

SHORT COMMUNICATION

Intravitreal pegaptanib sodium for Irvine-Gass syndrome

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PURPOSE. To report the novel use of intravitreal pegaptanib sodium for the treatment of refractory cystoid macular edema (CME) following cataract extraction.

METHODS. A 72-year-old man presented with decreased visual acuity in his right eye 8 months after uncomplicated phacoemulsification cataract extraction and intraocular lens implantation. His best-corrected visual acuity (BCVA) was 20/125 in the affected eye, and fundus examination revealed CME despite 6 months of oral and topical indomethacin therapy. Fluorescein angiography (FA) showed leakage in the central region with no signs of neovascularization, and optical coherence tomography (OCT) confirmed the diagnosis with thickening of the fovea. Because of his history of glaucoma, the patient chose to be treated with intravitreal pegaptanib sodium 0.3 mg.

RESULTS. At the 1-week follow-up, BCVA was 20/25, and the FA and OCT revealed almost total resolution of the CME with no adverse sequelae. Six months postinjection, the patient's BCVA remained 20/25 with no recurrence of CME. Perimetry revealed a stable fixation within 4° with slight reduction of sensitivity.

CONCLUSIONS. Vascular endothelial growth factor inhibition with intravitreal pegaptanib sodium appears to be of benefit in the treatment of chronic refractory CME with improvement of visual acuity. Studies evaluating pegaptanib's use in this setting with long-term follow-up are warranted to confirm its efficacy and safety. (*Eur J Ophthalmol* 2008; 18: 138-41)

KEY WORDS. Irvine-Gass syndrome, Optical coherence tomography, Pegaptanib sodium, Pseudophakic cystoid macular edema

Accepted: September 21, 2007

INTRODUCTION

Macular edema is a major complication of various diseases and may also occur following cataract surgery (Irvine-Gass syndrome). Treatments for macular edema include nonsteroidal, anti-inflammatory drugs, corticosteroids, and acetazolamide, but resistant cases are common (1-3). We present an interventional case report describing a glaucomatous patient with refractory cystoid macular edema (CME) following cataract extraction, successfully treated with intravitreal injection of pegaptanib sodium (Macugen; OSI Eyetech Inc. and Pfizer Inc.).

Case report

A 72-year-old man presented with decreased visual acuity (20/125) in his right eye 8 months after uncomplicated phacoemulsification cataract extraction and intraocular lens implantation. The patient signed a comprehensive consent form according to Good Clinical Practice guidelines before proceeding with all examinations and treatments. Upon a complete ophthalmologic examination, including fundus examination showing CME, fluorescein angiography (FA) showing leakage in the central region, and optical coherence tomography (OCT-3, Humphrey-Zeiss, San Leandro, CA) showing the presence of CME

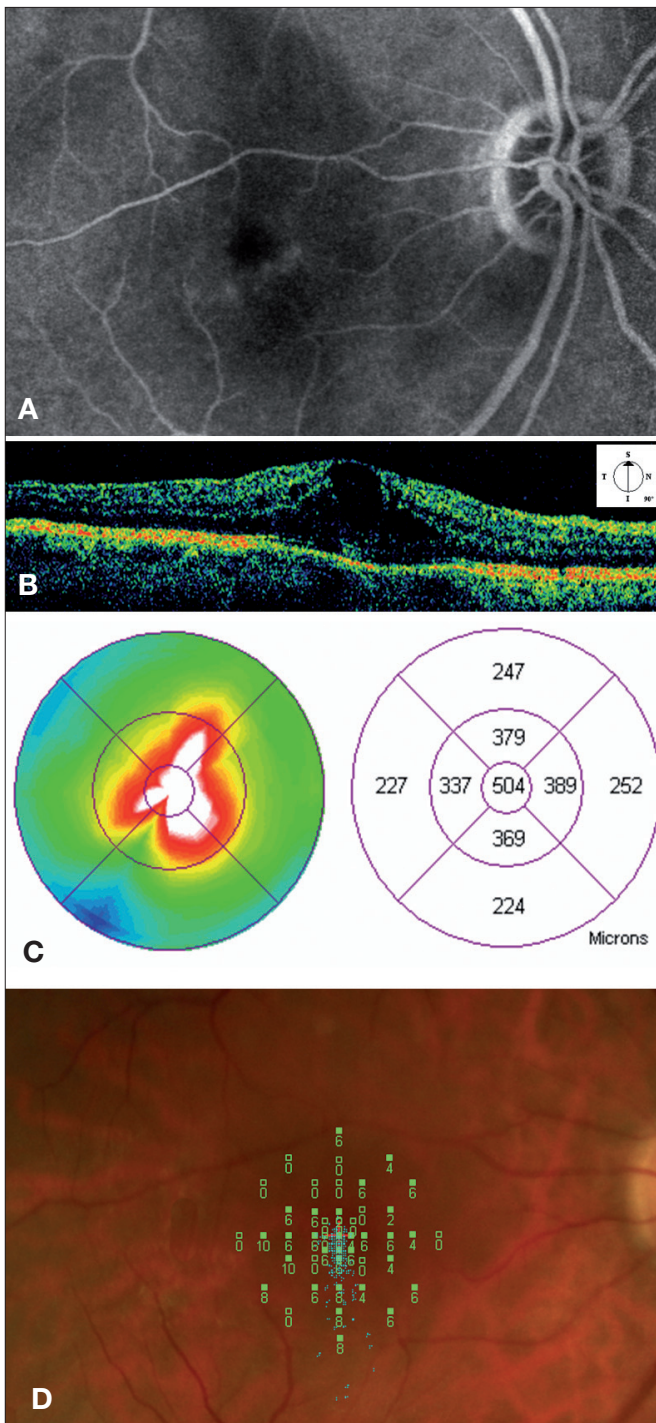


Fig. 1 - Fluorescein angiography frame showing leakage in the central region (top panel, **A**). Optical coherence tomography showing cystoid macular edema (middle upper panel, **B**) with thickening of the fovea (middle lower panel, **C**). Fundus-related perimetry (pattern macula 8° 0 dB with threshold strategy 4-2) revealing a stable fixation within 4° with a diffuse reduction of sensitivity (bottom panel, **D**).

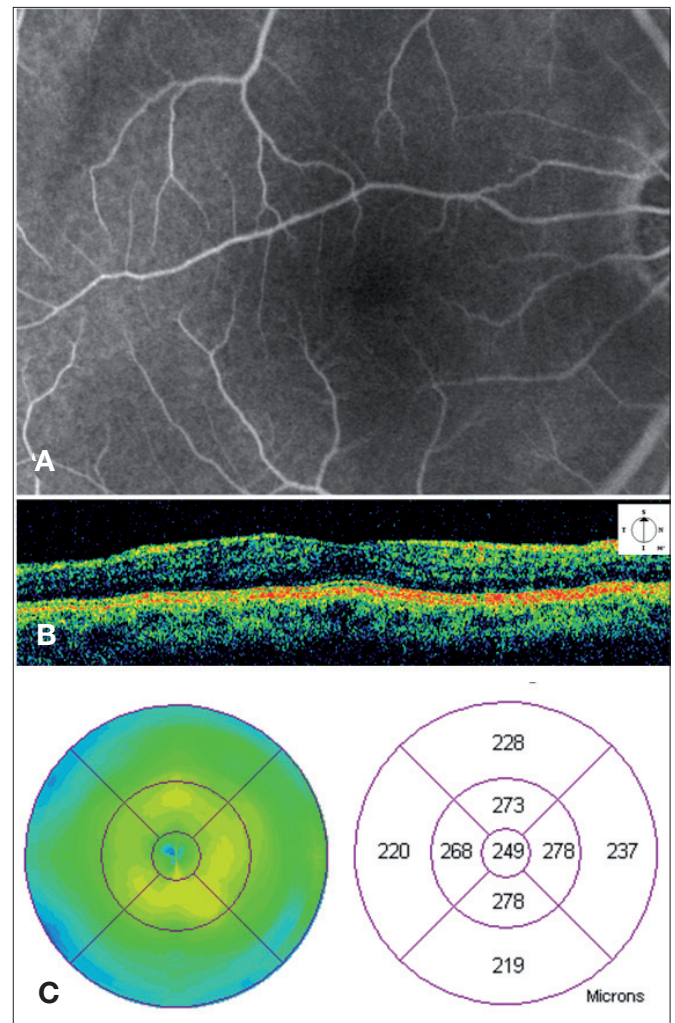


Fig. 2 - Fluorescein angiography (top panel, **A**) and optical coherence tomography revealing resolution of the cystoid macular edema 1 week after injection (middle and bottom panels, **B** and **C**).

with thickening of the fovea, the patient was diagnosed with Irvine-Gass syndrome. Although the patient was treated with oral (50 mg) and topical indomethacin 0.1% three times daily for 6 months, at the last follow-up visit best-corrected visual acuity (BCVA) remained 20/125 and fundus examination revealed unchanged CME. FA showed leakage in the central region typical of CME (Fig. 1A), and OCT scans confirmed the presence of CME (Fig. 1B) with thickening of the fovea (Fig. 1C). Fundus-related perimetry (MP-1 Micro Perimeter, Nidek Technologies, Padova, Italy), using pattern macula 8° 0 dB with 4-2 threshold strategy, revealed a stable fixation within 4° with a diffuse reduction of sensitivity (bottom panel, **D**)

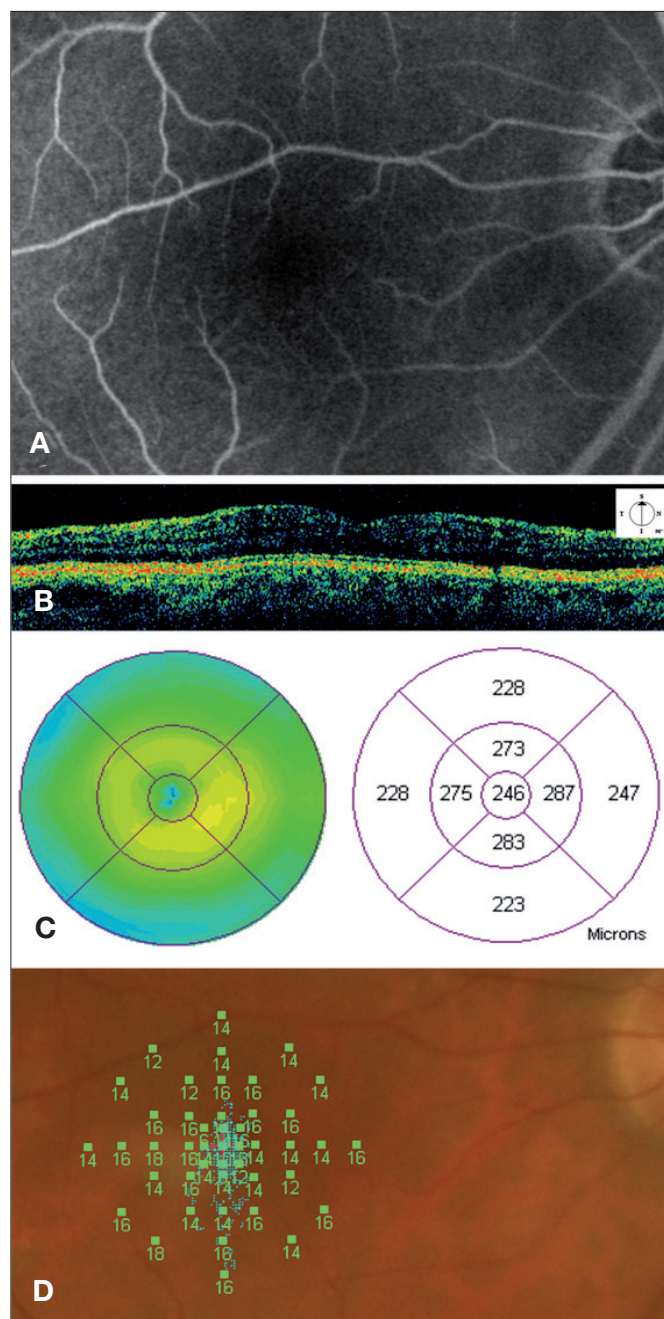


Fig. 3 - Fluorescein angiography (top panel, **A**) and optical coherence tomography revealing resolution of the cystoid macular edema 6 months after injection (middle upper and lower panels, **B** and **C**). Fundus-related perimetry (pattern macula 8° 0 dB with threshold strategy 4-2) revealing a stable fixation within 4° with slight reduction of sensitivity (bottom panel, **D**).

diffuse reduction of sensitivity (Fig. 1D). We excluded any possible cause of macular edema other than Irvine-Gass syndrome, such as choroidal neovascularization or

uveitis. The patient refused periocular or intravitreal corticosteroids because of the risks of the treatment in particular in glaucomatous patients. After discussing alternative treatment options and being presented with the option of intravitreal administration of pegaptanib sodium, an off-label application of pegaptanib, for a condition not covered by the original US Food and Drug Administration approval, the patient agreed to treatment. Intravitreal pegaptanib sodium (0.3 mg) was administered without complication. At the 1-week follow-up, BCVA was 20/25 in the right eye, and the FA and OCT revealed almost total resolution of the CME (Fig. 2, A–C). Six months after the injection, the patient's BCVA remained 20/25 with no recurrence of CME as evaluated by FA (Fig. 3A) and OCT (Fig. 3, B and C). Fundus-related perimetry using pattern macula 8° 0 dB with 4-2 threshold strategy revealed a stable fixation within 4° with slight reduction of sensitivity (Fig. 3D).

DISCUSSION

Phacoemulsification with intraocular lens implantation is currently one of the most frequently performed surgical procedures. Cystoid macular edema may occur as a complication in the postoperative course and may reduce visual acuity even if surgery is uncomplicated (4). Surgical trauma inflicted during ocular procedures triggers the arachidonic acid cascade responsible for the inflammatory response in the eye. Inhibition of prostaglandin synthesis and release reduces the inflammatory response induced by surgery and decreases the clinical symptoms of prostaglandin production (5). In some cases, the CME is refractory to usual medical treatments, including topical and oral nonsteroidals, as well as topical and oral corticosteroids. Triamcinolone, whether administered by subtenon or intravitreal injection, has been shown to be effective in cases of refractory pseudophakic CME (6, 7). However, the risk of glaucoma is considerable with the use of intraocular and periocular steroids (8).

Pegaptanib sodium is an RNA aptamer directed against the 165 amino acid isoform of vascular endothelial growth factor (VEGF165), and has proved to be an effective and safe treatment for both neovascular age-related macular degeneration (9) and diabetic macular edema (10). Viores et al (11) demonstrated that VEGF may contribute to blood–retinal barrier breakdown in a variety of ocular disorders, including CME following cataract extraction,

and that blockage of VEGF signaling may help to reduce some types of macular edema. Moreover, Mason et al (12) recently reported that intravitreal injection of bevacizumab, an antibody that binds all VEGF isoforms, successfully resolved two cases of CME following cataract extraction. Accordingly, we decided to inject 0.3 mg of pegaptanib sodium into the vitreous cavity of our patient. The CME resolved clinically, as well as by FA and OCT examinations, and the increase in visual acuity occurred within 1 week. There has been no recurrence of the CME nor have there been any complications from the intravitreal injection of pegaptanib sodium at 6 months follow-up. Based on our findings, intravitreal pegaptanib sodium seems to be of benefit in the treatment of chronic refractory CME with improvement of visual acuity. If confirmed in further studies, pegaptanib would provide a

safe and effective treatment option for patients with CME unresponsive to topical and oral nonsteroidal medications. Given that CME can recur and require further therapy, a longer follow-up with a large series of treated patients is suggested.

The authors have no proprietary interest in the materials used in this study.

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