Indapamide-Induced Transient Myopia, Choroidal Thickening and Angle Closure: A Case Report

İndapamid Kullanımına Bağlı Geçici Miyopi, Koroid Kalınlaşması ve Açı Kapanması: Bir Olgu Sunumu

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ÖZ

Sülfonamid türevi ilaçlar siliyer cisim ve uveada yapısal değişikliklere neden olabilmektedir. İndapamid (sülfonamid türevi bir diüretik, antihipertansif ajan) alımıyla ilişkili siliyer cisim ödemi ve siliokoroidal efüzyon, daha önce ultrasonik biyomikroskopi (UBM) ve klasik ultrasonografi ile gösterilmiştir. Bu olgu sunumunda; 52 yaşında erkek hasta akut bulanık görme ve künt göz ağrısı şikâyetiyle kliniğimize başvurdu. Hastanın ayrıntılı anemnezinde esansiyel hipertansiyon nedeniyle 2 haftadır günde bir kez 1,5 mg tablet İndapamid kullanıyor olduğu öğrenildi. İlk oküler değerlendirme neticesinde hastada her iki gözde belirgin miyopik refraktif kayma ve göz içi basıncında (GİB) artış saptandı. Ön segment ve EDI optik koherens tomografi (OKT) görüntülemeleri sonucunda belirgin sığ ön kamara, irido-korneal açı kapanması ve koroidal kalınlaşma tespit edildi. Bu olgu literatürdeki, indapamid kullanımına bağlı bilateral irido-korneal açı kapanması ve göz içi basıncı artışı ile seyreden üçüncü olgu sunumu olmakla beraber, koroidal kalınlaşmanın EDI-OKT yardımıyla gösterildiği ilk olgudur.

Anahtar kelimeler: indapamid, optik koherens tomografi, koroid, miyopi, ön kamara.

ABSTRACT

Sulphonamide-derived drugs may cause morphological changes in the ciliary body and uveal tract. Ciliary body swelling and cilio-choroidal effusion associated with indapamide (a sulfa-derivative diuretic, antihypertensive agent) intake have been demonstrated through ultrasound biomicroscopy (UBM) and classic ultrasonography previously. A 52-year-old male patient suffering from acute blurred vision and a dull eye pain referred to the ophthalmology clinic. The indapamide (1.5 mg tablet once a day) was prescribed to the patient two weeks before for essential hypertension. Bilateral significant myopic shift and intraocular pressure (IOP) elevation were determined at the initial examination. Anterior segment and enhanced depth imaging optical coherence tomography (EDI-OCT) scans revealed shallow anterior chamber, angle closure and choroidal thickening. This case is the third presentation with bilateral angle closure and increase in intraocular pressure (IOP) due to indapamide use and first one demonstrating choroidal thickening via EDI-OCT as a novel follow-up instrument.

Key words: indapamide, optical coherence tomography, choroid, myopia, anterior chamber

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INTRODUCTION

Indapamide is a sulfa-derivative diuretic agent that is mainly used for the treatment of essential hypertension and edema. Sulfonamides, tetracycline, hydrochlorothiazide and corticosteroids are the known drugs that are associated with increased IOP, acute myopia, angle-closure, ciliochoroidal effusion and edema.[1] To best of our knowledge, there were four case reports in the literature presenting indapamide associated ocular side effects and only two of them with angle closure and IOP elevation.[2-5] In this reports indapamide related ciliary body swelling, edema and choroidal thickening were demonstrated through UBM and ocular ultrasonography. EDI-OCT is now an indispensable clinical routine that enables in-vivo detailed evaluation of the choroidal structural changes.[6]

The current report is the third presentation of a case demonstrating angle closure, IOP elevation, myopic shift associated with indapamide intake and the first one showing choroidal thickening via EDI-OCT.

CASE REPORT

A 52-year-old male patient presented with a complaint of acute bilateral blurred vision and a dull ache around the eyecups starting three days ago. According to the medical history of the patient, once a day indapamide 1.5 mg tablet was prescribed two weeks before for essential hypertension. The patient had no additional systemic and ocular disease and no drug use. On the initial ocular examination refractive errors were -2.00D, -0.75*90D (OD), -2.25D, -1*90D (OS). Uncorrected and corrected Snellen visual acuities (VA) were bilateral 3/10 and 10/10 respectively. The anterior segment evaluation and gonioscopy revealed a shallow anterior chamber, angle closure (Shaffer Grade 0, 1, figure 1a) and clear aqueous with no cells. IOP values (Goldmann applanation tonometry) were 33 mmHg (OD), 34 mmHg (OS). The fundoscopy was bilaterally unremarkable with no peripheral choroidal detachment and cup to disc ratios were 0.3 in both eyes. Axial length measures were bilateral 22.60 mm. Anterior chamber depths (ACD) were 1.51 mm (OD), 1.62 mm (OS) and lens thicknesses were 3,60 mm (OD), 3.61 mm (OS) (Cirrus HD 500, Carl Zeiss Meditec, Dublin, CA, USA). The initial B-mode ocular ultrasonography revealed no suprachoroidal effusion (figure 3). After confirmation, the absence of other causes (angle closure glaucoma and pupillary block) indapamide treatment was terminated immediately. In the follow-up visits, patient received dorzolamide timolol fix combination twice a day. At the end of the 10th-day the final ocular examination was as follows: refractive errors: -0.25D, -0.75*90D (OD), -0.75*90D (OR), bilateral uncorrected VAs: 10/10, IOP: 12 mmHg (OD), 11 mmHg (OS), gonioscopy: bilateral Shaffer Grade 3 open angle (figure 1b), ACD: 3.52 (OD), 3.51 (OS), lens thickness: 3.56 mm(OD), 3.56 (OS). After angle resolution dorzolamide timolol combination treatment was stopped,

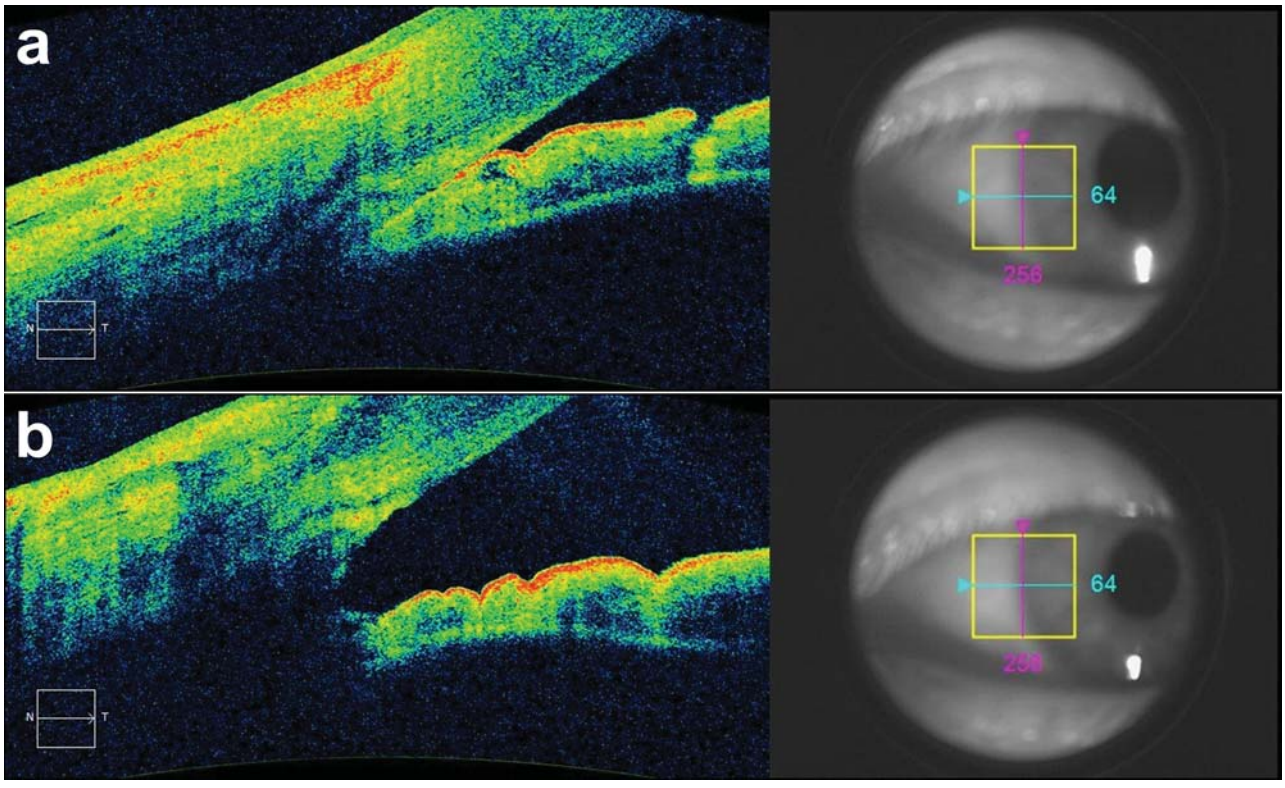

Figure 1. *Left eye nasal iridocorneal angle anterior segment OCT images of the patient. a: initial OCT scan, b: last OCT scan. After the cessation of the indapamide, the iridocorneal angle closure resolved gradually.*

no intraocular pressure increase was observed in the follow up. The choroidal thicknesses (CTs) were measured using EDI-OCT at the initial and 10th-day examination, (figure 2). It was noteworthy that initial CT measures (subfoveal: 401 µm, 1,5 mm temporal: 360 µm, 1,5 mm nasal: 360 µm) were thicker at all locations when comparing the last scan (subfoveal: 377 µm, 1,5 mm temporal: 344 µm, 1,5 mm nasal: 344 µm, figure 2). There were no remarkable suprachoroidal effusion and choroidal detachment in initial B-mode ocular ultrasonography (Figure 3).

DISCUSSION

The exact mechanisms of the ocular side effects due to sulfa-derivative drugs have not been fully clarified. In this regard, There are several proposed pathophysiological mechanisms. The change in osmotic content of the lens, forward displacement of the ciliary body and iris-lens complex have been suggested as the responsible factors for myopia during the use of this drugs.[7-9] These pathologic changes resolve after the cessation of the drugs.[7-9]Moreover, peripheral cil-

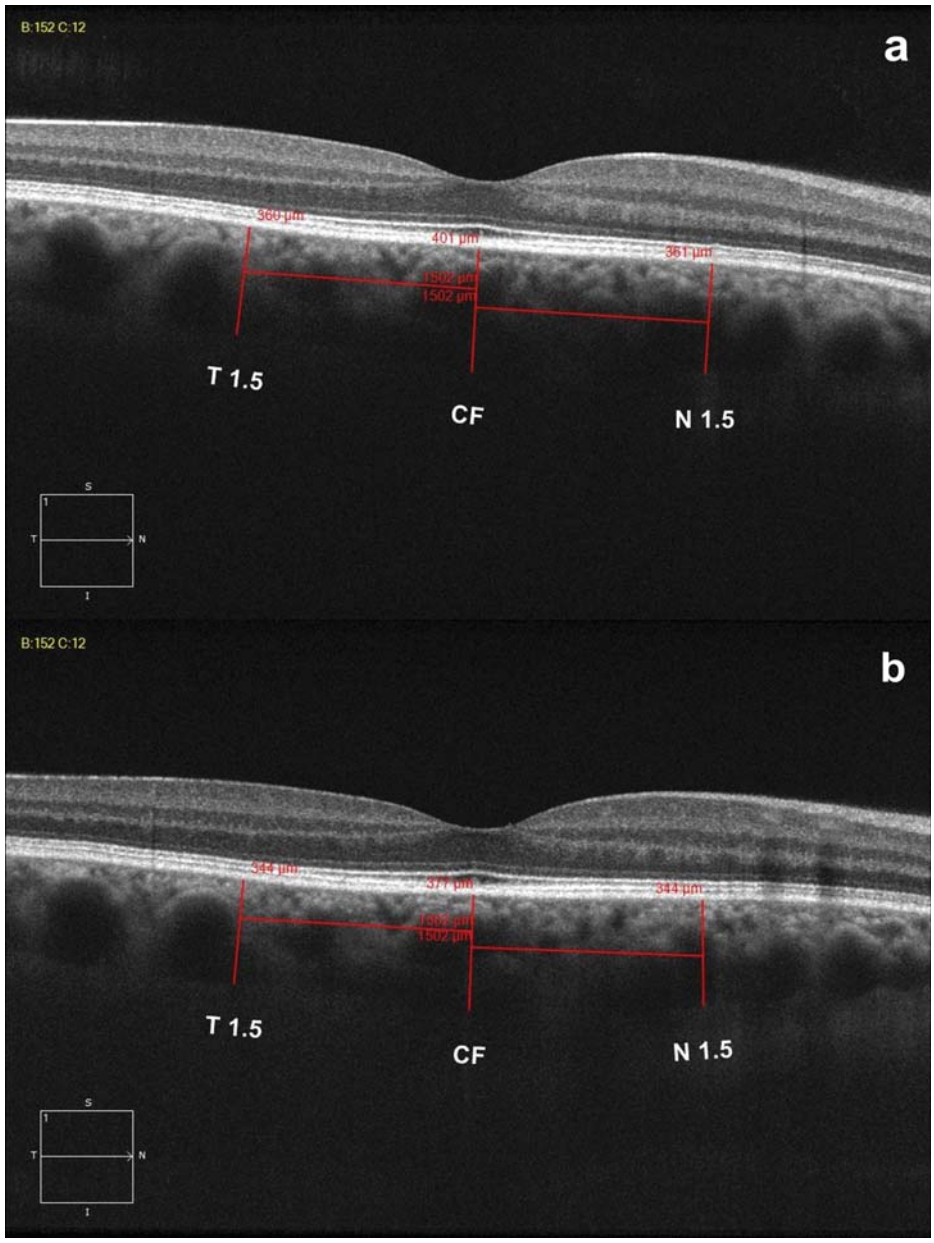

Figure 2: *a: Initial EDI-OCT scan, b: last EDI-OCT scan. CF: central fovea, N1.5: 1.5 mm nasal to the fovea, T1.5: 1.5 mm temporal to the fovea. The choroidal thickness measurements were performed manually considering hyperreflective outer border of the retinal pigment epithelium and inner scleral border. Also, vascular landmarks were considered to ensure consistency between two measurements. The loss of the reflectivity of the vascular wall borders and the outer choroidal border are noteworthy in the initial EDI-OCT scan, presumably reflecting choroidal effusion due to vascular leakage.*

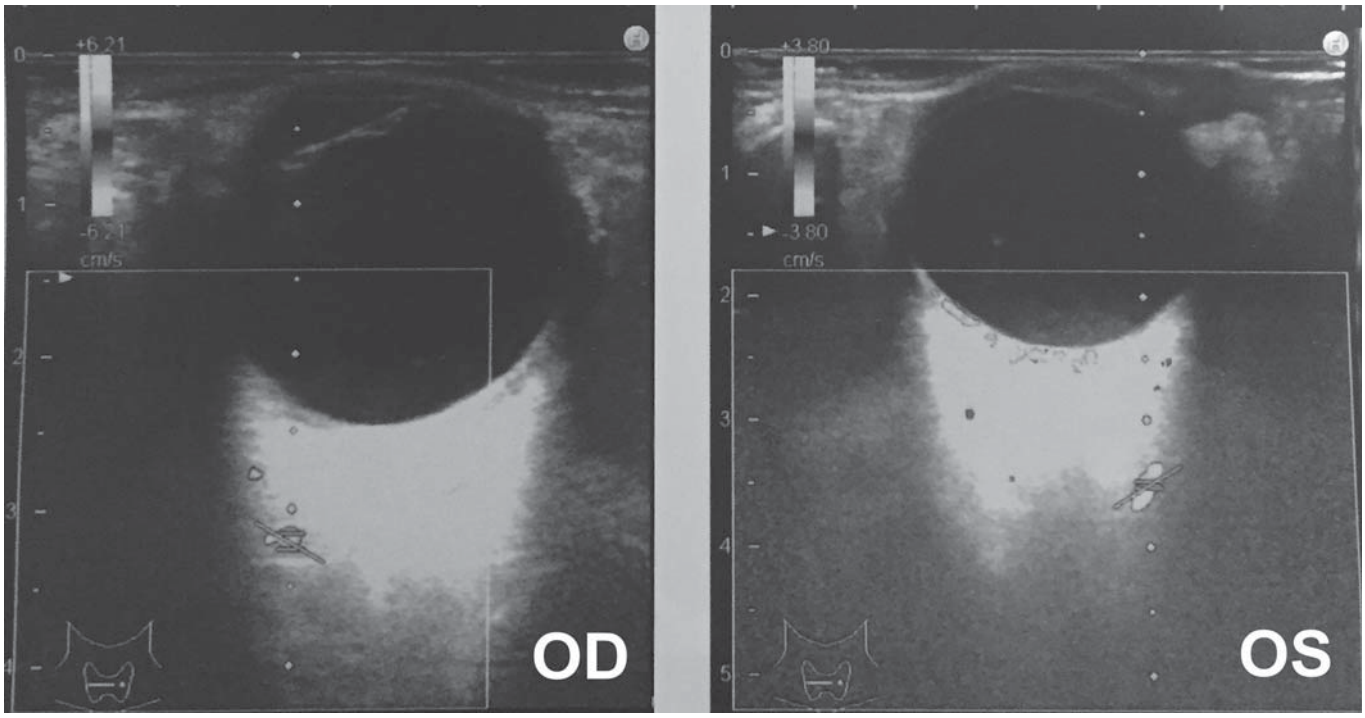

Figure 3. *Initial B-mode ocular ultrasonography: there were no remarkable suprachoroidal effusion and choroidal detachment.*

iochoroidal effusion, ciliary body edema and swelling due to sulfonamide agents may lead to relaxation of zonules and anterior rotation of the iris-lens diaphragm, anterior chamber shallowing, angle closure and subsequently IOP elevation.[8,10-12]Although the above mentioned pathophysiological events, it is thought to be that predominantly an immune or idiosyncratic reactions and a prostaglandin-mediated effect cause ocular side effects.[13] The most probable hypothesis that can explain the idiosyncratic response is the hapten hypothesis. The reactive drug metabolites binding to the proteins are recognized as immunogen agents by the body and induce an immune response.[5]

Also, sulfa-derivative drugs may affect prostaglandin metabolism. Thus, the stimulated production of prostaglandin agents may cause miosis, increased vascular permeability, ciliochoroidal edema and additionally, leukotrienes may cause ciliary spasm.[10,13]

Another important point in the differential diagnosis of the indapamide induced side effects is to exclude a possible angle closure glaucoma and pupillary block. Thus an unnecessary iridotomy will be avoided.[8]

There were four case reports in the literature presenting with indapamide induced ocular side effects and only two of them showed bilateral angle closure and IOP elevation.[2-5] Végh et al.[2] reported a case of acute myopia, angle-closure, shallow anterior chamber and mild IOP elevation due to oral indapamide and demonstrated ciliochoroidal edema and ciliary body swelling by using UBM. They also argued that ciliary muscle spasm was accompanied to the pathology and they showed regression in refractive changes and patient complaints with the use of cyclopentolate. Contrary, it has been argued that accommodation spasm is not a possible contributing factor for the development of myopia.[14] Presumably, an allergic or idiosyncratic ciliary body edema leads to anterior displacement of the iris lens complex and myopic shift and cyclopentolate use has no effect on the refractive status.[1,8] Similarly, we did not use cyclopentolate treatment and achieved spontaneous resolution in the refractive changes.

Senthil et al.[5] presented a case of 53 years old female patient with bilateral simultaneous acute angle closure and IOP elevation (OD: 53 mmHg, OS: 52 mmHg) after oral intake of 2.5 mg indapamide for essential hypertension. They showed 360 degree ciliochoroidal effusion and detachment by using UBM. They strated antiglaucomatous treatment and after 5 days of discontinuation of the drug the clinical findings resolved and IOP levels decreased at normal levels.

In another case presented by Blain et al.[4] a 38 years old male patient developed -5D sph myopia and shallow anterior chamber after the use of 2.5 mg indapamide tablet once a day. The IOP values were within normal ranges in this case. They showed ciliochoroidal detachment, and diffuse choroidal thickening using ultrasonography. The symptoms and clinical findings resolved with no treatment after the discontinuation of the indapamide.

EDI-OCT enables in vivo detailed and quantitative evaluation of the choroidal structural changes. We demonstrated

choroidal thickening by using EDI-OCT due to indapamide intake for the first time. The limitation of the our report was the absence of UBM in our clinic. So we could not present UBM images.

Sulfo-derivative agents have known significant ocular side effects. So during the treatment process, the clinicians should be aware of the side effects associated with these drugs, if necessary, these patients can be consulted with an ophthalmologist. Finally EDI-OCT may be a beneficial imaging modality to monitor ocular side effects due to indapamide.

Declaration of patient consent

The patient consent form was obtained from the patient.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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