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Intravitreal triamcinolone acetonide: Valuation of retinal thickness changes measured by optical coherence tomography in diffuse diabetic macular edema

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PURPOSE. *The authors studied the efficacy of intravitreal triamcinolone acetonide in a case series of patients with diffuse diabetic macular edema without evidence of vitreous-macular traction refractory to laser photocoagulation.*

METHODS. *Six eyes with clinically diffuse diabetic macular edema that failed to respond to at least two previous sessions of laser photocoagulation were included. The mean age of selected patients was 72.5 ± 13.8 years, with a preoperative best-corrected visual acuity reduced to 1.48 ± 0.18 logMar and a mean baseline intraocular pressure (IOP) of 15.17 ± 2.64 mmHg. The authors also studied macular thickness measured by optical coherence tomography (OCT 2000 scanner, Humphrey Instruments, San Leandro, CA) – in the preoperative period it was 640.8 ± 171.1 µm – and the fluorangiographic (Heidelberg Retina Angiograph, Heidelberg Engineering GmbH, Heidelberg, Germany) patterns, which showed pooling in tardy phases and leakage. Mean follow-up was 4 months.*

RESULTS. *In each patient the authors observed a significant improvement, both functionally and anatomically. Mean best-corrected visual acuity increased in the postoperative period to 0.94 ± 0.53 logMar. No patient showed decline of visual acuity at the end of follow-up. Base line macular thickness was reduced in the postoperative period to 312.2 ± 157.65 µm measured by OCT and fluorangiographic patterns showed a reduction of pooling and of leakage. The most common complications described in the literature were not observed and the increase of mean IOP in the postoperative period to 18.76 ± 5.72 mmHg was not significant.*

CONCLUSIONS. *Intravitreal triamcinolone acetonide may decrease macular edema and improve visual acuity in eyes with diffuse diabetic macular edema. (Eur J Ophthalmol 2004; 14: 321-4)*

KEY WORDS. *Diabetic macular edema, Optical coherence tomography, Triamcinolone*

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INTRODUCTION

Diabetic maculopathy includes every change implicating the macula that occurs during diabetic retinopathy that has an important prognostic role in short- and medium-term visual acuity (1-3).

The prevalence of macular edema, considering all its forms (focal, diffuse, and cystic), is approximately 9% of the diabetic population. The frequency of macular edema changes with disease duration in juvenile diabetes (IDDM): it escalates from 0% in patients with a disease duration of 5 years to 29% in patients with a disease duration of 20 years or more; in patients with diabetes diagnosed after 30 years of life (NIDDM), the frequency is 3% and 28%, respectively (4, 5).

Clinically significant macular edema is the main cause of visual loss in diabetic patients, and its treatment is codified by the experience of the Early Treatment Diabetic Retinopathy Study (ETDRS), which demonstrated a significant benefit of laser photocoagulation in diffuse diabetic macular edema (6).

In the last few years the use of intravitreal triamcinolone acetonide (iTAAC) has been performed to treat several forms of macular edema including diffuse diabetic macular edema (7-9).

We have studied the efficacy of iTAAC in a case series of patients with diffuse diabetic macular edema without evidence of vitreous-macular traction refractory to laser photocoagulation (10).

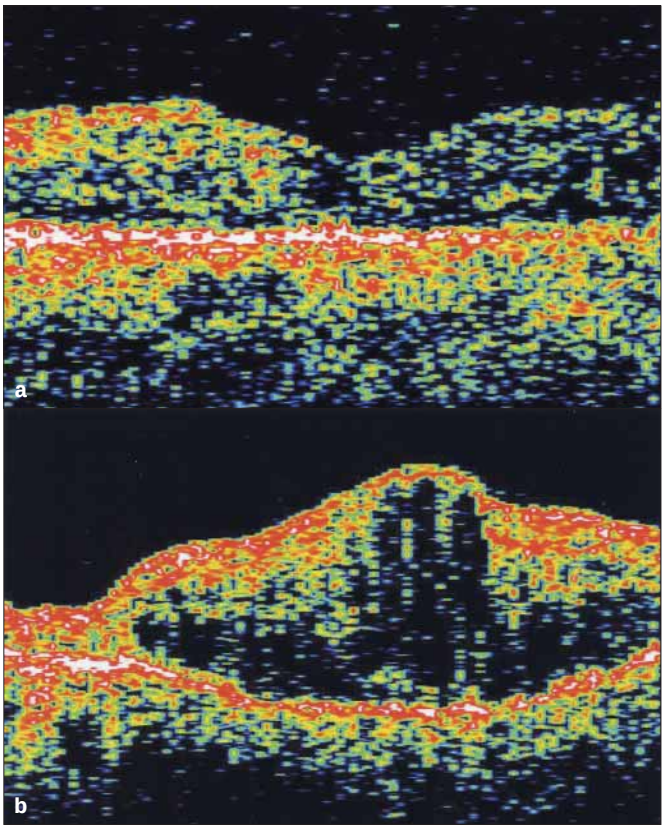

Fig. 1 - a) *Post-op;* **b)** *Pre-op.*

METHODS

From June 2002 to December 2002 we selected six diabetic patients with diffuse macular edema, all of them meeting the following criteria:

1. Posterior vitreous detachment (PVD);
2. Laser photocoagulation treatment (Nd:YAG laser) for at least 4 months;
3. Absence of other associated ocular pathology
4. Preoperative best-corrected visual acuity (BCVA) < 20/200;
5. Macular thickness > 250 µm.

The mean age of the selected patients was 72.5±13.8 years, with a preoperative BCVA reduced to 1.48 ± 0.18 logMar (preoperative BCVA 20/604), and a mean baseline intraocular pressure (IOP) of 15.17±2.64 mmHg. We also studied the macular thickness measured by optical coherence tomography (OCT; OCT 2000 scanner, Humphrey Instruments, San Leandro, CA) – in the preoperative period it was 640.8±171.1 µm (Figs. 1-3) – and the fluorangiographic (FA; Heidelberg Retina Angiograph, Heidelberg Engineering GmbH, Heidelberg, Germany) patterns, which showed pooling in tardy phases and leakage.

Intravitreal injection of triamcinolone acetonide was offered to patients after obtained informed consent. Patients were prepared by mydriatic eyewash 30 min before surgical operation. Disinfection was guaranteed by application on operating field of povidone-iodine 5% (Betadine, Alcon). Topical anesthesia was obtained by using ossibuprocaine 0.4% (Novesina, Novartis Farma s.p.a.), two drops each 5 minutes; three in total. TAAC (Kenacort 40 mg/ml, Bristol-Myers Squibb) not separated from vehicle was injected slowly 3.5 mm posterior to the limbus at a dose of 4 mg (0.1 ml).

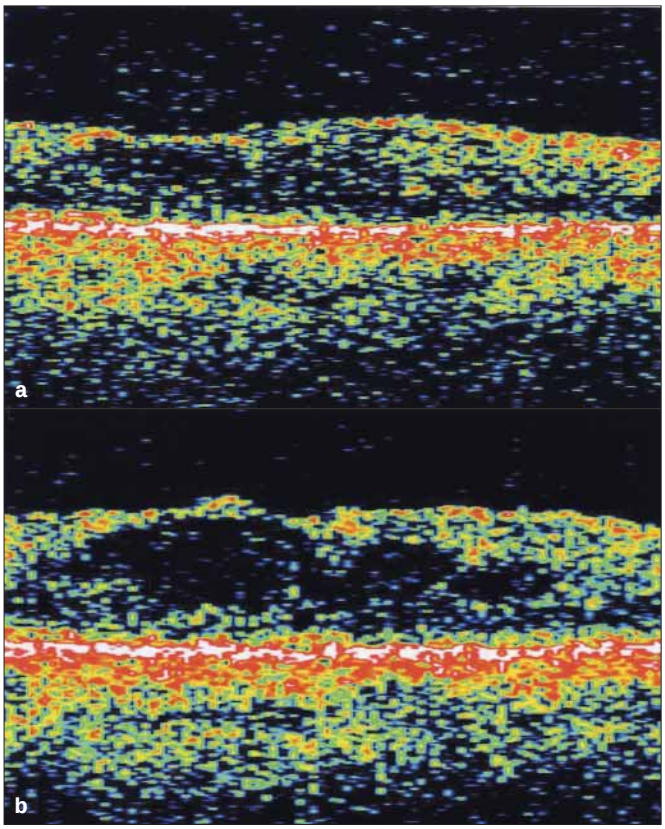

Fig. 2 - a) *Post-op;* **b)** *Pre-op.*

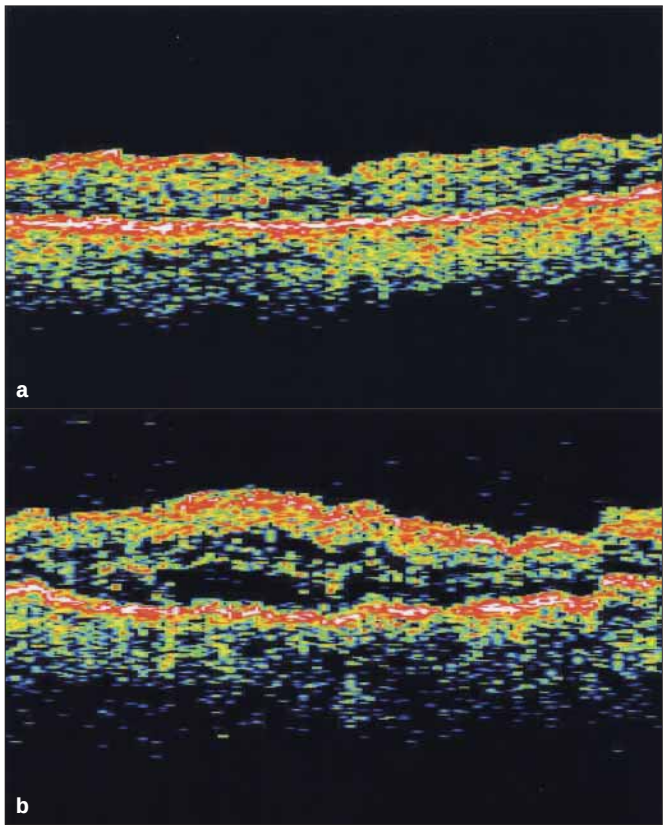

Fig. 3 - a) *Post-op;* **b)** *Pre-op.*

In each patient we performed a paracentesis of the anterior chamber.

Mean follow-up time was 4.8±1.4 months, analyzing at 1, 3, and 6 months BCVA, IOP, macular thickness measured by OCT, and fluorangiographic and ophthalmoscopic patterns.

RESULTS

In each patient we observed a significant improvement, both functionally and anatomically. BCVA increased in the postoperative period by six Snellen lines (BCVA preoperatively: 20/604, logMar 1.48 ± 0.18; BCVA postoperatively: 20/174, logMar 0.94 ± 0.53; t-test, p=0.06). Baseline macular thickness was reduced in the postoperative period by 328.7 ± 225.3 µm (preoperative: 640.8 ± 171.1 µm, postoperative: 312.2 ± 157.6 µm; t-test; p=0.018) measured by OCT (Figs. 1a, 2a, 3a). IOP increased (not significantly) by 3.5 ± 8.2 mmHg (preoperative15.17 ± 2.64 mmHg; postoperative18.67 ± 5.72 mmHg; t-test, p=0.34). Fluorangiographic patterns show a reduction of pooling and of leakage. We observed no opacity of the lens or the most common complications described in the literature (11-14).

DISCUSSION

The role of laser photocoagulation treatment in following evolution of vitreal conditions has been studied by many authors (15-17).

The macular edema pathogenesis is principally to bring back to increased vascular permeability. The accumulation of fluid determines structural changes in retinal cells.

Intravitreal TAAC favors both the reduction of blood-retinal barrier breakdown and accumulated fluid (18).

Our experience illustrates the evident efficacy of this

therapy; in fact, we observed in each patient a drastic reduction of retinal thickness at posterior pole and restoration of macular profile. We observed no adverse effects.

From a functional point of view we observed an important improvement concurrent with restoration of normal retinal and macular anatomy.

The efficacy of treatment, however, could be temporary; in fact, the metabolic conditions persist and the macular edema could recur. In any event, studying the vascular conditions in a retina that is not thickened allows the detection and treatment of possible points of focal loss.

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