($-16 + -1 \times 90$) OS. Anterior segment biomicroscopy revealed early cataract bilaterally. Fundi showed features suggestive of high myopia OU; OS additionally had a macular hole with posterior pole RD, confirmed by optical coherence tomography (OCT3, Carl Zeiss Meditec., Dublin, CA, USA; Figure 1a and b). With informed consent of the patient and approval of the Institutional Review Board, she underwent simultaneous cataract extraction with vitrectomy, internal-limiting-membrane (ILM) peeling using triamcinolone acetate, and perfluoropropane (16%) tamponade OS. Postoperatively, retina was reattached completely; the macular hole closed within a central patch of geographic atrophy (Figure 1c and d). BCVA gradually improved to 4/60. A small pocket of intraretinal fluid appeared near the hole edges 4 months postoperatively, but disappeared on subsequent follow-up (Figure 1e). She maintained the status quo on annual postoperative visits till the fourth year, when macula was observed to bloat up into a foveoschisis on OCT (Figure 1f); fundus appearance and BCVA remained unchanged. The foveoschisis remained stable over the next 6 months.

Comment

Tornambe⁴ suggested that a macular hole can occur in the absence of vitreous traction; microtrauma to the fovea during posterior vitreous detachment results in foveal hydration, deroofed by centrifugal tangential traction of ILM. A similar mechanism could have operated in this postvitrectomy macular hole, which closed with a central defect—a common observation in myopic staphylomata with RD.⁵ Its discontinuous edges were more vulnerable to fluid ingress due to weak adhesion to the degenerated retinal pigment epithelium, which would also result in poor pumping out of the intraretinal fluid. Indeed, minimal intraretinal fluid was noted postoperatively, which, however, resolved spontaneously. Because of the limitations of OCT imaging in a myopic staphyloma, we cannot rule the presence of a subtle epiretinal membrane producing the schisis. However, as both anteroposterior and tangential tractions were surgically eliminated, this possibility appears unlikely. For the same reason, we believe that foveoschisis would probably not progress to reopening of the macular hole over further follow-up.⁶ This unusual development—never reported previously as a postsurgical event—endorses the recent view that vitreous traction may not be critically important in pathogenesis of myopic foveoschisis.^{2,3} Further implications and actual incidence of this seemingly rare event can only be assessed by large scale interventional case studies with long-term OCT-documented follow-ups.

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Sir, Intravitreal bevacizumab for subfoveal choroidal neovascularization secondary to traumatic choroidal rupture

Bevacizumab, a humanized monoclonal antibody to vascular endothelial growth factor, has been given as an intravitreal injection for choroidal neovascularization (CNV) secondary to age-related macular degeneration,¹ myopia,² and angioid streaks³ with promising functional results. Here, we present a case of subfoveal CNV secondary to traumatic choroidal rupture, which regressed with intravitreal bevacizumab. We are unaware of such a report in world literature.

Case report

A 25-year-old woman presented with diminution of vision in her right eye for 1-month duration. The patient had sustained a blunt injury in the right eye with a ball 4 months back.

The best-corrected visual acuity (BCVA) was 20/200 OD and 20/20 OS. Anterior segment examination was normal bilaterally. The intraocular pressures were 10 mmHg bilaterally. Ophthalmoscopic examination of the right eye revealed a curvilinear choroidal rupture just temporal to the fovea with a small, orange-coloured subfoveal lesion and associated subretinal haemorrhage (Figure 1). Left eye fundus was normal.

Fluorescein angiography (FA) showed transmission hyperfluorescence corresponding to the choroidal rupture and an associated subfoveal hyperfluorescent lesion with late leakage of dye in the right eye. Optical coherence tomography (OCT) revealed the presence of a subfoveal CNV with intraretinal oedema in the right eye

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 (330 μ ; top right) and after 4 weeks (221 μ ; bottom right).

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