A Rare Complication: Hypotony and Cystoid Macular Edema due to Travoprost

Nadir Bir Komplikasyon: Travoprosta Bağlı Hipotoni ve Kistoid Maküler Ödem

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ABSTRACT

A 73 year-old man attended for routine follow-up. He had a history of primary open angle glaucoma in the left eye which was diagnosed 2 years ago. He had been using benzalkonium chloride - free travoprost 0.004% eye drop once a day for 2 years. His best corrected visual acuity was 20/60 and intraocular pressure 5 mmHg in the left eye. Dilated fundus examination demonstrated cystoid macular edema as confirmed by optical coherence tomography. In accordance to that the therapy was discontinued until the intraocular pressure value increased and visual acuity recovered after some time. Consequently hypotony and decrease in visual acuity re-occurred due to second application of travoprost and these findings were resolved after the therapy was stopped again.

Key Words: Travoprost, hypotony, cystoid macular edema.

ÖZ

Yetmiş üç yaşında erkek hasta rutin kontrol için başvurdu. Hastaya iki yıl önce primer açık açılı glokom tanısı konulmuştu. Hasta iki yıldır günde 1 damla benzalkonyum klorid içermeyen travoprost %0.004 kullanmaktaydı. Hastanın sol gözünde düzeltilmiş görme keskinliği 20/60 düzeyindeydi ve göz içi basıncı 5 mmHg idi. Dilate fundus muayenesinde ve optik koherens tomografide kistoid maküler ödem saptandı. Bunun üzerine göz içi basıncı yükselip bir süre sonra görme keskinliği düzelinceye dek tedavi kesildi. Travoprostun ikinci kez uygulanmasıyla hipotoni ve görme keskinliğinde azalma tekrar meydana geldi ve tekrar tedavi kesilince bulgular düzeldi.

Anahtar Kelimeler: Travoprost, hipotoni, kistoid maküler ödem.

INTRODUCTION

Prostaglandin analogues increase uveoscleral outflow of the aqueous, possibly by relaxation of the ciliary muscle associated with increased metalloproteinase activity. As the uveoscleral pathway is pressure-independent, it has been suggested that prostaglandin analogues could lower intraocular pressure (IOP) below episcleral venous pressure, and thereby may cause hypotony and choroidal detachment.[1] Macular edema can occur as a rare side effect in eyes treated with travoprost or other prostaglandin analogues. Pseudophakic eyes and eyes with other risk factors for macular edema are most likely to be affected, and phakic eyes without risk factors may not be at risk. The edema resolves, and visual acuity returns, upon cessation of prostaglandin therapy.[2] In this paper, we describe a patient who developed recurrent hypotony together with cystoid macular edema (CME) after re-treatment with travoprost, with no history of glaucoma surgery.

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CASE REPORT

A 73 year-old man attended for routine follow-up. Ten years ago, he underwent uncomplicated phacoemulsification surgery with a posterior chamber intraocular lens implant in the left eye. He had a history of primary open angle glaucoma (POAG) in the left eye which was diagnosed 2 years ago.

He had been using benzalkonium chloride (BAK) -free travoprost 0.004% eye drop once a day for 2 years. His best corrected visual acuity was p- in the right eye due to trauma 20 years ago and 20/60 in the left eye. Intraocular pressures were 19 mmHg in the right eye and 5 mmHg in the left eye. The central corneal thickness was 570 µm in right eye and 562 µm in the left eye.

Slit-lamp examination revealed intraocular lens in both eyes, the anterior chamber was deep and quiet. Dilated fundus examination demonstrated a cup-to-disc ratio of 0.5 in the left eye (Figure 1) and CME as confirmed by optical coherence tomography (OCT) (Figure 2a). The right fundus could not be observed. The anti-glaucoma treatment was stopped.

Three weeks later, visual acuity recovered to 20/40, the IOP was 13 mmHg and the CME was resolved in the OCT (Figure 2b). Five months later, he was examined again, when he was found to have a visual acuity of 20/40, and IOP of 24 mmHg in the left eye. Travoprost was re-started once a day. After 4 months, he returned with visual disturbance in the left eye (20/80) and left IOP of 4 mmHg. Dilated fundus examination revealed CME as confirmed by OCT.

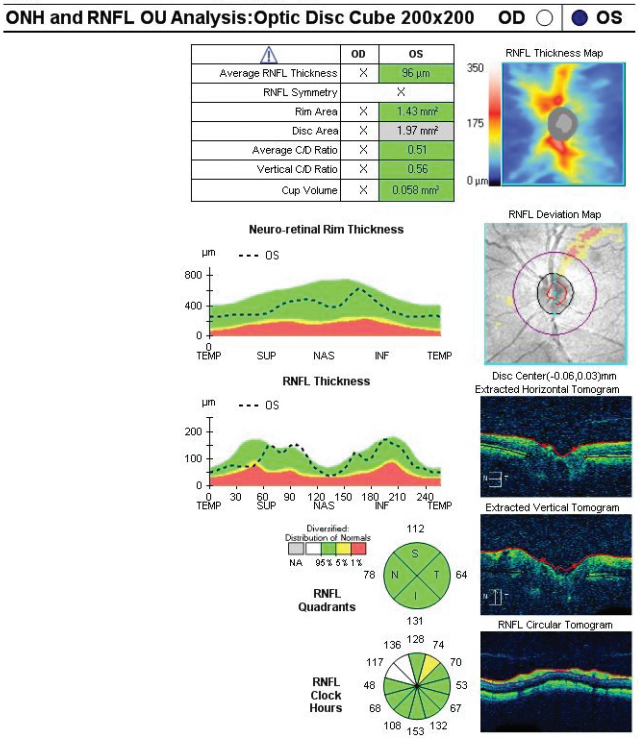
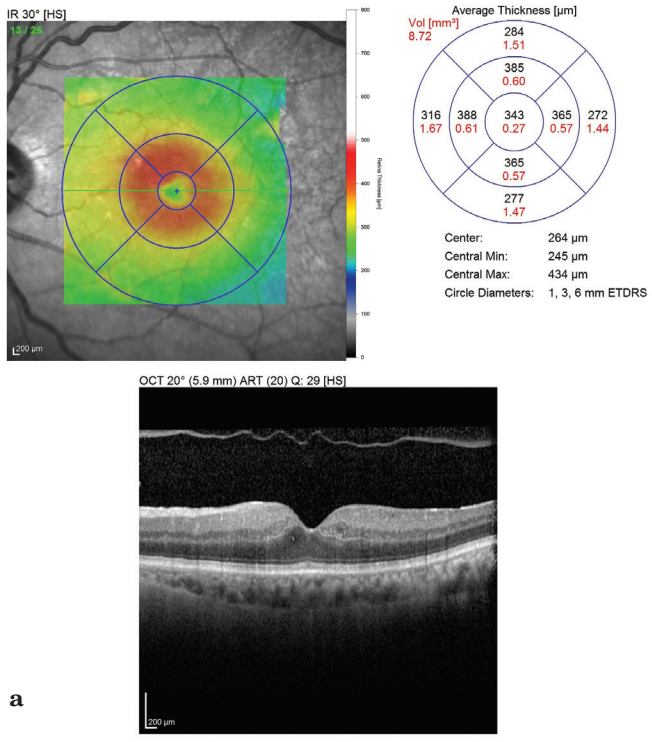


Figure 1: *Optical coherence tomography demonstrating a cup-to-disc ratio of 0.5 in the left eye.*

Topical travoprost treatment was discontinued and topical diclofenac was started 4 times a day. One month later, visual acuity recovered to 20/40 again and the CME was resolved in the OCT (Figure 3b). At 3 months follow-up his lOP in the left eye remained controlled at 16 mmHg without treatment.



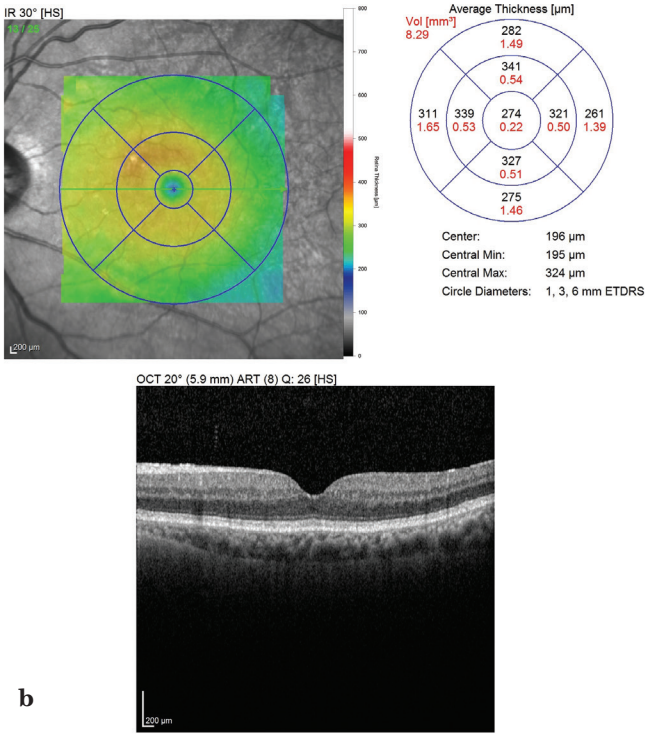


Figure 2a, b: *Optical coherence tomography demonstrating macular edema in the left eye (a). Three weeks later after discontinuation of the travoprost treatment, the macular edema was resolved (b).*

DISCUSSION

Hypotony and choroidal detachment after the use of topical latanoprost have been reported previously.[4,5] Alexander&Ramirez-Florez reported a patient with hypotony and choroidal detachment as a complication of travoprost after trabeculectomy surgery.[6] There is another report describing choroidal detachment in eyes with chronic angle closure on travoprost therapy.[7] To our knowledge, hypotony has not been previously reported in a patient with POAG on topical travoprost therapy but no history of glaucoma surgery.

There are a lot of reports about CME development as a complication of travoprost therapy (both BAK free form and the oldest form) in pseudophakic patients.[3,8,9] The risk factors for the development of CME in pseudophakic eyes, due to the use of topical prostaglandin analogues are, a surgical history of glaucoma operations, complicated cataract surgery, aphakia, scleral buckling or vitrectomy, as well as a history of uveitis or other retinal diseases. In cases without known risk factors, CME can appear within 2-3 months of cataract surgery, whereas in high risk cases, CME can even appear several years after the surgery.[3,10] In cases involving known risk factors, the damage to the blood-aqueous barrier is protracted resulting in the continuance of conditions that lead to CME in the long term.[11] In our patient, CME occurred together with hypotony which might be another unknown risk factor for the development of CME in patients receiving prostaglandin analogues. This CME might be considered as a maculopathy secondary to hypotony, but in this case there were no the other hypotony maculapathy findings as choroidal folds.

In our patient, recurrent hypotony together with CME after re-treatment with travoprost had developed. At the first examination, the IOP value was very low due to travoprost usage and visual acuity was decreased due to CME. In accordance to that the therapy was discontinued until the IOP value increased and visual acuity recovered after some time. Consequently hypotony and decrease in visual acuity re-occurred due to second application of travoprost and these findings were resolved after the therapy was stopped again. This case is different and interesting because hypotony developed in a patient with POAG on topical travoprost therapy but no history of glaucoma surgery. The recurrence of hypotony and decrease in visual acuity upon re-treatment might be considered as a proof, that hypotony and CME are directly related with the usage of travoprost. Another important aspect is that CME development was also observed together with hypotony. In conclusion, the patients who receive travoprost or the other prostaglandin analogues should be observed carefully for such findings and informed about the situation.

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