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SHORT COMMUNICATION

Spontaneous resolution of vitreomacular traction following ranibizumab (Lucentis) injection

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PURPOSE. *Spontaneous resolution of vitreomacular traction syndrome in diabetic patients is a rare phenomenon that has been poorly described in the literature.*
METHODS. *Case presentation.*
RESULTS. *The authors present a case of spontaneous resolution of vitreomacular traction following intravitreal administration of ranibizumab.*
CONCLUSIONS. *In patients with vitreomacular traction syndrome and diabetic macular edema, the combination of the possible vitreous liquefaction and mechanical increase of vitreous volume caused by an intravitreal injection with a degree of reduction in retinal thickness caused by the effect of vascular endothelial growth factor inhibition could play a role in the resolution of vitreomacular traction. (Eur J Ophthalmol 2008; 18: 301-3)*

KEY WORDS. *Vitreomacular traction, Spontaneous resolution, Macular edema, Ranibizumab, Lucentis*

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INTRODUCTION

Although vitreomacular traction syndrome (VTS) comprises a well-defined clinical entity, only a few cases of VTS on the background of diabetic macular edema (DME) have been described in the literature (1, 2).
We report a case of spontaneous resolution of vitreomacular traction following intravitreal administration of ranibizumab in a patient with proliferative diabetic retinopathy (PDR) and macular edema.

Case report

A 69-year-old Caucasian diabetic man presented to our department in February 2007 with a 4-day history of visual loss in his left eye. He had a history of PDR and for that he started treatment with panretinal photocoagulation (PRP) in September 2006. Best-corrected visual acuity (BCVA) was 20/200 in his left eye (LE). Clinical examination revealed significant macular edema and preretinal and vitreous hemorrhages nasal to the optic disk and inferiorly respectively. Fluorescein angiography (FA) showed new vessels elsewhere (NVE) and a few PRP shots within and out of the retinal arcades (Fig. 1A). Optical coherence tomography (OCT) showed a thickened fovea with cystoid changes. The thick, hyperreflective, and taut posterior hyaloid was partially detached from the posterior pole exerting traction on the fovea. The steep hyaloid curve and the thickened cystoid fovea were suggestive of vitreomacular traction (Fig. 1B). Although pars plana vitrectomy was recommended, the patient refused surgical treatment and therefore 0.5 mg ranibizumab was administered intravitreally (using a 30-gauge needle) with a view to continue the PRP therapy 4 to 6 weeks following the injection.
Six weeks after the intravitreal injection, the patient re-

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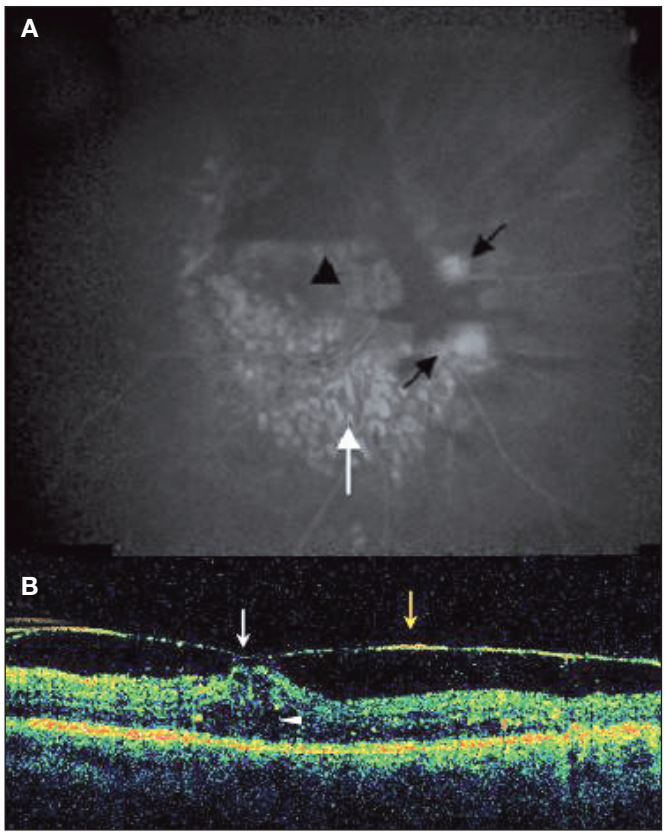

Fig. 1 - *Left eye at presentation.* **(A)** *Fluorescein angiography of the left eye using a Staurenghi 230 SLO lens and therefore image is inverted with wide field. Photograph shows new vessels elsewhere (black arrows), old panretinal photocoagulation scars (white arrow), and preretinal hemorrhages (arrowhead). Quality is poor due to diffuse vitreous hemorrhage.* **(B)** *Optical coherence tomography of the left eye showing a thickened fovea with cystoid changes (arrow head) and a hyperreflective posterior hyaloid (yellow arrow) exerting traction on the fovea (white arrow).*

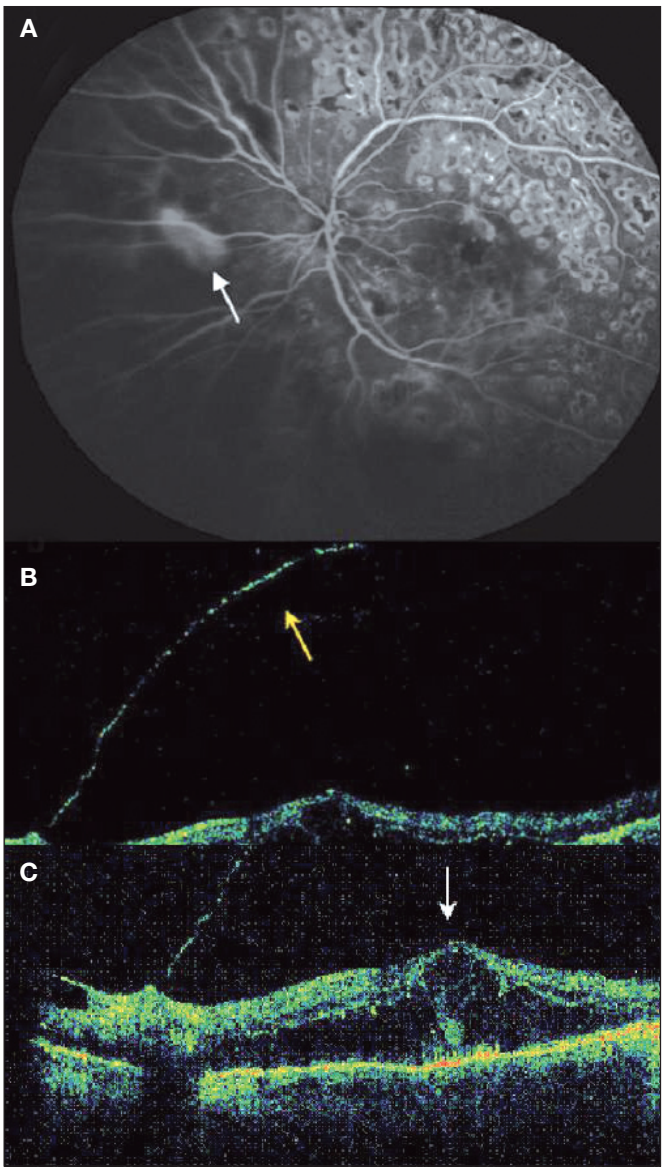

Fig. 2 - *Left eye 6 weeks after injection.* **(A)** *Fluorescein angiography synthesis showing absorption of preretinal and vitreous hemorrhages. There are residual new vessels elsewhere (white arrow).* **(B)** *Optical coherence tomography (OCT) of the left eye showing detachment of the posterior hyaloid with release of the vitreomacular traction (yellow arrow).* **(C)** *OCT of the left eye showing increased foveal thickness and macular edema (white arrow) 6 weeks after the injection.*

ported improvement in his vision in the left eye. BCVA improved to 20/60 in his LE. FA showed regression of a number of NVE with leakage in the perifoveal region (Fig. 2A). OCT scan revealed a detachment of the posterior hyaloid with release of the vitreomacular traction (Fig. 2B), but macular edema appeared deteriorated with increase in foveal thickness (Fig. 2C).

DISCUSSION

DME is a major cause of visual impairment but the implicated pathophysiologic mechanism is not yet completely understood. Massin et al (3) suggested that VTS with DME could be seen on OCT as a thick and hyperreflective posterior hyaloid that is taut over the posterior pole, but remains attached to the disk and to the top of the elevated macular surface. In our case DME seems to be associated with VTS since OCT features include a thick, hyperreflective, and taut posterior hyaloid that exerts traction

on the fovea as described by Massin et al.

The incidence of spontaneous resolution of VTS has been reported as 11% (1) and a case of spontaneous resolution of VMT 1 month following PRP therapy in a patient with diabetic retinopathy has also been documented (4). In the present case, spontaneous resolution of VTS occurred in a patient with DME 6 weeks after intravitreal injection of ranibizumab.

In VTS, eyes have a risk of developing cystoid macular changes that tend to persist (5) and therefore retinal thickness is increased. It is likely that, when VTS coexists with diabetic retinopathy, cystoid macular edema formation could be aggravated by the same factors that are responsible for DME in the absence of VTS. Nguyen et al (1) recently suggested that VEGF could be a stimulus for retinal thickening, a conclusion that is supported by the improvement in foveal thickness that is achieved with repeated injections of ranibizumab. In the same study, several patients had a large reduction in foveal thickness by 7 days after the first intraocular injection (1).

In patients with VTS and DME, the combination of the possible vitreous liquefaction and mechanical increase of vitreous volume caused by an intravitreal injection with a degree of reduction in retinal thickness caused by the effect of VEGF inhibition could play a role in the resolution of vitreomacular traction. In the present case, spontaneous resolution of VTS in a patient with DME could have been facilitated by intravitreal injection of ranibizumab. The disproportional improvement in his BCVA against the increase in foveal thickness could be explained by the absorption of vitreous hemorrhage following the documented regression of the new vessels. Also, it is likely that the reason for the increased macular edema 6 weeks after the injection is the elapsed drug activity in the inner retina–blood barrier. It is possible that repeated ranibizumab administration, as suggested by Nguyen et al, could further reduce the foveal thickness.

To our knowledge, this is the first reported case of VMT resolution in a diabetic patient following ranibizumab injection. Further studies are needed in order to determine the possibility of the association between injection of ranibizumab and spontaneous resolution of VTS.

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