

Bilateral Acute Iris Transillumination without Prior Systemic Antibiotics

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Abstract

Purpose: To describe a case of bilateral acute iris transillumination (BAIT) with no history of systemic infections or antibiotics intake before the attack.

Methods: This study included the review of the clinical record of the patient.

Results: A 29-year-old male was referred to the glaucoma clinic with presumed bilateral acute iridocyclitis with refractory glaucoma. Ophthalmic examination revealed bilateral pigment dispersion, marked iris transillumination, dense pigment deposition in the iridocorneal angle, and high intraocular pressure. The patient was diagnosed with BAIT and was followed for 5 months.

Conclusion: The diagnosis of BAIT can be elicited even without a prior history of systemic infection or antibiotics intake.

Keywords: Iridocyclitis, Iris transillumination, Pigment dispersion

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INTRODUCTION

Bilateral acute iris transillumination (BAIT) is a lately introduced term describing bilateral disease characterized by acute extensive iris transillumination associated with pupil sphincter paralysis and pigment dispersion.¹ According to a recent review, 93 cases were described in 19 articles.² BAIT was first described as a side effect of treatment with systemic moxifloxacin in 2004.¹ The exact etiology of the disease is still unknown. A number of antibiotics as well as viral and bacterial infections were suggested as possible predisposing factors.³ The presence of the pigments in the anterior chamber is often misdiagnosed as other entities mostly pigment dispersion syndrome (PDS) and iridocyclitis.^{4,5}

We present a case with BAIT that was misdiagnosed as bilateral acute iridocyclitis. Unlike other case reports of a similar entity, our patient did not encounter any recent systemic infections or received any antibiotics before his ocular symptoms.

CASE REPORT

A 29-year-old Egyptian male working as an air conditioner technician presented to our casualty department with acute blurred vision, redness, photophobia, and pain in both eyes with a history of recurrent mouth ulcers. The patient had no special habits. On ophthalmological examination, his corrected distance visual acuity (CDVA) was 20/80 OD and 20/70 OS. He had a bilateral ciliary injection, +3 cells, and corneal edema, and his intraocular pressure (IOP) was 40 mmHg OD and 45 mmHg OS. The patient was suspected to have acute iridocyclitis. He was referred to the ocular immunology and uveitis clinic for a second opinion. Laboratory workup was carried out, including a complete blood picture, purified protein derivatives, venereal disease research laboratory test, chest X-ray, human leukocyte antigen B27, antinuclear antibodies, antineutrophil cytoplasmic antibodies, and

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angiotensin-converting enzyme. Infectious and autoimmune diseases were ruled out, and a possible diagnosis of idiopathic uveitis was given. He was referred to our glaucoma clinic for controlling the intractable high IOP.

On showing in our clinic, he was on topical prednisolone acetate (Pred Forte, Allergan, Westport Co., Mayo, Ireland) (eight times a day), cyclopentolate T.D.S, combined timolol 0.5%, dorzolamide 2% B.I.D, and brimonidine tartrate 0.2% B.I.D with oral acetazolamide 250 m T.D.S for 4 weeks. His CDVA was 20/30 in both eyes with IOP dropped to 32 mmHg OD and 34 mmHg OS. Slit-lamp examination revealed bilaterally 2+ pigment dispersion in the

anterior chamber with no Krukenberg’s spindles, extensive transillumination defects in 360° of the iris, and bilaterally dilated pupils irresponsive to light [Figure 1a-d]. A cup-to-disc ratio of 0.5 OD and 0.6 OS with unremarkable findings of the posterior segment was detected [Figure 2]. Gonioscopy demonstrated open angles with dense pigment obscuring all iridocorneal angle components and sectoral posterior bowing of the iris but no peripheral anterior synechiae [Figure 3a and b].

During the medical history review, the patient denied receiving any systemic antibiotics or having recent systemic infections apart from coronavirus disease-2019 (COVID-19) infection almost 1 year before the onset of his ocular symptoms. Only systemic nonsteroidal anti-inflammatory tablets (Epifenac [diclofenac sodium] 50 mg) were prescribed for finger bruises. The patient was followed up for 3 months until the resolution of pigment dispersion was noticed. Topical prednisolone acetate was tapered and cyclopentolate was stopped. His IOP dropped to 18 OD and 20 mmHg OS with topical combined timolol 0.5% and dorzolamide 2% B.I.D, and his CDVA was stable at 20/25 OU.

Optic nerve analyses using 24–2 Humphrey visual field (Carl Zeiss Meditec, Inc.) showed a normal visual field of the right eye and paracentral deep scotoma in the left eye [Figure 4a and b]. Optical coherence tomography (Triton version 10.13) showed superotemporal thinning of the retinal nerve fiber layer (RNFL) of the right eye, whereas the left eye shows circumpapillary RNFL thinning in both superotemporal and inferotemporal sectors which is parallel to macular thinning. At 8 months, more thinning was noticed in RNFL in both eyes [Figure 5a and b]. This thinning might have been masked by the early disc edema.

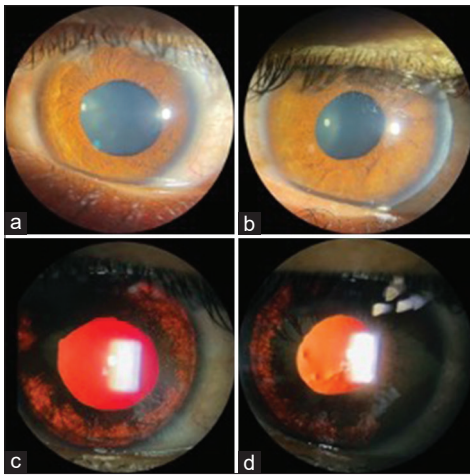


Figure 1: Anterior segment photographs for the right (a) and left eye (b) showing dilated pupils. Slit-lamp examination using retroillumination for the right (c) and left eye (d) demonstrating extensive iris transillumination

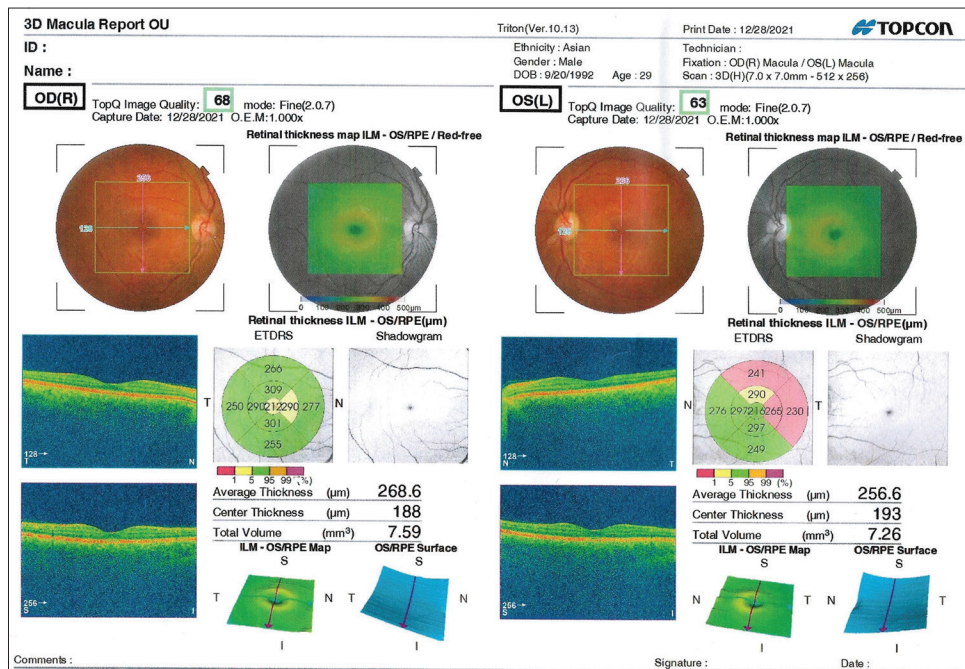


Figure 2: Optical coherence tomography for the macular thickness revealing almost normal thickness in the right eye and superotemporal thinning in the left eye

After 5 months, IOP dropped to 10 mmHg OU. Knowing the self-limiting nature of the disease, the patient was encouraged to stop his antiglaucomatous drops and continue following up. No other attacks were reported. On his last visit, CDVA was stable at 20/20 OU (refraction -0.25 diopter in axis 50° OD and -0.25 in axis 135° OS). These collective findings implied the diagnosis of BAIT syndrome. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

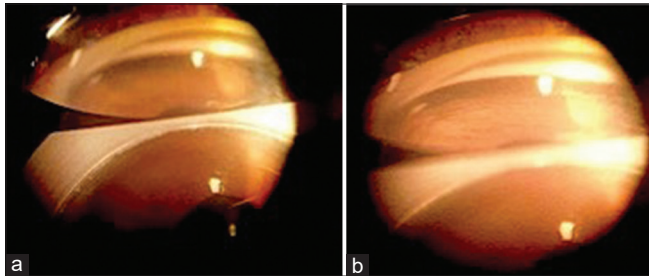


Figure 3: Gonioscopy of the inferior quadrant of the right (a) and left eye (b) demonstrating dense hyperpigmentation of the angles

DISCUSSION

The spectrum of findings that distinguish BAIT includes bilateral extensive iris transillumination, pigment dispersion in the anterior chamber, and an unresponsive dilated pupil. Symptoms develop in an abrupt manner and are rapidly responsive to topical corticosteroid therapy. An interactable IOP rise is a common complication. The exact underlying etiology stays mysterious.³

Although the clinical findings in BAIT seem to be distinctive from other diagnoses with iris depigmentation, the examination findings can overlap. Our patient was misdiagnosed with a case of acute iridocyclitis owing to the acute onset of the symptoms, the dispersed pigments resembling aqueous cells, dilated pupil, and the iris atrophy that could be seen after viral iridocyclitis attacks. However, viral iridocyclitis is often unilateral with sectorial iris atrophy, posterior synechiae, and keratic precipitates all of which were not demonstrated in our case. Further investigations were warranted, including aqueous sampling for viral testing that would be also comprehensive

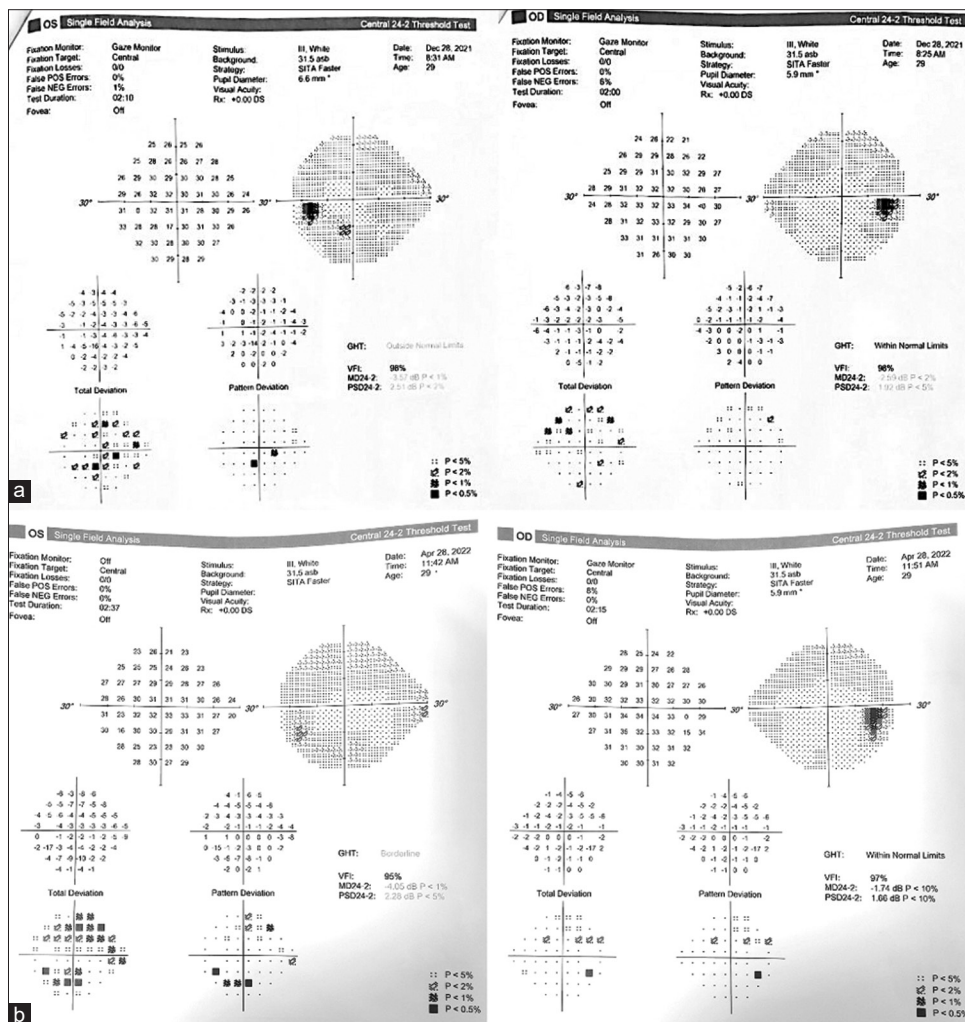


Figure 4: (a) Humphrey visual field report after 5-month follow-up showing a normal visual field of the right eye and paracentral deep scotoma in the left eye. (b) At the 8-month follow-up almost normal visual field of the right eye with around 1 dB drop in the mean deviation in the left eye

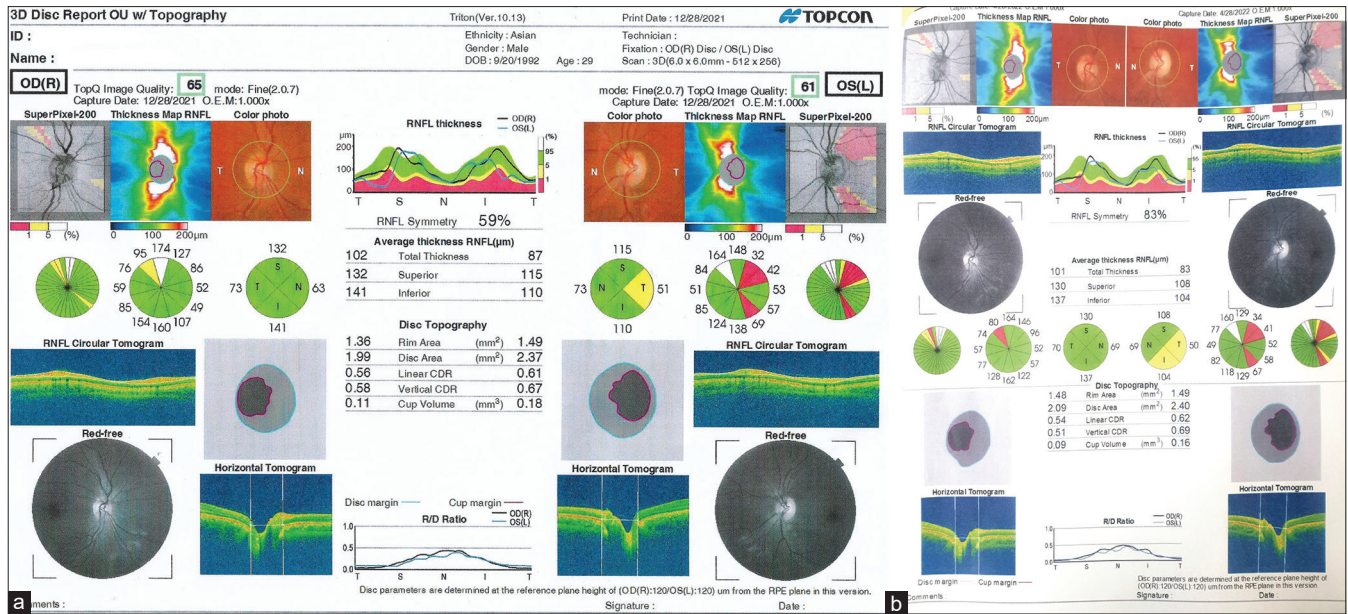


Figure 5: (a) Optical coherence tomography at 5-months follow-up showing superotemporal thinning in the retinal nerve fiber layer in the right eye with thinning in the superior and inferior temporal fibers in the left eye. (b) At 8 months, there was more thinning in the circumpapillary retinal nerve fiber layer in both eyes

for the differentiation, but unfortunately, it was not available.⁶ Gonul and Bozkurt published a similar case report in which the patient was misdiagnosed as having bilateral iridocyclitis. However, in their report, the patient had a history of upper respiratory tract infection treated with systemic cefazolin 3 months before his ocular presentation.⁷

Substantial overlap can exist between the findings of PDS and BAIT, leading to diagnostic perplexity. Bilateral dense hyperpigmentation of the trabecular meshwork with the posterior bowing of the iris and the pigments dispersed in the anterior chamber together with the high IOP added to the young age of the patient all can be seen in both entities. However, symptoms such as injection with severe photophobia encountered by patients with BAIT are not classic for PDS. Furthermore, the extensive geographic iris transillumination spreading from the pupil margin to the peripheral iris differs from the typical radial mid-peripheral spoke-like pattern in PDS. The onset of PDS is also less brutal than that described in BAIT.⁸

Posterior iris bowing was noticed in our patient; a finding that has been often described as a typical feature of PDS. The posterior bowing in our patient was sectorial rather than diffuse as in PDS. The underreporting of this finding in the formerly published reports may be due to the fact that it is a subtle finding which is detected on imaging more than on gonioscopy. It was postulated that the posterior bowing in BAIT patients might be a result of the loss of the iris pigment epithelial structural support. This theory may also explain the pupil distortion found in BAIT patients. This characteristic pupil distortion may also help to discriminate between two entities.⁹

Pseudoexfoliation syndrome (PEX) can also be added to the differential diagnosis of such cases as it shares the iris transillumination and hyperpigmentation of the angle with high IOP. The iris defects in PEX syndrome are located around the pupils with characteristic fibrillar material deposition on the lens surface and pupillary margin. Furthermore, it is usually diagnosed in older patients.¹⁰

Bilateral depigmentation of the iris (BADI) is also a newly described clinical entity that can be contemplated in the list for differential diagnosis. BADI is characterized by symmetrical geographic or even diffuse depigmentation of the iris resulting in significant alteration of iris stromal texture as well as the color with a more benign course and a brief period of pigment discharge, a lower incidence of IOP rise that is only transient, and reversibility of iris changes reported in some patients.^{11,12} The main differences between both diseases are the nontransilluminating depigmentation of the iris stroma and the reactive pupil in BADI.

Patients with BAIT tend to have self-limiting courses except for the abrupt rise in the IOP that require aggressive treatment.⁹ Rivera-Valdivia *et al.* reported one case with BAIT that developed bilateral glaucoma requiring Ahmed glaucoma valve implantation.¹³ In former studies, two patients failed to have IOP control with solely topical drops and required peripheral iridectomy in one patient and laser iridoplasty in the other.^{14,15}

The exact trigger of BAIT remains challenging. Antibiotics such as moxifloxacin were implicated in few reports.^{16,17} Nevertheless, a history of antibiotic usage was not reported in all the patients that were assumed to have BAIT. Tugal-Tutkun *et al.* published a series of 26 patients diagnosed with BAIT in whom only 35% received moxifloxacin.

However, 73% of the included patients shared a common history of respiratory illness. They postulated that BAIT may be triggered by a viral infection rather than moxifloxacin toxicity. This postulation was driven by the lack of published data that report any similar conditions following intravitreal, intracameral, or topical moxifloxacin usage.³ Degirmenci *et al.* reported a case of BAIT who had an ongoing bacterial urinary tract infection. They hypothesized that triggering factors may not be only viral but also bacterial infections as well.¹⁸

We believe our case fits in the diagnosis of BAIT even without a prior history of systemic infection or antibiotics. This report might aid in raising the awareness of this clinical entity to avoid excessive diagnostic evaluation or even treatments. BAIT might be kept in mind during the diagnosis of iridocyclitis and PDS.

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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