Sir,

Foveoschisis after vitrectomy for myopic macular hole with secondary retinal detachment

Macular foveoschisis is a recently identified cause for visual loss in pathological myopia.[1] Factors contributing to foveal splitting include vitreoretinal traction, chorioretinal atrophy, axial elongation, and recently, arteriolar traction.[2,3] We report a foveoschisis that occurred many years after surgery for retinal detachment (RD) associated with a myopic macular hole.

Case report

A 49-year-old lady presented to us with decreased vision OS for 2 months. Best-corrected visual acuities (BCVAs) were 6/12 ($-11 + -2 \times 90$) OD and hand motions

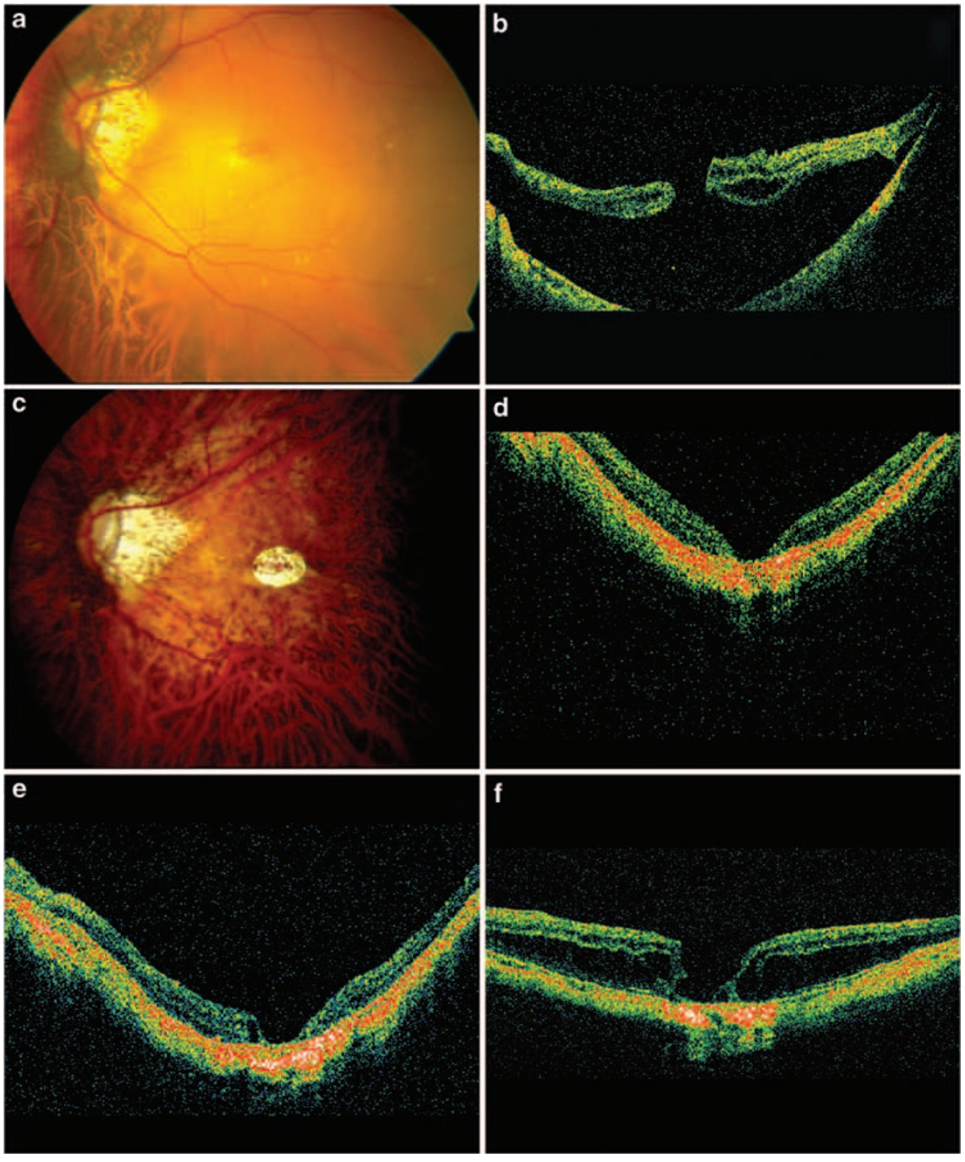

Figure 1 Postsurgical developments in a myopic macular hole with associated retinal detachment. (a) Posterior pole of the left eye shows a tilted optic disc with myopic crescent, and a horizontally oval macular hole with extensive subretinal fluid, which masks the retinal pigment epithelial (RPE) atrophy around the hole. Subretinal precipitates suggest chronicity of the retinal detachment. (b) Optical coherence tomogram (vertical 10 mm scan) shows the macular hole, the surrounding sensory retinal detachment, and the posterior staphyloma. (c) One month after vitrectomy, the retina is reattached with closure of the macular hole. The central geographic patch of RPE atrophy is now prominent. (d) Repeat OCT (10 mm horizontal scan) confirms the anatomical outcomes; and reveals the residual central defect between the flattened edges of the macular hole ('flat open' configuration), as well as the presence of posterior staphyloma. Central sub-RPE hyperreflectivity is suggestive of geographic atrophy. (e) A small blister is evident within the nasal edge of the closed hole as early as 4 months on repeat mode OCT, which, however, disappeared on subsequent visits. (f) In a 4-year review, a full-blown foveoschisis has developed, with irregular but attached central edges. Status quo continued for 6 months.

(−16 + −1 × 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 acetonide, 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

Tornambe[4] 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.

References

1 Takano M, Kishi S. Foveal retinoschisis and retinal detachment in severely myopic eyes with posterior staphyloma. *Am J Ophthalmol* 1999; **128**: 472–476.
2 Wu PC, Chen YJ, Chen YH, Chen CH, Shin SJ, Tsai CL *et al.* Factors associated with foveoschisis and foveal detachment without macular hole in high myopia. *Eye* 2009; **23**: 356–361.
3 Ikuno Y, Gomi F, Tano Y. Potent retinal arteriolar traction as a possible cause of myopic foveoschisis. *Am J Ophthalmol* 2005; **139**: 462–467.
4 Tornambe PE. Macular hole genesis: the hydration theory. *Retina* 2003; **23**: 421–424.
5 Ikuno Y, Sayanagi K, Oshima T, Gomi F, Kusaka S, Kamei M *et al.* Optical coherence tomographic findings of macular holes and retinal detachment after vitrectomy in highly myopic eyes. *Am J Ophthalmol* 2003; **136**: 477–481.
6 Gaucher D, Haouchine B, Tadayoni R, Massin P, Erginay A, Benhamou N *et al.* Long-term follow-up of high myopic foveoschisis: natural course and surgical outcome. *Am J Ophthalmol* 2007; **143**: 455–462.

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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 streaks[3] 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