An unexpected complication in bilateral acute iris transillumination: Cystoid macular edema

Cigdem Altan, Berna Basarir, Cem Kesim

A 47-year-old male presented with bilateral 4 + circulating pigment in the anterior chamber, diffuse iris transillumination, dilated pupils unresponsive to light, and high intraocular pressure (IOP) levels in both eyes. Visual acuity and IOP improved bilaterally with topical steroid and antiglaucomatous therapy. In the 10th month, bilateral cystoid macular edema (CME) was developed and resolved after subtenon triamcinolone injections. CME recurred after cataract surgery in the right eye which was treated with intravitreal dexamethasone implant injection. CME was recurred in the left eye and treated with intravitreal dexamethasone implant at the same setting with cataract surgery. CME can be seen in the course of bilateral acute iris transillumination (BAIT). This is the first BAIT case presenting with bilateral CME.

Key words: Bilateral acute iris transillumination, complication, cystoid macular edema

Bilateral acute iris transillumination (BAIT) is a clinical entity which features acute pigment discharge originating from iris pigment epithelium and creates diffuse iris transillumination, accompanied with irregularly mydriatic pupil due to variable sphincter paralysis and occasional intraocular pressure (IOP) rise.[1] Although association with the use of systemic fluoroquinolones and other antibiotics is suspected, the etiopathogenesis of this entity remains unclear.[1,2]

We herein present a case with BAIT who unexpectedly developed bilateral cystoid macular edema (CME) during a recurrent attack. This is the first case of CME in BAIT which is reported in the literature.

Case Report

A 47-year-old male patient applied for emergency service with complaints of decreased vision, redness, and photophobia in both eyes. His visual acuity (VA) was counting fingers at 1-m left eye (OD) and at 50-cm right eye (OS) with IOP levels measuring 34 mmHg OD and 20 mmHg OS, respectively. The patient was given topical corticosteroids and also oral acetazolamide 250 mg t.i.d. and topical brinzolamide + timolol maleate fixed combination.

On the 1st day visit, VA improved to 20/67 in the right and 20/120 in the OD. Slit-lamp examination revealed bilateral corneal edema with Descemet's folds, dense pigment precipitates on the corneal endothelium, 4 + circulating pigment in the anterior chambers, posterior synechiae with broad bases, and pigment dusting over anterior lens capsules in both eyes. He had diffuse transillumination of the iris and mydriatic pupils unresponsive to light in both eyes [Fig. 1]. Heavy angle pigmentation was observed on gonioscopy in both eyes [Fig. 2]. Fundus examination including optic nerve heads was normal. He had no systemic illness, but he had received oral moxifloxacin and amoxicillin because of an upper respiratory tract infection 2 weeks before the onset of ocular symptoms. The morphologic changes of irides were documented by anterior segment optical coherence tomography (OCT) imaging [Fig. 2]. These findings suggested BAIT diagnosis. The patient was advised to continue antiglaucomatous therapy and topical corticosteroid drops every hour.

On the 3rd day visit, VA improved to 20/25 OD and 20/32 OS, and IOP levels decreased to 10 mmHg for the OS and 14 mmHg for OD.

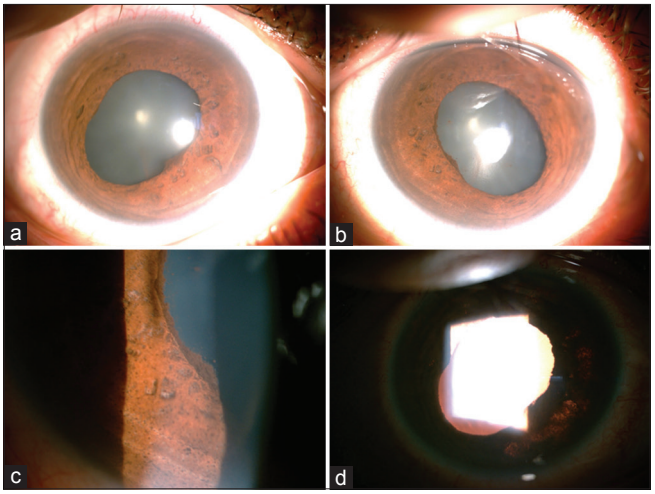

Figure 1: Biomicroscopic findings of two eyes. The mydriatic pupils are unresponsive to light in the right (a) and the left (b) eyes; posterior synechiae with a broad base and a thick layer of iris pigment smeared on the surface of the lens and pigment dusting on the surface of the lens (c); severe diffuse iris transillumination on retroillumination (d)

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Department of Eye Clinic, University of Health Science, Beyoglu Eye Training and Research Hospital, İstanbul, Turkey

Correspondence to: Dr. Cigdem Altan, Konaklar Mah. Hukukcular Sit. C4/18 4 Levent, İstanbul, Turkey. E-mail: cigdem_altan@yahoo.com

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On the 3rd week visit, the patient presented with bilateral 4 + pigment in the anterior chambers, atonic pupils, diffuse iris transillumination, and posterior synechiae with 20/20 VA on each eye. IOP levels were regulated to 17 and 16 mmHg with triple topical antiglaucomatous medication. Pigment discharge in the anterior chamber persisted for first 8 months, while VA and IOP levels remained stable; topical steroid dose was sometimes reduced gradually. The patient referred with acute exacerbations on the 3rd and 8th month, presenting redness, pain, and photophobia. Additional topical corticosteroids were administered in recurrent periods. In the 10th month follow-up, the patient presented with blurred vision which was reduced to 20/60 bilaterally. While slit-lamp examination revealed bilateral posterior subcapsular cataracts (PSCCs), fundus examination and macular OCT demonstrated bilateral CME [Fig. 3a and b]. Central macular thicknesses were 658 µm in the right and 451 µm in the OD. There was no finding other than late cystoid pooling and disc hyperfluorescence in the fluorescein angiography [Fig. 3c and d]. The patient underwent laboratory evaluation including full blood count, liver enzymes, blood urea nitrogen, plasma creatinine, viral serology, venereal disease research laboratory test, and intradermal purified protein derivative test, which were found in normal limits. With these findings, the patient received subtenon triamcinolone injections bilaterally, which responded well with resolution of edema and visual improvement up to 20/25 for each eye. IOP levels remained stable with dual medication.

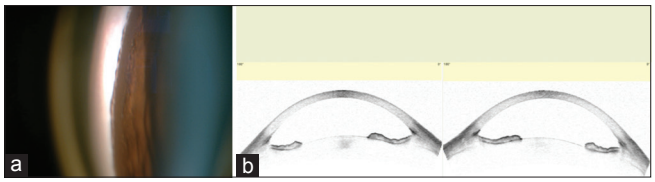

Figure 2: Dense pigment accumulation on gonioscopy obscuring all angle structures (a); anterior segment optical coherence tomography (b)

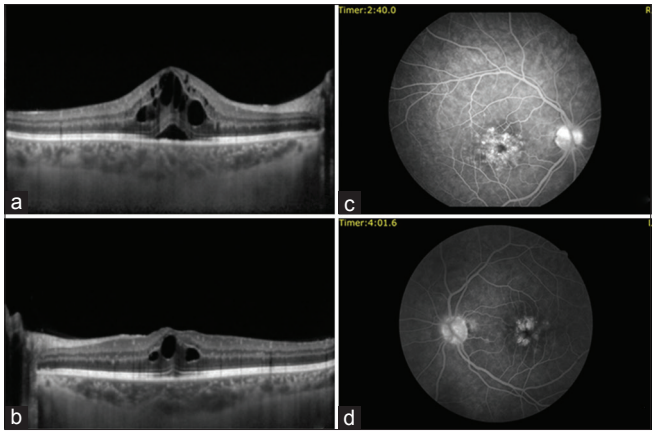

Figure 3: The macular edema was shown by macular optical coherence tomography (a and b), there is no finding other than late cystoid pooling and disc hyperfluorescence in the fundus fluorescein angiography (c and d)

On the 24-month follow-up, the patient underwent uncomplicated phacoemulsification and intraocular lens implantation in the capsular bag surgery on the OS. His VA improved to 20/20 after cataract surgery, but in 4 weeks, he suffered micropsia and visual distortion. Fundus examination demonstrated right CME which later resolved with intravitreal dexamethasone implant injection and CME did not recur during 12 months in the OS. Meanwhile, the untreated OD presented with CME, reducing vision to finger counting at 50-cm, along with 2 + pigment and PSCC. The

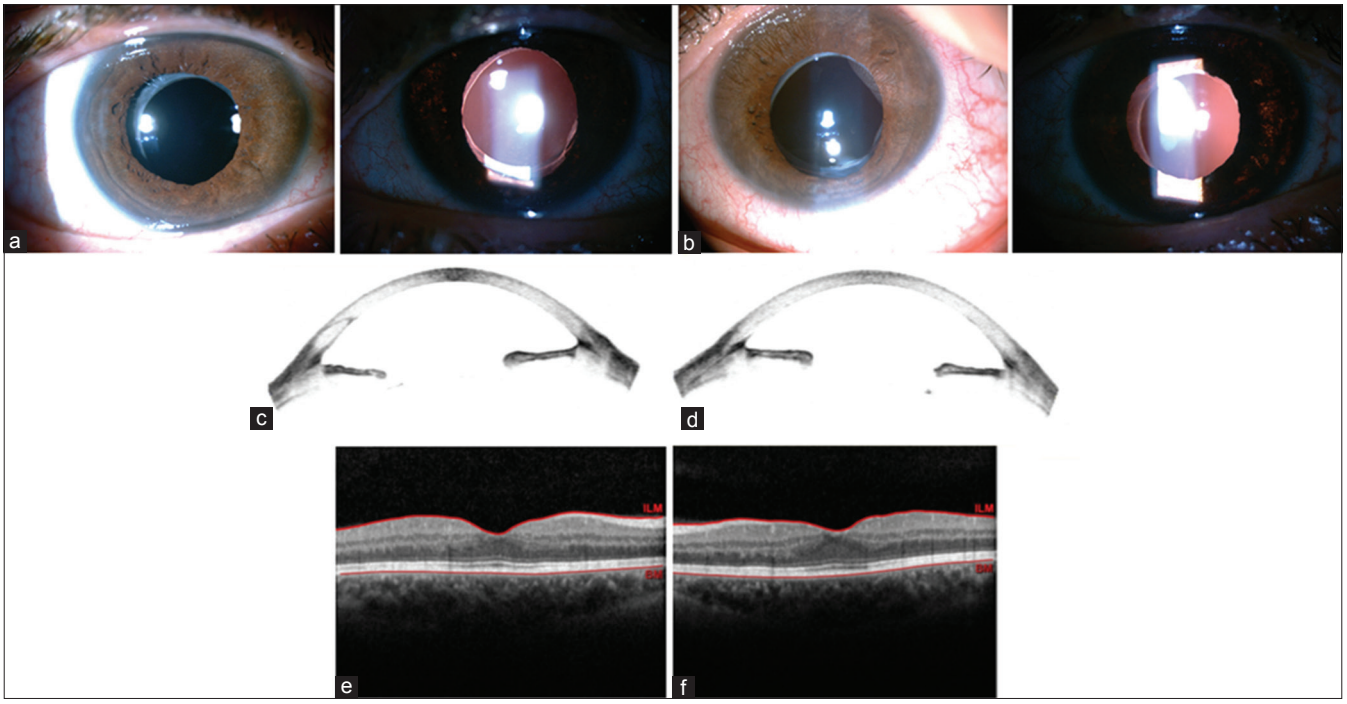

Figure 4: The anterior segment images, anterior segment optical coherence tomography images, and macular optical coherence tomography images of the right (a, c, and e) and the left eyes (b, d, and f) at the last visit

same surgical procedure was applied to the OD which was followed by intravitreal dexamethasone implant injection. In the following 9 months, neither eye has had any recurrences. VA remained 20/20 bilaterally; atonic middilated pupils and centralized posterior chamber intraocular lenses were present in the last performed visit [Fig. 4].

Discussion

BAIT was first described as a different clinical entity by Tugal-Tutkun *et al*.[1] following previously reported similar cases which had been attributed to the use of oral moxifloxacin by their authors.[3-5] According to the widest series of patients, Tugal-Tutkun *et al*. defined bilateral diffuse iris transillumination, mydriatic, irregular pupil border, and pigment showering with IOP rise as characteristic features of the disease. All the patients had an acute onset of ocular symptoms including severe photophobia and red eyes mostly. The duration of the disease was reported to be between 2 weeks and 14 months (median 2 months).[1]

Our case had all characteristic signs mentioned above and also had a history of previous upper respiratory tract infection and oral moxifloxacin use. However, our case differs from BAIT entity with its relatively long duration (approximately 39 months), recurrences following topical steroid tapering, and, more importantly, bilateral CME that has not been described yet as a BAIT feature. The prolonged clinical course along with recurrent attacks might have triggered CME development, which accentuates the inflammatory constituent in the pathogenesis of disease. It should be acknowledged that CME in the OS might have developed due to cataract surgery, the condition well-known as Irvıne–Gass and Norton syndrome.[6,7] However, CME development in the OD before surgery can exclude this condition.

Conclusion

To the best of our knowledge, this is the first BAIT case presenting bilateral CME. Given that our case also required a long-term follow-up with recurrent episodes, it is important to make further investigation on the etiology and proper management including inflammation control along with IOP regulation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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