Central Serous Chorioretinopathy Associated with Latanoprost Use

Latanoprost Kullanımına Bağlı Santral Seröz Koriyoretinopati

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ABSTRACT

A 55 years old man presented to our clinic with a 3 month history of blurred vision and micropsia in his left eye. He was previously diagnosed with unilateral glaucoma and treatment was initiated with topical latanoprost 0.005% and brimonidine tartrate/timolol maleate 0.2%/0.5% combination for the left eye in another clinic. The symptoms occurred one month after the initiation of glaucoma treatment. Fundus examination, optical coherence tomography and fluorescein angiography revealed a juxtafoveal neurosensory detachment which was coherent with central serous chorioretinopathy (CSC). After the cessation of latanoprost treatment, his clinical status improved and almost complete resolution of neurosensory detachment was observed spontaneously. The use of topical latanoprost may lead to CSC. Clinicians should consider this complication especially in patients presented with decreased visual acuity while using latonoprost

Key Words: Central serous chorioretinopathy, latanoprost, glaucoma.

ÖZ

Elli beş yaşında erkek hasta kliniğimize üç aydır devam eden sol gözde bulanık görme ve mikropsi şikayetleriyle başvurdu. Hastaya daha önce başka bir klinikte tek taraflı glokom tanısıyla sol göze topikal %0.005 latanoprost ve brimonidin tartarat/timolol maleat %0.2/%0.5 kombinasyonu başlanmış. Hastanın şikayetleri glokom tedavisinden bir ay sonra başlamış. Fundus muayenesi, optik koherens tomografi ve floresein anjiografide santral seröz korioretinopati (SSK) ile uyumlu olan jusktafoveal nörosensoryal dekolman izlendi. Latanoprost tedavisinin kesilmesini takiben spontan olarak hastanın kliniğinin düzeldiği ve neredeyse tama yakın nörosensoryal dekolmanda çözülme izlendi. Topikal latanoprost kullanımı SSK'ye neden olabilir. Klinisyenler, latanoprost kullanırken görme azalması şikayeti ile başvuran hastalarda bu komplikasyonu göz önünde bulundurmalıdır.

Anahtar Kelimeler: Santral seröz korioretinopati, latanoprost, glokom.

INTRODUCTION

Central serous chorioretinopathy (CSC) is a sporadic disease occurring in young and middle-aged adults and is characterized with neurosensory and retinal pigment epithelial (RPE) detachment.[1] Although CSC has been described as a benign and self-limiting disease, approximately 5% of patients may develop multifocal and recurrent form which may be associated with permanent visual loss. As for our knowledge, associated risk factors are male gender, high blood pressure, type A achievement-oriented personality and steroid treatment.[2] Latanoprost (prostaglandin F2α analogue) is an effective antiglaucoma drug which provides a reduction of 25-35% in intraocular pressure (IOP).[3] The most common reported ocular side effects of latanoprost are burning, conjunctival hyperemia, hypertrichosis, iris discoloration, iritis and cystoid macular edema.[4] The present report describes a very rare complication of latanoprost use: a case with central serous chorioretinopathy.

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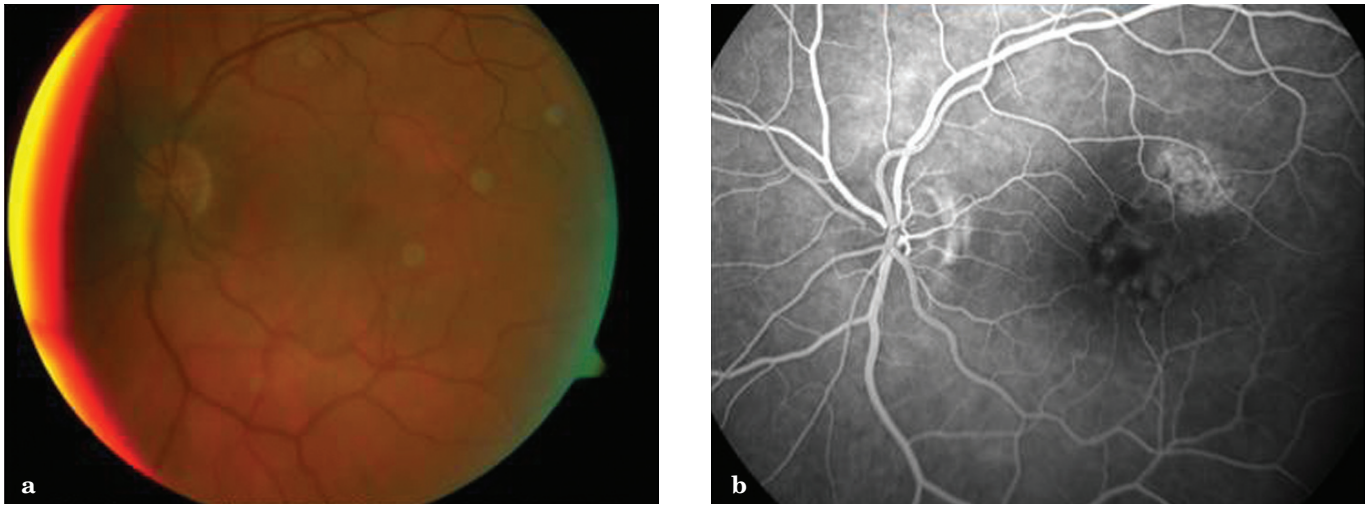

Figure 1a,b: *Fundus photograph (a) and fluorescein angiography of the left eye at the presentation (b).*

CASE REPORT

A fifty five years old man recently diagnosed with unilateral glaucoma presented to our clinic with the complain of blurred vision and micropsia in his left eye for 3 months. Latanoprost and brimonidine tartrate/timolol maleate 0.2%/0.5% combination treatment had been initiated to the left eye in another clinic 4 months ago. The best-corrected visual acuity (BCVA) was 20/20 in the both eyes. IOP was 13 mmHg in the left eye with antiglaucomatous medication and 14 mmHg in the right eye without any medication. Both eyes were phakic and slitlamp examination was unremarkable. Fundoscopic examination showed a macular elevation with RPE hypertrophy in the left eye and fluorescein angiograph (FA) revealed a few focal leakages at the superiotemporal macular region and serous detachment with RPE hypertrophy blockage at the foveal region (Figure 1).

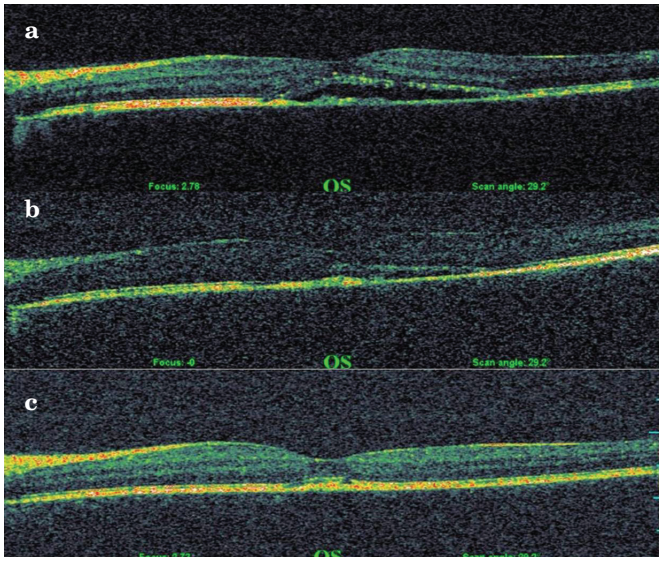

Figure 2a,c: *Optical coherence tomography (OCT) images at (a) presentation, (b) first month of latanoprost cessation, (c) last visit.*

Optical coherence tomography (OCT) was performed and serous detachment was observed in the left eye (Figure 2a). The fundoscopic examination, FA and OCT findings of the right eye were unremarkable. Possible serous detachment causes; inflammatory and infectious etiologies, hypertension, previous steroid treatment and major depression were investigated and excluded. We were puzzled to see CSC in a 55 years old man who had no risk factor. Finally, the diagnosis of latanoprost associated CSC was made on the basis of clinical appearance of the macula, OCT and FA findings. Latanoprost treatment was discontinued and a follow-up with dilated fundus examinations and OCT was suggested once a month. At the first month of follow-up, BCVA was observed to be 20/20 with a progress in his subjective complaints. We observed a progressive decrease in subretinal fluid by OCT (Figure 2b).

At the third visit, two months after latanoprost discontinuation, the patient's symptoms and objective clinical findings resolved and OCT revealed an almost complete resolution in the serous detachment. At the last visit, at the sixth month of latanoprost cessation, BCVA was observed to be 20/20 with no subjective complaints. OCT revealed an irregularity at the photoreceptor segments with a complete resolution of serous detachment (Figure 2c).

DISCUSSION

We present a case of a 55 years old man who was diagnosed with latanoprost associated CSC on the basis of fundus examination, OCT and FA findings.

CSC typically affects males between the ages 20 and 45, however, there are case reports of CSC occurring in patients of 60 years or older.[5] Although several hypotheses have been proposed, the pathogenesis of CSC still remains unclear.

These include abnormal choroidal vascular hyperpermeability, physical and functional defects in Bruch's membrane and RPE. Latanoprost is a prostaglandin F2α analogue which reduces IOP by enhancing uveoscleral outflow. Prostaglandins are the most known chemical transmitters contributing to inflammation in systemic diseases, including ocular inflammatuary diseases. It has been reported that prostaglandin F2α analogues may cause uveitis and iritis and these adverse effects are most common with latanoprost.[6] It has also been suggested that prostaglandins may affect vascular permeability.[7] We assume therefore that latanoprost treatment in this case may have contributed to the inflammation and caused CSC. Artunay et al., was the first to describe latanoprost associated CSC in a 65 years old woman. They hypothesized that topical latanoprost may increase choroidal vascular permeability and cause accumulation of serous fluid to the subretinal space. Özkan et al.,[8] also reported a case of serous retinal detachment associated with latanoprost.

Besada and associates suggested that the use of topical antiglaucomatous medications may have aggravated a pre-existing vitreo-retinal traction and consequently contributed to the development of CSC.[9] Intra-cameral aqueous pressure reduces in comparison with chorio-retinal fluid pressure which promoted a forward displacement of vitreous and induced vitreo-retinal traction that resulted in the development of CSC. This scenario is also possible for this case as a partial or complete posterior vitreous detachment was not observed in the OCT. We could assume that the vitreoretinal interface adhesion was intact for this case.

We believe that CSC may result from the interplay of many factors and prostaglandins seem to be one of these acting factors. This case demonstrates that topical latanoprost is likely to be related with the development of CSC. Ophthalmologists should consider and be cautious about the development of CSC as a possible adverse effect of topical latanoprost therapy.

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