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 Bird[6] found that the Bruch's membrane and hydrophobic deposits like drusen might act as a barrier to fluid flow. Gass[7] 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

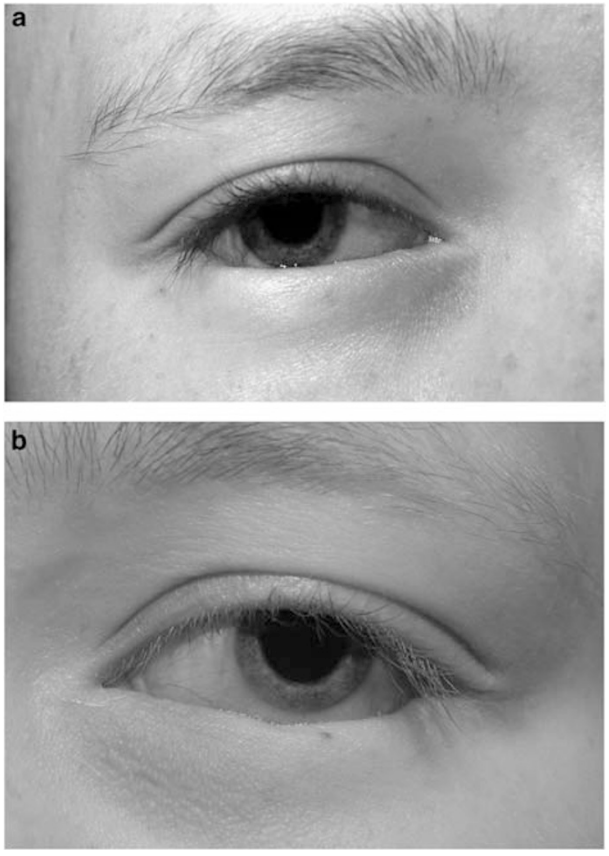

Figure 1 (a and b) Right and left eye photographs showing lower lid entropion.

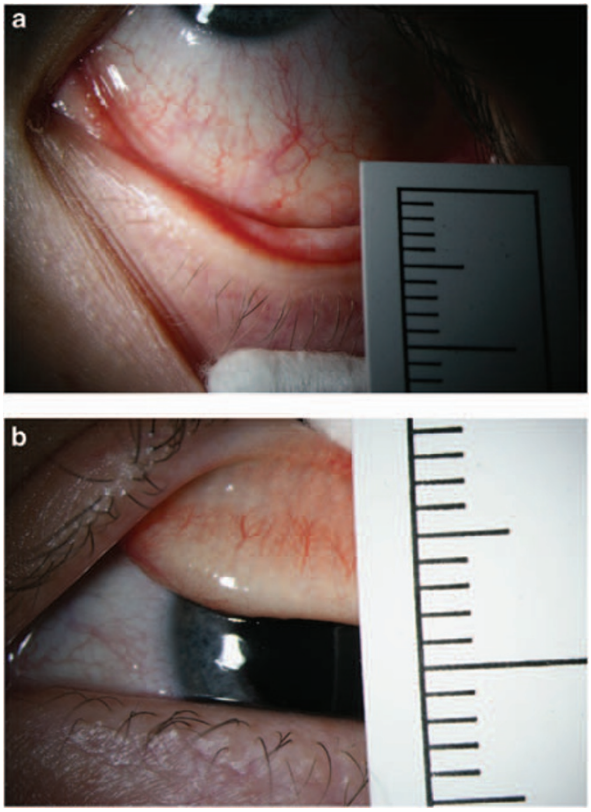

Figure 2 (a) Left lower eye lid photograph illustrating the tarsal plate height of 2.5 mm. (b) Right upper eye lid photograph displaying the tarsal plate height of 5.5 mm.

achieved and remained for 6 months before recurrence. The previous grafts were then augmented with auricular cartilage grafts. Four months later the entropion recurred; mild blepharospasm and horizontal lower lid laxity were noted bilaterally. Bick's procedures with everting sutures were performed. She had another recurrence after 2 months, which was managed with intra-orbicularis injections of Botulinum toxin. She has had no further recurrence for the past 4 months.

Comment

The average height of the tarsus in females is 8.54 mm in upper and 5.84 mm in lower lids.[2] Atrophy of the tarsal plate is known to cause involutional entropion.[2,3] Small tarsal plates in our patient contributed to the development of the entropion which manifested later with increased orbicularis tone and over-riding of orbicularis components. Clinically, this manifested at the age of 15 years and may well be related to the growth spurt during puberty. This case highlights a small tarsal plate as a causative factor for recurrent entropion in a young patient, which, to the best of our knowledge, has not yet been reported. The management of such patients is complex and may require a systematic approach.

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