

# Treatment of peripapillary choroidal neovascularization with intravitreal bevacizumab

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**PURPOSE.** *Peripapillary choroidal neovascularization (CNV) is an uncommon condition and often shows a growth tendency towards the fovea during spontaneous progression that threatens visual acuity. Treatment of peripapillary CNV is difficult. The authors report results of intravitreal bevacizumab therapy for peripapillary CNV.*

**METHODS.** *Four patients with CNV located in the temporal or superior peripapillary area received intravitreal bevacizumab injections. Ophthalmologic examinations including OCT were performed at baseline and at 6-week intervals. Fluorescein angiography was performed at baseline and depending on clinical and OCT findings. The mean follow-up was  $34 \pm 20$  (22–69) weeks.*

**RESULTS.** *The patients received an average of  $3.5 \pm 3.1$  (1–8) injections. In all patients fluorescein angiography showed inactivation of peripapillary CNV. No further increase in size was observed in any of the patients. The OCT showed a decrease of intraretinal and subretinal fluid. No intraocular or systemic side effects were observed.*

**CONCLUSIONS.** *In this series of patients, intravitreal bevacizumab appears to be efficacious. A progression of peripapillary CNV could be prevented in all patients and the lesion was successfully inactivated. Anti-VEGF treatment with bevacizumab represents a promising therapy option for peripapillary CNV. (Eur J Ophthalmol 2009; 19: 163-5)*

**KEY WORDS.** *Bevacizumab, Peripapillary choroidal neovascularization, VEGF*

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## INTRODUCTION

Peripapillary choroidal neovascularization (CNV) is a rare disorder that can lead to loss of central vision if left to its natural course. Treatment is difficult and several options like surgical removal of the CNV, photocoagulation, and photodynamic therapy (PDT) have been tried with varying success. We report four cases of peripapillary CNV with favorable response to intravitreal bevacizumab.

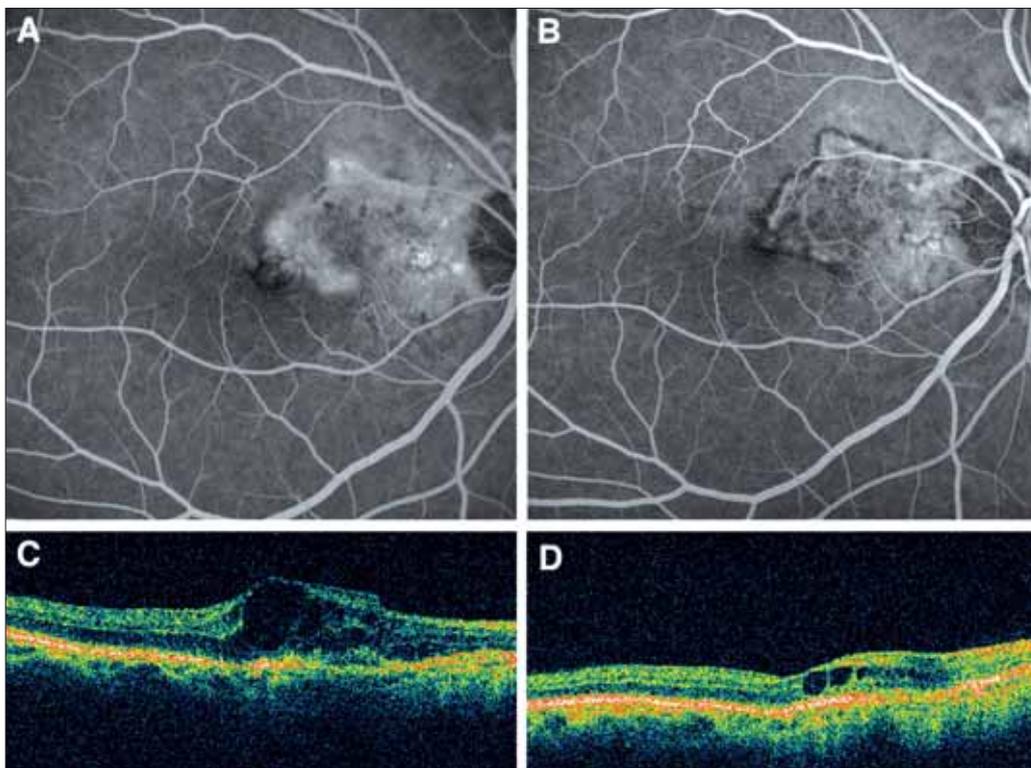
### Case 1

An 80-year-old woman presented with a new paracentral temporal scotoma and metamorphopsia in her left eye (OS). Best-corrected visual acuity (BCVA) at presentation was 20/32. Peripapillary CNV was present at the temporal margin of the optic disc with leakage extending towards the fovea, subretinal he-

morrhages, and hard exudates. OCT showed intraretinal and subretinal fluid within the papillomacular bundle with foveal involvement. The patient received off-label treatment with intravitreal bevacizumab (8 injections over a course of 63 weeks), which led to inactivation of the CNV with complete resorption of intra- and subretinal fluid, hemorrhages, and exudates. No increase or recurrence of CNV occurred during a total follow-up of 69 weeks. Atrophic changes of retinal pigment epithelium and choriocapillaris developed in the area of CNV. However, BCVA decreased to 20/100 despite favorable morphologic aspect of the central retina, possibly due to defects in the overlying nerve fibers.

### Case 2

An 82-year-old man presented with progressive decrease in visual acuity OS. He had dry age-related macular de-



**Fig. 1 - (A)** Fluorescein angiography demonstrates peripapillary choroidal neovascularization (CNV) with extension towards the foveal center and leakage. **(C)** Corresponding optical coherence tomography (OCT) shows cystoid macular edema. **(B)** Five months after initiation of bevacizumab treatment, no further growth or leakage could be observed; corresponding OCT **(D)** reveals reduction of intraretinal fluid.

generation with geographic atrophy bilaterally (OU) and CNV in the superior peripapillary area with bleeding and fluorescein leakage OS. Visual acuity was 20/50 OS at initiation of bevacizumab therapy. He received two injections of 2.5 mg bevacizumab with a spacing of 6 weeks. At last visit, 22 weeks after initiation of treatment, the subretinal hemorrhage had completely resolved and there was no more exudation. BCVA was 20/40.

### Case 3

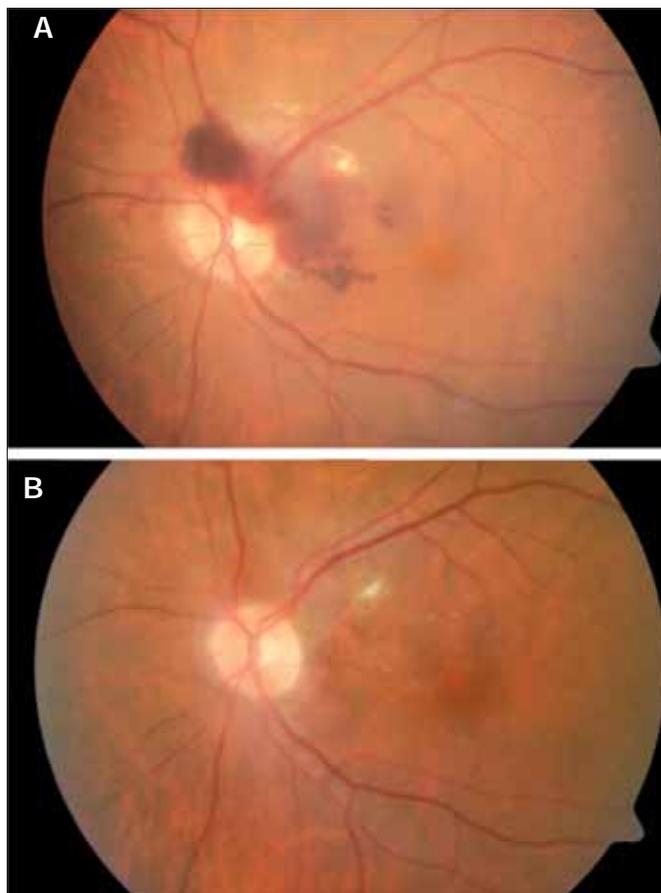
A 76-year-old man presented with peripapillary CNV in the right eye (OD) located temporally of the optic disk. The CNV had already spread to the fovea resulting in significant cystoid macular edema (Fig. 1). Visual acuity was 20/80 OD. He received intravitreal bevacizumab treatment (three injections of 1.25 mg bevacizumab over a period of 13 weeks), which led to inactivation of the lesion. No increase in size of the CNV or recurrence could be observed during a total follow-up of 25 weeks. Macular edema could be reduced, but was still present on last visit (Fig. 1). Visual acuity was 20/200; the patient felt he benefited from the treatment.

### Case 4

A 76-year-old man presented with loss of visual acuity OS. BCVA was 20/500. Ophthalmic examination and fluorescein angiography revealed CNV in the superotemporal peripapillary area leading to severe macular edema (Fig. 2). The patient received one injection of 2.5 mg bevacizumab leading to complete resorption of macular edema, inactivation of CNV, and an increase in visual acuity of 14 lines to 20/20 at last visit (Fig. 2). No recurrence of CNV occurred within a follow-up of 25 weeks.

## DISCUSSION

Peripapillary CNV is uncommon. Most cases are idiopathic or due to ARMD. Associations with other ocular conditions like high myopia, ocular histoplasmosis syndrome, multifocal choroiditis, optic disc drusen, and angioid streaks have been described (1, 2). The natural course of peripapillary CNV is unfavorable because of frequent involvement of the fovea by direct subfoveal extension or subretinal fluid, hemorrhage, or exudate. The Macular



**Fig. 2 - (A)** A 76-year-old patient with choroidal neovascularization (CNV) in the superotemporal peripapillary area leading to extensive bleeding and exudates. **(B)** Inactivation of CNV 4 months after one injection of bevacizumab.

Photocoagulation Study Group reported a loss of visual acuity of at least two lines in more than 60% of untreated eyes with nearly one-fourth having a vision of 20/500 or worse at the 3-year follow-up (3). Various treatment options have been proposed including laser photocoagulation, surgical excision, and PDT. Rosenblatt et al reported successful treatment with PDT in patients with peripapillary CNV associated to ARMD and ocular histoplasmosis syndrome (4). Although they did not report any adverse effects, no long-term data exist and damage to the optic nerve cannot be ruled out because of the close position of the treated area to the disk. Recently two cases of peripapillary CNV were described in which treatment with intravitreal bevacizumab led to inactivation of the membrane and good visual outcome (5, 6). In the case series presented we could observe favorable responses of intravitreal bevacizumab treatment in four patients with

peripapillary CNV due to ARMD. The treatment appeared to inactivate the CNV and led to reduction of intraretinal edema and subretinal fluid. No leakage and no further growth were observed on angiographic controls. During a mean follow-up period of  $34 \pm 20$  weeks, no intraocular or systemic adverse effects of intravitreal bevacizumab were observed. Loss of visual acuity in Case 1 might be explained by damage to nerve fibers within the papillomacular bundle during the regression of CNV.

In conclusion, anti-VEGF treatment with intravitreal bevacizumab represents a promising treatment option for peripapillary CNV.

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