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The authors have no proprietary or financial interest in any material or device mentioned.

Eye (2009) **23,** 1237–1238; doi:10.1038/eye.2008.137; published online 16 May 2008

Sir,

Intravitreal pegaptanib in severe proliferative diabetic retinopathy leading to the progression of tractional retinal detachment

The antivascular endothelial growth factor aptamer pegaptanib may induce short-term regression of retinal neovascularisation secondary to diabetes.[1] We present two diabetic patients who underwent intravitreal pegaptanib for persistent retinal neovascularisation and developed the progression of a previously stable tractional retinal detachment (TRD).

Case reports

Case 1

A 26-year-old woman with severe proliferative diabetic retinopathy underwent extensive scatter laser photocoagulation (PRP) leading to the regression of neovascularisation and a localised right inferotemporal TRD.

After 6 months, the TRD appeared unchanged, but fresh vitreous and preretinal haemorrhages were observed. Informed consent was obtained for intravitreal pegaptanib (0.3 mg/0.9 ml).

Ten days after injection, the TRD appeared stable (Figure 1). By 5 weeks, TRD progression had resulted in macular elevation and reduced visual acuity from 6/12 to 6/36 (Figure 2).

The patient underwent vitrectomy, delamination, endolaser, and sulphur hexafluoride gas with minimal intraoperative haemorrhage. After 2 months, the visual acuity was 6/12.

Case 2

A 48-year-old woman with proliferative diabetic retinopathy underwent extensive PRP and developed TRD associated with regressed inferotemporal neovascularisation. After 8 months, active neovascularisation recurred although the TRD remained unchanged (Figure 3a and b).

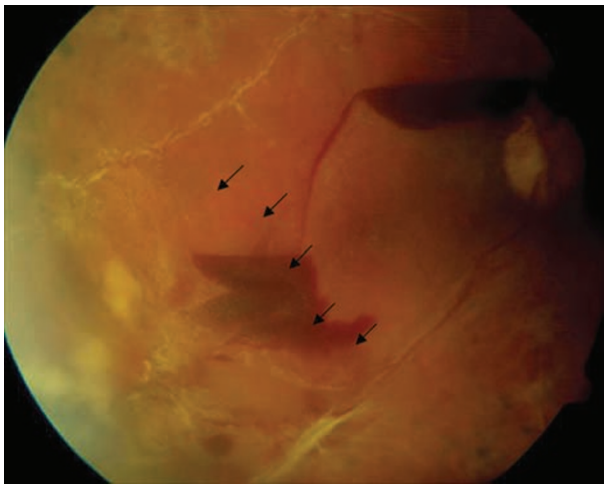

Figure 1 Right fundus photograph of patient 1, 10 days after intravitreal pegaptanib injection. Vitreous and preretinal haemorrhages are present. There is a stable tractional retinal detachment at the inferotemporal arcade (arrows indicate the extent).

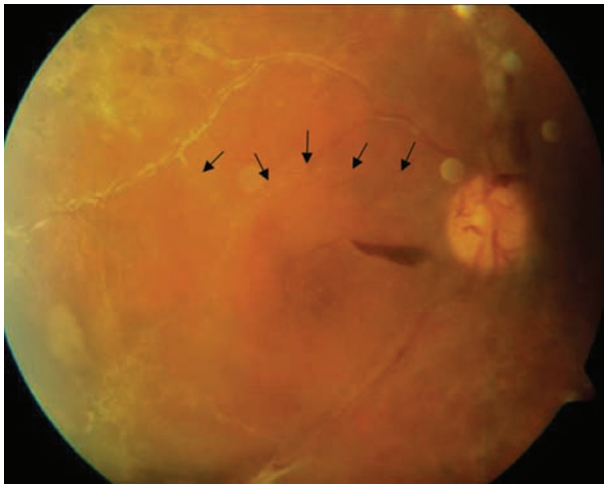

Figure 2 Right fundus photograph of patient 1, 5 weeks after intravitreal pegaptanib injection. The vitreous and preretinal haemorrhages are greatly reduced and the neovascularisation has regressed. However, the tractional retinal detachment has extended to involve the fovea (arrows show the extended edge).

Informed consent was obtained for intravitreal pegaptanib (0.3 mg/0.9 ml). Three weeks later, although neovascularisation and preretinal haemorrhage had improved, the TRD had progressed and visual acuity reduced from 6/36 to counting fingers (Figure 4a and b). The patient underwent right vitrectomy, delamination, and octafluoropropane gas. Minimal intraoperative haemorrhage permitted a low infusion pressure during surgery. After 6 weeks, the visual acuity was 6/12.

Comment

The preexisting TRD had remained stable for 6 months and 8 months in cases 1 and 2, respectively before progressing within the weeks of intravitreal pegaptanib.

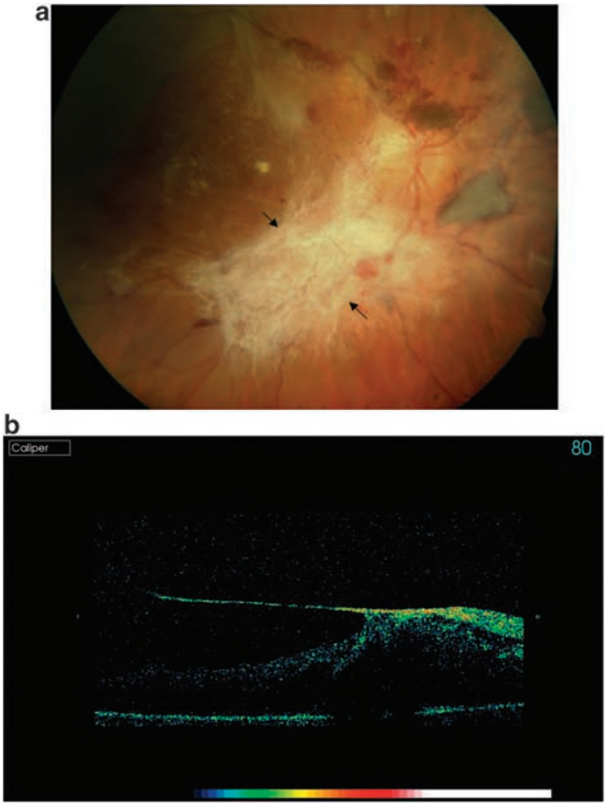

Figure 3 (a) Right fundus photograph of patient 2, 3 weeks before intravitreal pegaptanib injection. Preretinal haemorrhage and neovascularisation are present. There is extensive preretinal fibrovascular tissue (arrows show the width) and tractional retinal detachment involving the inferotemporal arcade. (b) Right optical coherence tomogram of the inferotemporal region 3 weeks before intravitreal pegaptanib injection (in presence of vitreous haemorrhage).

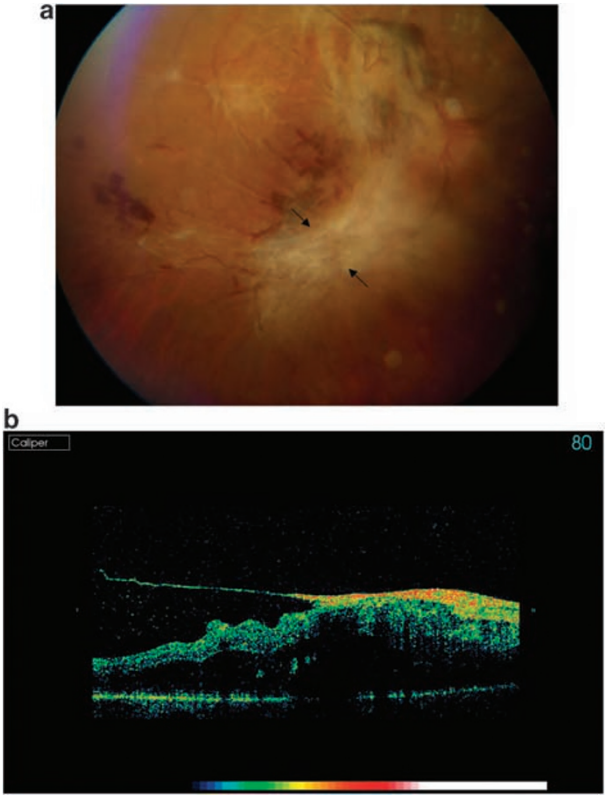

Figure 4 (a) Right fundus photograph of patient 2, 3 weeks after intravitreal pegaptanib injection. Preretinal haemorrhage and neovascularisation have largely resolved. The tractional retinal detachment has progressed to involve the fovea and the fibrovascular tissue has contracted (arrows indicate the shortened width) with macular traction folds. (b) Right optical coherence tomogram of the same inferotemporal region 3 weeks after intravitreal pegaptanib injection showing increased preretinal fibrous tissue and retinal traction.

Reports exist of TRD progression following intravitreal bevacizumab in proliferative diabetic retinopathy.[2,3] However, there are currently no reports in the literature associating intravitreal pegaptanib with TRD progression.

Intravitreal pegaptanib did, however, result in less intraoperative haemorrhage than expected. This reduced the risk of further circulatory compromise in a diabetic due to prolonged elevated intraocular pressure while attempting to achieve haemostasis.

We feel that the rapid progression of the TRD in these patients is largely attributable to the pegaptanib. Although intravitreal pegaptanib may potentially be a useful management tool in severe proliferative diabetic retinopathy, in patients with preexisting TRD, we would advise caution and careful monitoring following its use.

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Radhika Krishnan would like to declare on behalf of the authors that they have no competing interests regarding this report and have not received any public or private support in relation to it.

Eye (2009) **23,** 1238–1239; doi:10.1038/eye.2008.179; published online 13 June 2008