0,3
T (148) [treatment 11]	***OS***				
T (148) [treatment 12]	***OD***				
T (151)	--	OD-/OS-	OD-/OS-	12∞/16∞	0,6 / 0,3
T (159)	--	OD+/OS+	OD-/OS-	15∞/20∞	0,4 / 0,3
T (163) [treatment 13]	***OS***				
T (163) [treatment 14]	***OD***				
T (167)	--	OD-/OS-	OD-/OS-	10∞/11∞	0,7 / 0,4
T (181)	--	OD+/OS+	OD-/OS-	12∞/12∞	0,7 / 0,5
T (184) [treatment 15]	***OS***				
T (188) [treatment 16]	***OD***				
T (200)	--	OD-/OS-	OD-/OS-	13∞/14∞	0,7 / 0,5

*using travoprost 0.004%. ¤ using timolol maleate 0.5% and travoprost 0.004%.
#using timolol maleate 0.5%, dorzolamide 2% and travoprost 0.004%. ∞ using timolol maleate 0.5%, dorzolamide 2% and travoprost 0.004%, brimonidina 0,1%.
VA = visual acuity; IOP = intraocular pressure.

Table 1: Descriptive data of clinical progression of patient along time (weeks) and of treatment with intravitreal 1.5 mg bevacizumab injections.

the single or combined antiglaucoma medications used were timolol 0.5%, dorzolamide 2%, travoprost 0.004% and Brimonidine 0,1% (Table 1). Although travoprost could exacerbate ocular inflammation, all attempts to withdraw this medication led to an inappropriate IOP control. Artificial tears were used because of the side effects reported occasionally by the patient (conjunctival hyperaemia and foreign body sensation). At the end of the follow-up period visual acuity was 0.7 OD and 0.5 OS, IOP was controlled OU and a very mild cataract was observed in both eyes (Table 1).

Discussion

Intraocular bevacizumab has been used in the treatment of neovascular glaucoma in patients with proliferative diabetic retinopathy [4,5,7] and in cases of retinal vein occlusion [1,5,8]. The intravitreal or intracameral administration of this drug has beneficial effects on the regression of iris or angle neovascularization [4,7], and provides good short-term control of IOP [1,8-10].

The mechanism by which bevacizumab reduces IOP is still unknown. It may improve the opening of the anterior chamber angle and increase filtration and simultaneously improve the performance of the trabecular meshwork after neovascularization regression. Other hypothesis would be an unlikely direct effect of the drug on the production of aqueous humor [1].

Corticosteroids also have a known beneficial effect in the treatment of neovascular eye diseases and may induce regression of iris neovascularization [11]. However, no topical corticosteroids were used in the present report because the patient also had persistent retinal neovascularization in addition to anterior chamber neovessels and because the episodes of high IOP were not efficiently treated by clinical means with conventional antiglaucomatous therapy.

The present report describes the long-term follow-up of a patient with neovascular glaucoma secondary to proliferative diabetic retinopathy. In our patient, intravitreal bevacizumab induced regression of iris, angle and persistent retinal neovascularization. The patient's IOP and visual acuity levels fluctuated, but were satisfactory at 200 weeks. During follow-up, no ocular or systemic complications due to the use of intravitreal bevacizumab were observed, which confirmed the safety of a prolonged treatment.

Bevacizumab was effective for the long-term control of retinal and anterior segment neovascularization and IOP although injections had to be repeated and antiglaucoma medication had to be used in the present report. Future studies with a larger number of patients should

be conducted to confirm the efficacy and safety of this therapy for longer treatments than the one described in this case report.

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