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SHORT COMMUNICATION

Retinal pigment epithelial tear after ranibizumab therapy for subfoveal fibrovascular pigment epithelial detachment

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PURPOSE. *To describe the unusual complication of retinal pigment epithelial (RPE) tear after intravitreal ranibizumab (Lucentis®) for subfoveal fibrovascular pigment epithelial detachment (PED) and its effective management.*
METHODS. *Chart review for case report of RPE tear after ranibizumab.*
RESULTS. *An inferior RPE tear was documented by fluorescein angiography, fundus photography, and optical coherence tomography (OCT) 1 month after receiving repeat ranibizumab injection in the right eye of a patient with bilateral subfoveal fibrovascular PED. He had undergone multiple bevacizumab followed by ranibizumab injections for neovascular age-related macular degeneration (AMD) in both eyes, starting 6 months previously. Subsequent antivascular endothelial growth factor (VEGF) therapy improved vision of right eye from 20/200 to 20/40, despite RPE tear.*
CONCLUSIONS. *RPE tear may form after anti-VEGF therapy, including ranibizumab injection. Further anti-VEGF therapy may preserve or improve vision. To the authors' knowledge, this is first case report of effective suppression of neovascular activity with bevacizumab after an RPE tear following ranibizumab therapy. (Eur J Ophthalmol 2007; 17: 674-6)*

KEY WORDS. *Bevacizumab, Intravitreal injection, Lucentis®, Ranibizumab, Retinal pigment epithelial rips, Retinal pigment epithelial tears*

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INTRODUCTION

Retinal pigment epithelial (RPE) tears are known to develop spontaneously as well as after conventional laser and photodynamic therapy (1, 2). Multiple recent case reports also described RPE tears following anti-vascular endothelial growth factor (anti-VEGF) therapy, such as pegaptanib (3) and bevacizumab (4), and rarely after ranibizumab (Lucentis®) (5). We report a case of RPE tear after ranibizumab injection and its subsequent effective management.

Case report

A 75-year-old man presented in October 2006 with a history of two bevacizumab injections followed by two ranibizumab injections for each eye with subfoveal fibrovascular pigment epithelial detachment (PED) due to age-related macular degeneration performed elsewhere since 6 months ago. The neovascular activity had subsided in his left eye (LE), although there was persistent occult choroidal neovascularization associated with a subfoveal PED despite multiple anti-VEGF injections for his right eye (RE). The PED in his RE measured 16.9 mm^2. Best-corrected visual acuity (BVCA) was RE: 20/200, LE: 20/40. After documenting active neovascularization with fundus photography (FP), fluorescein angiography (FA), and optical coherence tomography (OCT), additional 0.5 mg of ranibizumab was injected into his RE. One month later, he presented with an RPE tear inferior to the fovea and persistent neovascular activity in RE, confirmed by

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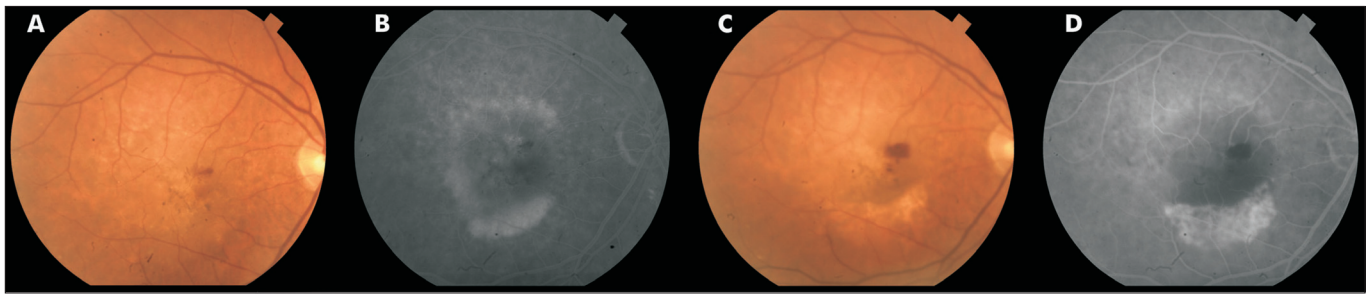

Fig. 1 - *A subfoveal fibrovascular pigment epithelial detachment with active neovascularization in the right eye is seen on* **(A)** *fundus photograph and* **(B)** *fluorescein angiogram before repeat ranibizumab injection in October 2006. A retinal pigment epithelial tear is seen inferior to the fovea on* **(C)** *fundus photograph, and* **(D)** *fluorescein angiogram, 1 month after repeat ranibizumab injection.*

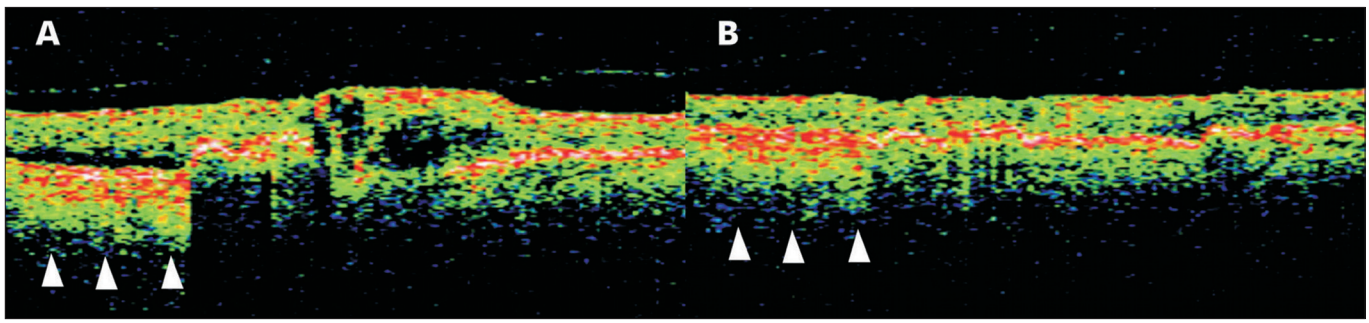

Fig. 2 - *Optical coherence tomography shows a* **(A)** *retinal pigment epithelial tear inferior to the fovea and residual submacular fluid 1 month after repeat ranibizumab injection, and* **(B)** *resolution of submacular fluid and neovascular activity following additional bevacizumab injection 4 months later (arrowheads: increased depth of signals due to absence of retinal pigment epithelium at site of tear).*

FA, FP, and OCT (Figs. 1 and 2). BCVA remained at 20/200, RE. Owing to the unfavorable response of his RE to ranibizumab injections, he requested switching back to bevacizumab therapy. His RE then received 2.5 mg of bevacizumab following the RPE tear. One week later, he reported marked visual recovery corresponding to progressive decrease in submacular hemorrhage and fluid in his RE. BCVA was 20/80, RE. One month later, there was further decrease in submacular fluid and hemorrhage in RE. BCVA was improved to 20/40, RE. Additional bevacizumab injections were given for both eyes 6 weeks later. The macula of both eyes remained dry and without recurrent neovascularization subsequently. The BCVA was 20/40 for each eye 6 months after his initial presentation.

DISCUSSION

The most likely mechanism of an RPE tear after anti-VEGF therapy is mechanical contraction of the choroidal neovascularization under the PED (4). Certain investigators have also proposed the modulation of biological activities and permeability of the choroidal neovascular membrane by the anti-VEGF therapy, leading to degradation of the RPE integrity (4). Foveal sparing and continued suppression of neovascular activity appeared to have contributed to visual preservation in this case. Our case shows that vision can be preserved or even improved with additional anti-VEGF therapy despite the RPE tear. It also highlights the vagary of responses to various anti-VEGF agents at different time points, as switching from ranibizumab back to bevacizumab resulted in rapid resolution of neovascular activity and marked visual improvement for this case. Although prior application of bevacizumab was followed by three ranibizumab injections before the RPE tear formed, we cannot rule out the partial contribution of bevacizumab to the RPE tear for this case. To our knowledge, this is the first case report of effective suppression of neovascular activity with bevacizumab after

formation of an RPE tear following ranibizumab therapy. Further study is warranted to determine the incidence, visual effects, and therapeutic options for RPE tears after ranibizumab therapy.

The authors have no financial or proprietary interest in any products or techniques mentioned in this report.

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