



Figure 1 Fundus photographs (left-hand column), fluorescein angiography (FA) results, and optical coherence tomography (OCT) results (right-hand column) along the horizontal meridian from temporal to nasal, revealing the presence of a subfoveal choroidal neovascular membrane. Preinjection (top) and 4 weeks postinjection photographs (bottom) are shown. FA shows staining of the membrane, and OCT shows shrinkage of the neovascular membrane with resolution of intraretinal oedema following intravitreal bevacizumab therapy. Retinal thickness measured by OCT at baseline (330μ ; top right) and after 4 weeks (221μ ; bottom right).

(Figure 1). The central macular thickness (CMT) on OCT was 330μ OD and 201μ OS.

After a written consent was signed by the patient, an off-label intravitreal bevacizumab injection (1.25 mg) was given in the right eye. At 4 weeks, BCVA improved to 20/50 OD and FA showed staining of the neovascular membrane. The CMT on OCT decreased to 221 μ OD with a reduction in lesion size (Figure 1). After 6 months, the vision was maintained at 20/50 OD with a scarred subfoveal membrane.

Comment

Choroidal ruptures are breaks in the choroid, Bruch's membrane, and the retinal pigment epithelium that occur from blunt ocular trauma. In 15–30% of patients, CNV may occur and lead to haemorrhagic or serous macular detachment with concurrent central vision loss. The formation of CNV is strongly associated with proximity of the rupture to the fovea and the length of the rupture.⁴ Photodynamic therapy has been reported for the management of such membranes;⁵ however, certain limitations like high expenses involved and post-treatment risk of vision loss are present.

In this case, bevacizumab not only hastened the regression of the neovascular membrane but also provided superior visual outcome. Intravitreal bevacizumab is also well tolerated and no adverse effects were observed. The results observed in this case are provocative and require further investigation.

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Retinal pigment epithelium tear after intravitreal bevacizumab injection for polypoidal choroidal vasculopathy

A retinal pigment epithelium (RPE) tear is a well-recognized complication of pigment epithelial detachments (PEDs) in age-related macular degeneration (AMD), as well as in polypoidal choroidal vasculopathy (PCV).¹ Recently, several reports have recognized that an RPE tear can occur after anti-vascular endothelial growth factor (VEGF) therapy such as bevacizumab, ranibizumab, and pegaptanib for AMD.² In this report, we present a patient with PCV who developed an RPE tear after an intravitreal bevacizumab injection. On the basis of serial findings of optical



Figure 1 Findings of serial optical coherence tomography in the right eye after intravitreal bevacizumab injection. The image was along the arrow in Figure 2b. (a) Pre-avastin OCT showed subretinal fluid and large PED. Steeply protruding RPE (arrowhead) possibly corresponded to the part of polypoidal lesion. (b) After the first injection, PEDs were markedly flattened. (c) Before the second injection, an obvious dimple (arrow) was found between diffuse smooth and wavy RPE detachments. (d) After the second injection, the RPE tear was found. Parts of retracted RPE band were visible. An increased signal in the deep choroidal layer probably caused by the loss of overlying RPE layer was also noted (between arrowheads).

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coherence tomography, a possible mechanism was suggested as being responsible for the formation of RPE tears.

Case report

An 80-year-old man was found to have bilateral subfoveal PCV with earlier photodynamic therapy in the left eye and without any treatment in the right eye. Visual acuity was 20/40 in the right eye and 1/60 in the left eye. In the right eye, optical coherence tomography showed some subretinal fluid and a large PED with focal wavy configurations (Figure 1a). Ophthalmoscopy (Figure 2a) and fluorescein angiography (Figure 2b) revealed exudative changes and serous subfoveal PED with late leakage. Indocyanine green angiogram showed a branching vascular network (arrowhead) and polypoidal lesions (arrows) at early phase (Figure 2c) and late phase (Figure 2d). On the basis of the findings of an indocyanine green angiogram, the polypoidal lesions are located on the underside of the more irregular, contracted portion of the RPE. Part of the polypoidal lesion probably resided in the protruding portion of the vertical scan image of the optical coherence tomography findings (Figure 1a, arrowhead). After informed consent was obtained, the patient received bilateral intravitreal injections of 2.5 mg bevacizumab in 0.1 ml. He noted a significant improvement of vision to 20/30 in the right eye over the next few weeks, and subretinal fluid as well as PED decreased significantly (Figure 1b). However, 4 months later, his right vision declined to 20/50. Recurrent subretinal fluid and marked elevation of PED were revealed by optical coherence tomography. Within one large PED, two separate areas with

different features could be observed (Figure 1c). The upper part seemed wavy and contracted, and the lower part seemed smooth and distended. There was an obvious dimple (arrow) between these two features of PED. Another bevacizumab injection was given in the right eye. The patient noted worsening of metamorphopsia 1 week after the injection. Reevaluation 4 weeks after the injection revealed visual acuity of 20/70 in the right eye and an RPE tear was noted (Figure 2e). Fluorescein angiography proved the presence of an RPE tear with macula involvement (Figure 2f). Indocyanine green angiography revealed that the earlier polypoidal lesion and the branching choroidal vessels became less apparent at early phase (Figure 2g) and late phase (Figure 2h). Using optical coherence tomography, an RPE tear was disclosed at the edge of an earlier smooth feature of PED. The RPE band showed a wavy and retracted configuration, as well as a thickened appearance with adjacent RPE loss (Figure 2d).

Comment

RPE tears are reported in 0.8–2.2% of patients with AMD after intravitreal injection of bevacizumab.^{3,4} They usually occur in large irregular PEDs associated with choroidal neovascularization.^{2,3} On the basis of an association between VEGF and PCV revealed by histopathologic findings, Gomi *et al*⁵ recently reported patients experiencing relatively better vision and reduction of subretinal fluid after intravitreal bevacizumab in PCV. Eyes with PCV are usually accompanied by serosanguineous PEDs



Figure 2 (a) Baseline funduscopic examination of the right eye showed exudative changes with a serosanguineous PED. (b) Fluorescein angiogram revealed serous subfoveal PED with late leakage. (c) Indocyanine green angiogram indicated a branching vascular network (arrowhead) and polypoidal lesions (arrows) at early phase. (d) Indocyanine green angiogram revealed a hot spot associated with polypoidal lesions at late phase. (e) The RPE tear after intravitreal bevacizumab injection for PCV. Funduscopic examination of the right eye showed a tear of the RPE with mild exudative change. (f) Fluorescein angiogram showed an RPE tear within the macula, with a window defect adjacent to hyperpigmented subretinal tissue. (g) Indocyanine green angiography revealed that the earlier polypoidal lesion and the branching choroidal vessels became less apparent at early phase and (h) late phase.

and may have potential risks similar to AMD with large PEDs after anti-VEGF therapy. However, we are unaware of any earlier reports of an RPE tear occurring after intravitreal bevacizumab injections for PCV.

Several mechanisms for the generation of RPE tears had been proposed. Chuang and Bird⁶ found that the Bruch's membrane and hydrophobic deposits like drusen might act as a barrier to fluid flow. Gass⁷ suggested that the tangential force exerted by the fibrovascular contraction of choroidal neovascular membrane might be responsible for RPE tears. Moreover, VEGF played a role in RPE barrier dysfunction.⁸ The maintenance of the tight junction could be disrupted by VEGF. In the development of PED in exudative AMD, the distending force caused by rapid fluid accumulation intervenes with the contractile force of fibrovascular membrane. Treatments such as photodynamic therapy, laser photocoagulation, and anti-VEGF treatment may also induce an acute contraction of the fibrovascular membrane and increase the contractile force.¹⁻³ These two opposite forces may lead to the separation of RPE from Bruch's membrane and eventually cause the already weakened RPE to tear. As a larger and higher PED seemed to be more disposed to RPE tear,^{3,9} and the PEDs of PCV were found to be generally larger than the PEDs of the other aetiology,^{1,4} it is possible that PCV may be more prone to RPE tear. In our case, optical coherence tomography findings disclosed a highly irregular large PED with an obvious dimple, which might represent a boundary between the distended and the contracted parts of the PED. These two features within one PED showed an imbalance between hydrostatic and tangential forces. This may be an important indicator of the formation of RPE tears.

In summary, in this report, we showed that an RPE tear occurred after intravitreal bevacizumab injections for PCV with large irregular PED. Further studies are needed to evaluate the incidence and the amount of risk to eyes of developing RPE tears in polypoidal lesions with anti-VEGF therapy.

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Sir,

Small tarsal plates causing recurrent lower lid entropion in a young adult

Lower lid entropion is commonly associated with several age-related structural changes.^{1,2} Small tarsal plate is a less well known factor in its pathogenesis.² We describe a young patient in which significantly small tarsal plates were responsible for development of bilateral recurrent entropion, which proved difficult and challenging to manage.

Case report

An 18-year-old Caucasian female was referred with bilateral recurrent lower lid entropion (Figure 1) for the past 3 years. Previously she had everting sutures and lower lid retractor advancement at another hospital that failed. There was no conjunctival cicatrisation or forniceal shortening. The remaining ocular examination was normal. She had no features of congenital ectodermal dysplasia and no other associated craniofacial anomaly.

Bilateral lower lid hard palate grafts were performed. Intra-operatively the height of tarsal plates was measured as 2.5 mm in lower lids and 5.5 mm in upper lids (Figure 2). Resolution of the entropion was