European Journal of Ophthalmology / Vol. 17 no. 3, 2007 / pp. 451-453

SHORT COMMUNICATION

Macular hole and intravitreal injection of triamcinolone acetonide for macular edema due to central retinal vein occlusion

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PURPOSE. *To report a case of macular hole progression after intravitreal injection of triamcinolone acetonide (IVTA) for chronic macular edema secondary to nonischemic central retinal vein occlusion (CRVO).*
METHODS. *A 33-year-old woman with massive macular edema after CRVO underwent IVTA. Optical coherence tomography (OCT) and fluorescein angiography were performed before and after the procedure.*
RESULTS. *At the 1-week IVTA injection control, the patient's best-corrected visual acuity improved from 20/400 to 20/200 and OCT detected a progression of macular hole stage.*
CONCLUSIONS. *IVTA steroid injection may provide a significant improvement in macular edema, but injection-related complications may occur such as this uncommon macular reaction resulting in permanent visual loss. (Eur J Ophthalmol 2007; 17: 451-3)*

KEY WORDS. *Central retinal vein occlusion, Macular edema, Macular hole, Triamcinolone acetonide*

Accepted: January 19, 2007

INTRODUCTION

Intravitreous injection of triamcinolone acetonide (IVTA) is increasingly being incorporated into the management of macular edema (ME) of several diseases such as central retinal vein occlusion (CRVO), on the basis of promising results from several clinical studies (1-7).
Potential vision-threatening complications of IVTA have been recently reviewed (8). The aim of this work is to report an uncommon case of macular hole progression after IVTA.

Case report

In September 2004, a 33-year-old woman was referred to us with the diagnosis of nonischemic CRVO occurring in April 2004 in the left eye. Best-corrected visual acuity (BCVA) in the left eye was 20/400, there was no iris rubeosis, and intraocular pressure was 15 mmHg in both eyes. She underwent fundus biomicroscopy and fluorescein angiography (Fig.1, A and B), which showed the presence of macular edema with little retinal ischemia. Optical coherence tomography (OCT) showed a massive edema in the left eye and an early macular hole (9, 10) (Fig.1, C and D). In the horizontal scan (Fig. 1D), a hyper-reflective tuft was evident above the foveal depression. Biomicroscopy did not show central vitreoretinal tractions.
Four mg of triamcinolone acetonide (Kenalog) was intravitreally injected in the left eye in accordance with the guidelines for intravitreal injection (11). There were neither complications before and in peri- and postinjection management nor reflux through the injection site. At the first follow-up control, 1 week later, the patient reported an im-

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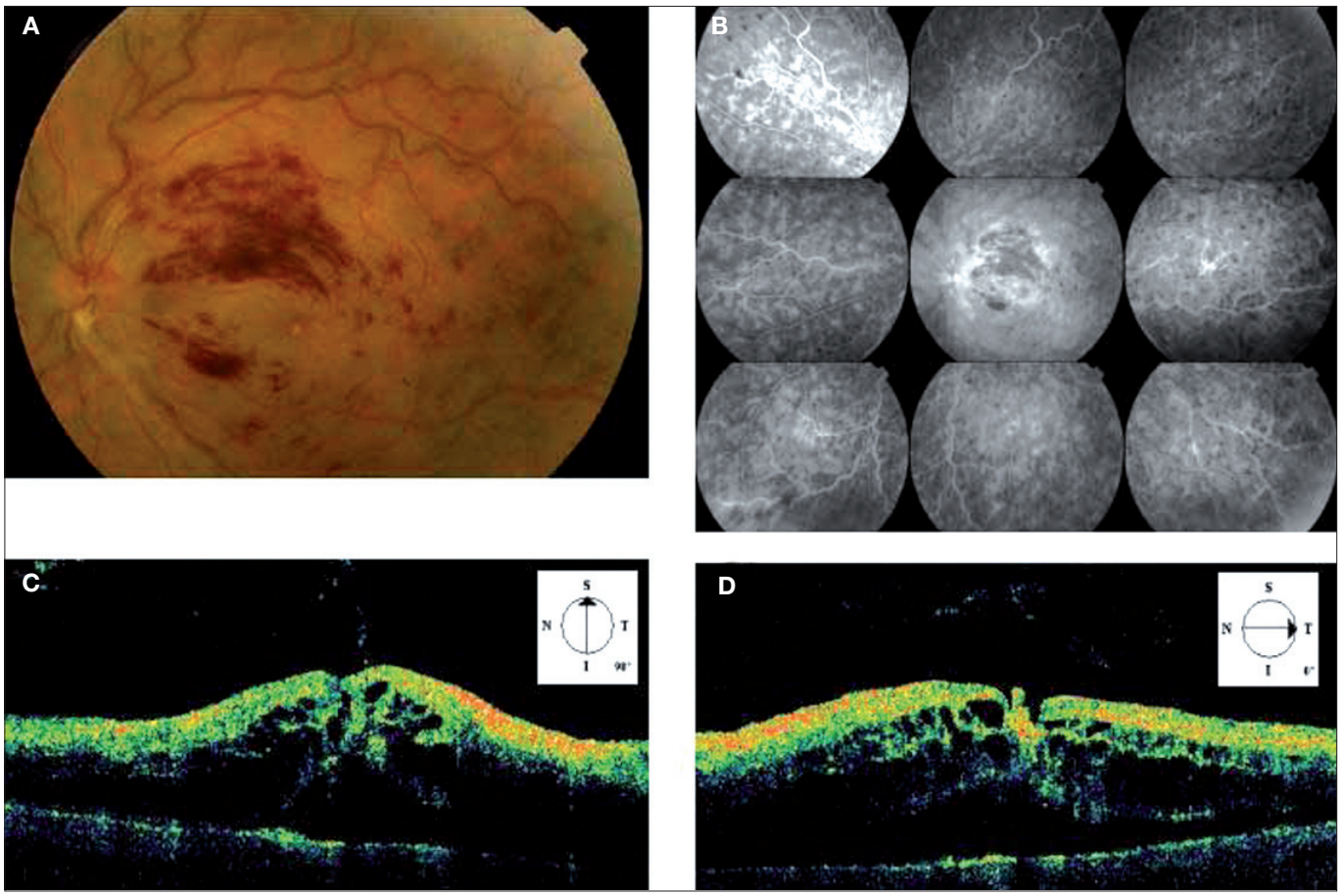

Fig. 1 - *Biomicroscopy* **(A)**, *panretinal fluorescein angiography* **(B)**, *and vertical optical coherence tomography* **(C)** *at baseline showing an edematous central retinal vein occlusion. A horizontal optical coherence tomography scan* **(D)** *evidenced a hyper-reflective tuft in the foveal depression.*

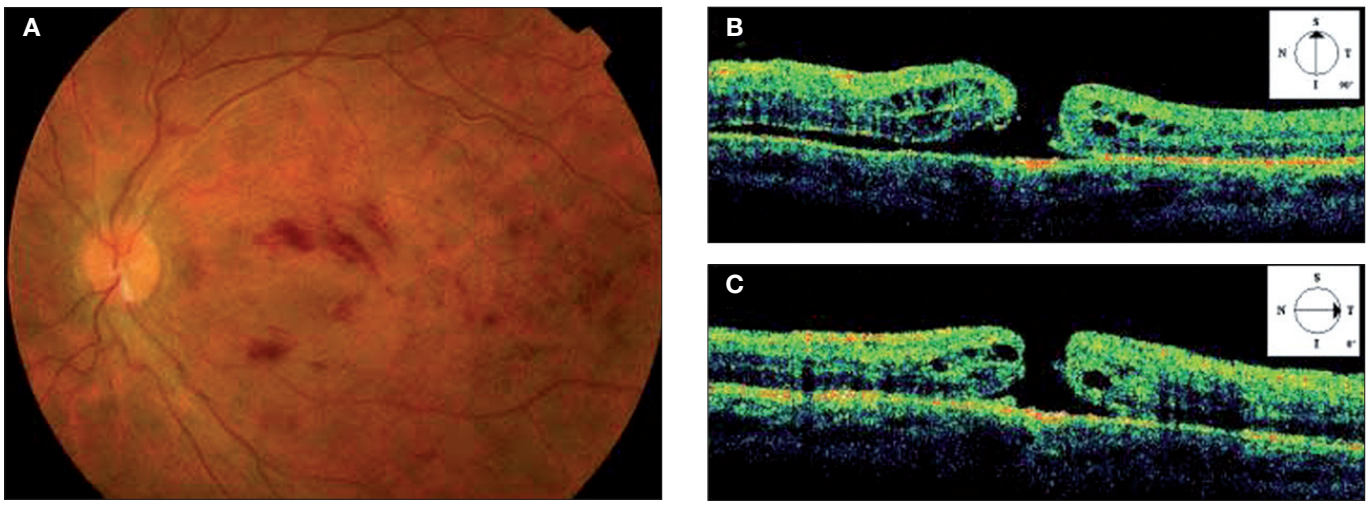

Fig. 2 - *Biomicroscopy* **(A)** *and two different optical coherence tomography scans* **(B-C)** *1 week after intravitreal injection of triamcinolone acetonide.*

provement in her vision. BCVA in the treated eye was 20/200 and intraocular pressure was 18 mmHg. The biomicroscopy of the left eye, as well as showing reduction in the number of hemorrhages, highlighted a round red foveal spot that OCT confirmed to be a stage 4 macular hole (9, 10) (Fig. 2, A–C). We saw the patient for the last time 1 month after the injection and her BCVA and intraocular pressure remained stable.

DISCUSSION

ME is a common sight-threatening nonspecific response of the retina to a variety of diseases such as CRVO. It involves the breakdown of the inner blood–retinal barrier and consists of an abnormal vascular permeability resulting in fluid accumulation and macular thickening, detectable by OCT.

Until now, no medical therapies for ME are approved; laser photocoagulation for ME due to CRVO has been shown to be effective in reducing macular thickness but it has no benefits on VA (12).

Therefore, new therapies have been suggested in recent years, including IVTA, due to steroids' potential inhibition of VEGF expression and prostaglandins release, both involved in ME pathogenesis. The large clinical experience of IVTA injection has provided a reassuring record of safety and the benefits often far exceed the well-known risks related to the injection procedure (rise in intraocular pressure, cataract, and endophthalmitis) (8).

In our case the hyper-reflective tuft detectable in foveal area on some of the baseline OCT scans could be a vitreal traction. This vitreal traction in association with the sudden retinal thickness reduction and the mechanical effects of the IVTA on the vitreous surface could be involved in the rapid progression of the macular hole.

This could also be related to a concomitant posterior vitreous detachment. Therefore we underline the role of baseline OCT pattern analysis in order to assess the eligibility of patients and prevent potential reactions of the vitreoretinal interface. OCT should be considered a routine examination to perform before the injection: multiple scans should be acquired and carefully evaluated.

The authors have no proprietary interests.

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