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SHORT COMMUNICATION

Intravitreal bevacizumab for the treatment of feeder vessel of subfoveal choroidal neovascularization

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PURPOSE. *To report in a single case the effectiveness of bevacizumab in the treatment of feeder vessels (FVs) of subfoveal choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD).*
METHODS. *One patient received intravitreal bevacizumab (Avastin) (1.25 mg) which was repeated 4 weeks after the first intravitreal injection. Fluorescein angiography and optical coherence tomography (OCT) were performed. Visual acuity (VA) was also checked.*
RESULTS. *Occlusion of the FVs was observed 4 weeks after starting treatment. During the follow-up period and in the final examination 13 months after the first injection, the FVs remained occluded. Complete resolution of macular edema and improvement of the VA were noticed after the second bevacizumab intravitreal injection.*
CONCLUSIONS. *This report documents the successful occlusion of FVs of subfoveal neovascularization after two intravitreal bevacizumab injections in a patient who was followed up for 1 year after the occlusion. Further long-term investigation is warranted given the promising 12-month results. (Eur J Ophthalmol 2007; 17: 853-6)*

KEY WORDS. *Age-related macular degeneration, Bevacizumab, Feeder vessel, Fluorescein angiography, Optical coherence tomography, Subfoveal neovascularization*

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INTRODUCTION

Vascular endothelial growth factor (VEGF) plays an essential part in the function of choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD) (1).
In patients with CNV lesions its feeder vessels (FVs) have been demonstrated in a percentage ranging from 22% to 42% and are usually distinguished by videoangiography or high speed fluorescein angiography (FA) and indocyanine green angiography (ICG) (2). FVs probably enter the choriocapillaries in close proximity to the other penetrating vessels that form the choriocapillaries/CNV communication (3).
FVs treatment is difficult since most of them remain resistant to photocoagulation and photodynamic treatment (PDT) (2, 4). Since bevacizumab, a monoclone antibody to VEGF, has shown promising results in the treatment of CNV secondary to AMD (5), we offered an intravitreous injection of bevacizumab in a patient with FVs of subfoveal CNV secondary to AMD. We decided to inject bevacizumab instead of ranibizumab since ranibizumab was not officially available in Greece at that time. We also chose to use bevacizumab instead of pegaptanib since our experience with bevacizumab at that time was positive.

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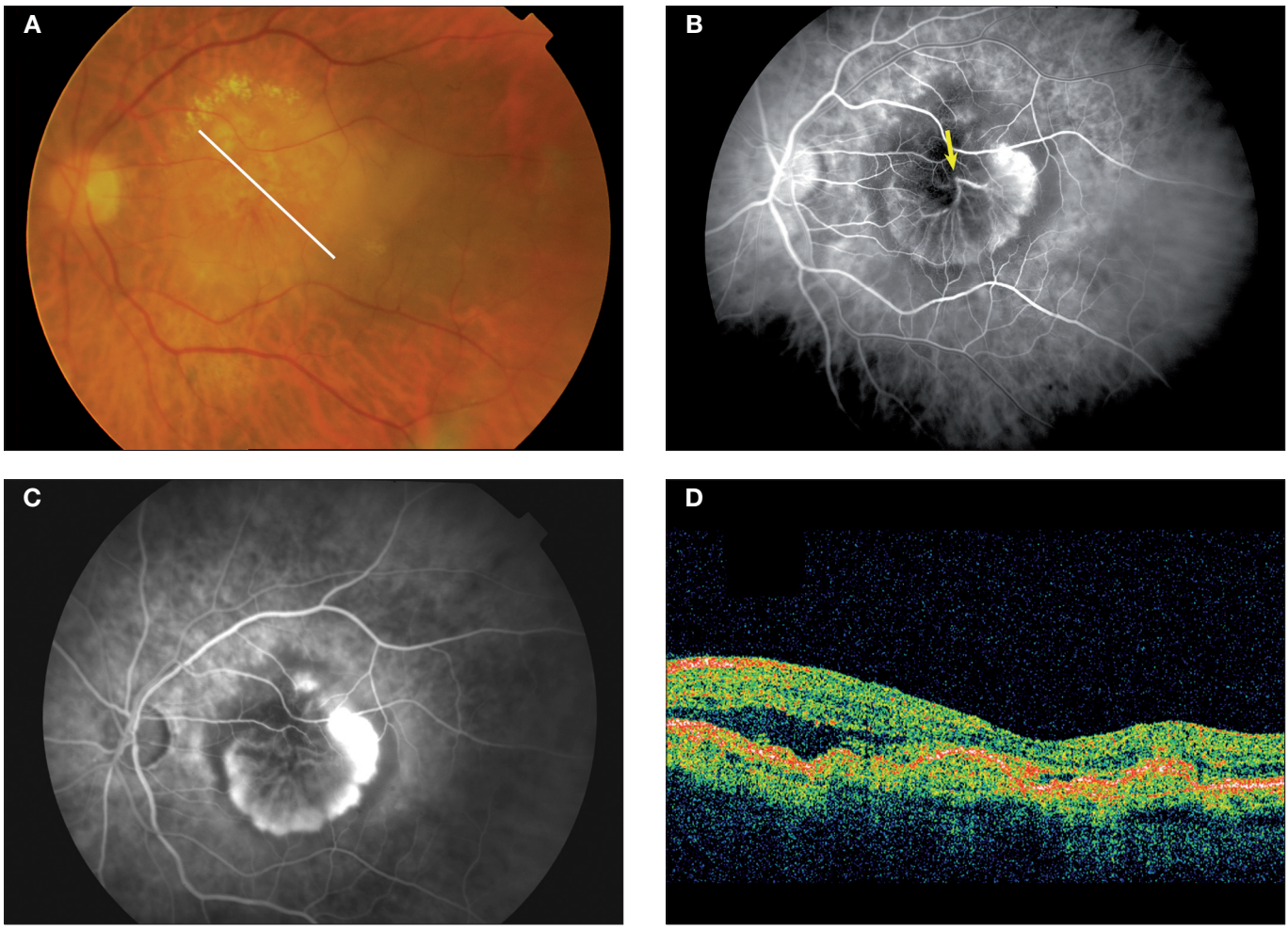

Fig. 1 - *Color photograph of a 73-year-old woman with subfoveal choroidal neovascularization (CNV).* **(A)** *Elevated sensory retina is seen before treatment. The line shows the direction of optical coherence tomography scan.* **(B)** *Fluorescein angiography (very early phase) shows a feeder vessel of CNV emerging from the fovea (arrow). The neovascular loops that connected with the feeder vessel have a shape similar to semicircular bicycle wheel.* **(C)** *Late fluorescein angiography phase shows the coexistence of occult CNV located in the upper part of the CNV lesion. Leakage of dye from the subfoveal neovascular loops is also evident.* **(D)** *Optical coherence tomography before treatment. Intraretinal fluid accumulation is evident.*

Case report

A 73-year-old woman with vision loss in her right eye for 3 years and gradual loss of vision in her left eye was diagnosed with bilateral AMD in February 2006. Visual acuity (VA) at presentation was 20/400 in the left eye. The patient had unsuccessful PDT treatment in the left eye 3 months before examination.

The left eye before and after intravitreal injection of bevacizumab was studied by FA and OCT (Stratus OCT III, Carl Zeiss, Dublin, CA, USA) and VA was checked with Snellen charts.

FA revealed the presence of a single large subfoveal feeder vessel in the left eye. The FV was connected with subfoveal CNV loops which had a shape similar to an incomplete bicycle wheel (Fig. 1). The identification of the feeder vessels was based on its appearance in the early phase of FA and on its relation to the choroidal circulation. Occult CNV lesions during the course of the angiography were also observed. The size of the CNV lesion was 3100 µm. Since the identification of FVs was so clear in early FA phases, high speed video ICG angiography was not performed. After an informed consent off-label pars plana intravitreal injection of bevacizumab (1.25 mg in 0.05 mL)

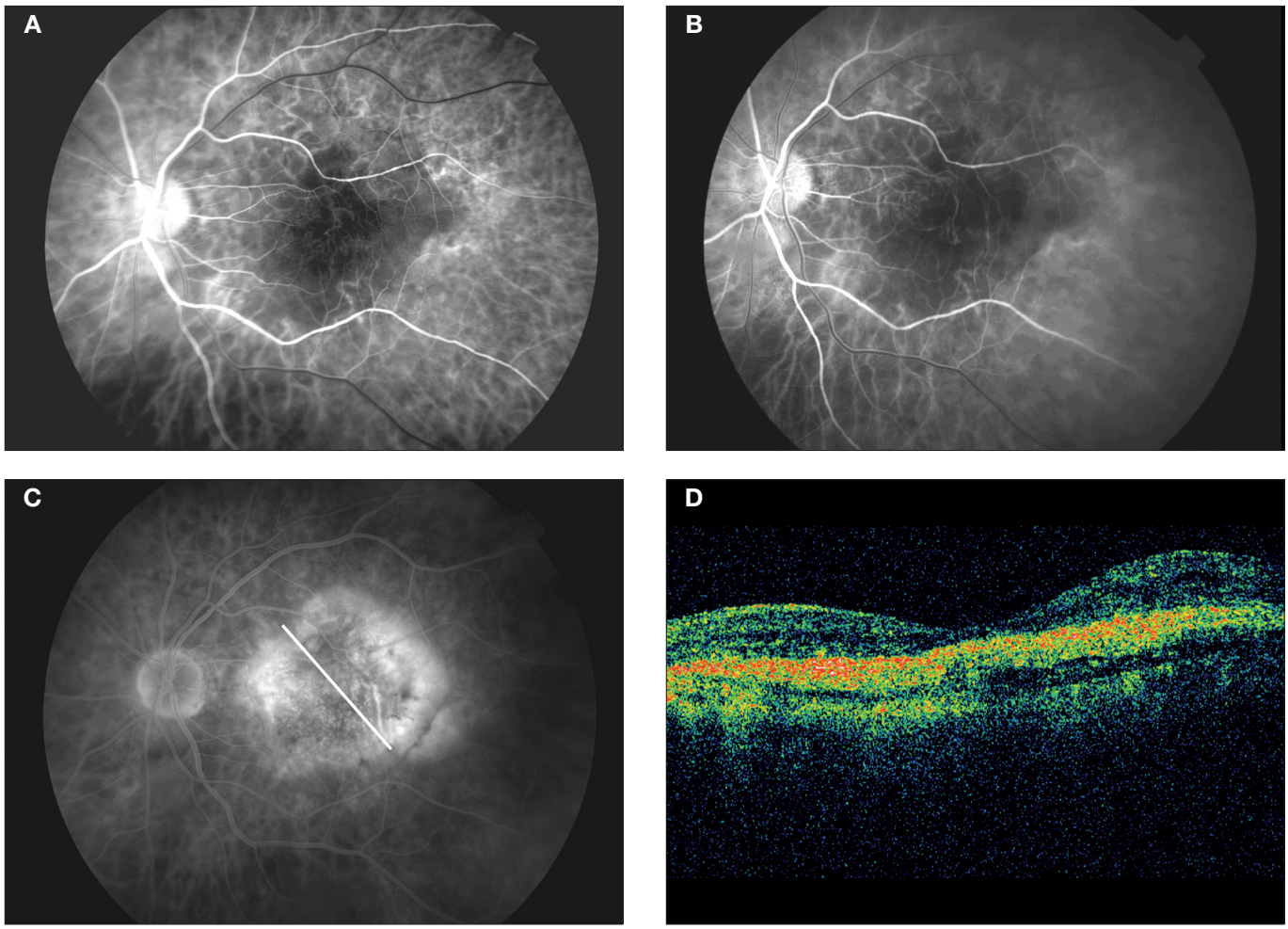

Fig. 2 - (A) *Fluorescein angiography (early phase) 4 weeks after bevacizumab injection. The feeder vessel and the connected subfoveal neovascularization have disappeared.* **(B)** *Fluorescein angiography in the final examination 13 months after the first bevacizumab injection: the feeder vessels and the connected subfoveal neovascular loops are not evident. Also there is no leakage from the occult choroidal neovascularization vessels.* **(C)** *Late phase fluorescein angiography in the final examination. No leakage is evident. The line shows the direction of optical coherence tomography scan.* **(D)** *Optical coherence tomography shows resolution of the intraretinal fluid. The retina in the fovea is very thin.*

was performed. Four weeks after the first injection occlusion of the FVs and the associated neovascular loops were noticed by FA (Fig. 2). Since some leakage from the occult vessels still existed and OCT showed a small amount of macular edema, reinjection of bevacizumab was performed. Four weeks after the second injection FVs remained occluded. OCT showed resolution of macular edema while VA improved from 20/400 at baseline to 20/100.

Since then and during monthly examinations, the patient attained stability regarding VA, macular thickness, and occlusion of the FVs. At the final examination in March 2007 after two injections the situation remained unchanged (Fig. 2).

DISCUSSION

The idea of treating subfoveal neovascular membranes by precise occlusion of its feeder vessels perhaps could be considered the best approach, but so far the applied treatments have given doubtful results.

Photocoagulation of feeder vessels resulted in various amount and duration of anatomic and functional stability.

Photocoagulation with argon laser caused full thickness retinal damage and choriocapillaries damage as well (2, 6). Diode laser that penetrates deeper and spares the overlying retina is poorly absorbed by hemoglobin and therefore the vascular closure proved to be difficult. PDT treatment resulted in partial occlusion of FVs and it is not recommended as a monotherapy for CNV (4).

Intravitreal injection of bevacizumab has shown promising results in the treatment of CNV secondary to AMD. Published studies have shown considerable improvement in VA and in reduction of retinal thickness even though follow-up is limited (5). An important question concerns the mechanism of occlusion of FVs by anti VEGF therapy in patients with AMD. The answer could be related to the fact that VEGF blockade induces long-term changes in the functional properties of the neovasculature of CNV. Moreover, the selective ablation of immature blood vessels, which has been observed in establishing human tumors after anti-VEGF therapy, can also offer a possible explanation (7). We are unaware of previous reports related to the bevacizumab treatment of FVs associated with CNV and did not find any reference to it in a computerized search using MEDLINE.

The fact that our case responded remarkably well to bevacizumab intravitreal injection for a period of 13 months after starting treatment suggests that this sort of treatment could be considered a therapeutic option in cases of FVs associated with subfoveal CNV secondary to AMD.

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