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SHORT COMMUNICATION

Case report

Corneal neovascularization possibly associated with latanoprost therapy

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PURPOSE. *To report a case of corneal neovascularization possibly associated with latanoprost therapy.*

METHODS. *Case report: A 67-year-old man developed a progressive stromal corneal neovascularization in his right eye within eight months of a corneal trauma. At admission, he was receiving latanoprost 0.005% therapy. His topical medications were rearranged: latanoprost was replaced with carteolol hydrochloride 1% twice daily bilaterally and prednisolone acetate 1% was added twice daily in the right eye.*

RESULTS. *One month later, he presented regression of the corneal neovascularization and an increase in visual acuity.*

CONCLUSIONS. *Latanoprost, an arachidonic acid derivative, could have directly or indirectly stimulated the corneal neovascularization in this patient with a history of nonpenetrating corneal trauma. (Eur J Ophthalmol 2003; 13: 88-90)*

KEY WORDS. *Corneal neovascularization, Latanoprost therapy, Corneal trauma*

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INTRODUCTION

The cornea is normally avascular, but under certain conditions capillaries invade from the limbal vascular plexus. Inciting insults include corneal trauma, mild chemical burns, inflammation, and infection (1, 2). Different patterns of neovascularization are associated with a wide variety of insults, but they are generally grouped under three headings: superficial vascularization, vascular pannus, and deep stromal vascularization. Here we report a patient with corneal neovascularization who had a nonpenetrating ocular trauma on his right eye four months before he was started on latanoprost 0.005% therapy for pseudoexfoliative glaucoma.

Case report

A 67-year-old man was referred with corneal neovascularization extending from the nasal limbus to the paracentral area in his right eye. He had suffered partial thickness corneal laceration with a piece of reed on the nasal side of his right eye a year earlier. He had been given topical antibiotic drops for two weeks by a private practitioner and the lesion had healed, leaving a corneal scar. Four months later, he was started on topical latanoprost 0.005% for the diagnosis of pseudoexfoliative glaucoma, and two months later, a hyperemic limbal lesion appeared in the area of the trauma (Fig. 1). Although he was given topical anti-inflammatory drops the lesion kept growing, reducing his visual acuity.

When the patient was referred to our department, his best-corrected visual acuity was 20/160 in his right eye and 20/20 in his left eye. Biomicroscopic examination revealed corneal stromal neovascularization originating from the nasal limbal area and reaching the midpupillary field in his right eye. There was pseudoexfoliation material at the pupillary borders of both eyes. His fundus examination showed a cup:disc ratio of 0.2 bilaterally. Gonioscopic examination detected no neovascularization at the iridocorneal angle. In-

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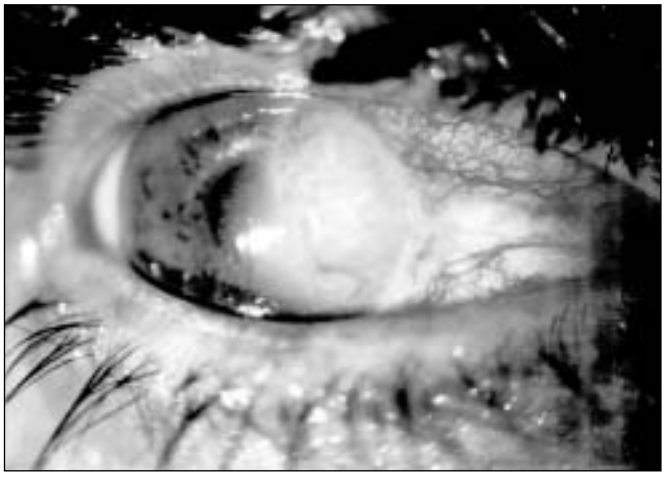

Fig. 1 - *Corneal neovascularization and nasal conjunctival hyperemia during latanoprost therapy.*

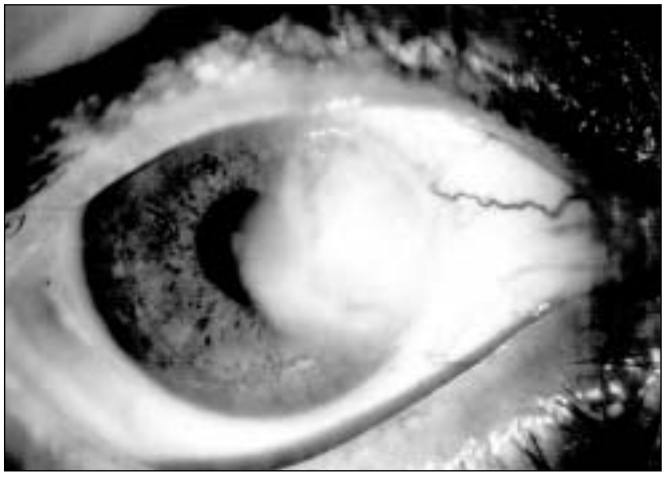

Fig. 2 - *Regression of the corneal neovascularization after starting topical prednisolone and withdrawal of latanoprost.*

traocular pressure (IOP) was 20 mmHg bilaterally. He was using latanoprost 0.005% once daily bilaterally. The patient was started on topical prednisolone acetate 0.1% twice daily in the right eye and latanoprost was replaced with carteolol hydrochloride 0.1% twice daily bilaterally.

One month later, his visual acuity reached 20/63 in the right eye. Biomicroscopic examination showed regression of the neovascularization (Fig. 2). IOP was 19 mmHg in the right eye. Once the regression was observed, prednisolone acetate was stopped within two weeks. As a coincidental finding, the patient described diplopia on left gaze. Ocular movement of his left eye was restricted temporally. Magnetic resonance imaging showed a soft tissue lesion filling the nasopharyngeal cavity on the left side. At the next visit one month later, before radiotherapy for the nasopharyngeal tumor, there was no progression in the corneal lesion.

DISCUSSION

The etiology of corneal neovascularization is not completely understood, and much of the present data are derived from animal models. Numerous factors have been proposed to explain it, including vasoproliferative factors, vasoactive amines, plasminogen activators, corneal anoxia, prostaglandins, and leukocytes. Both the leukocyte infiltration and corneal neovascularization occurring in response to experimental thermal corneal insults may result from local prostaglandin synthesis (3). These arachidonic acid derivatives produce certain manifestations of ocular inflammation, besides increasing vascular permeability and causing vasodilatation. Prostaglandins, particularly of the prostaglandin E series, are associated with neovascularization in several ocular sites, including the cornea (4). In an experimental rabbit model agents that inhibited prostaglandin synthesis suppressed corneal stromal polymorphonuclear leukocyte infiltration and neovascularization (5).

Although the exact reason is not known, arachidonic acid derivatives seem to play an important role in corneal neovascularization. Latanoprost, which is one of these derivatives, could have directly or indirectly stimulated the neovascularization in our patient with a history of nonpenetrating corneal trauma. In this case, it is not clear whether the regression was the result of starting topical prednisolone, switching from latanoprost to carteolol, or both. We believe that both strategies probably contributed. We are not aware of any previous reports of an association between latanoprost therapy and corneal neovascularization.

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