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CASE REPORT

Bilateral Acute Iris Transillumination Mimicking Anisocoria

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Abstract

Anisocoria, defined as a ≥ 0.4 mm difference in pupil diameter, may result from physiological or pathological causes. While often benign, it can occasionally indicate serious neurological or ophthalmological conditions. Bilateral acute iris transillumination (BAIT) is a rare, recently defined entity characterized by iris pigment dispersion and sphincter paralysis. A 54-year-old female presented to the emergency department on noticing unequal pupil sizes. Neurological examination and cranio-cervical magnetic resonance imaging were unremarkable. She was referred to ophthalmology for further evaluation. Slit-lamp examination revealed bilateral iris transillumination defects and pupillary mydriasis, more prominent in the left eye. No signs of uveitis, glaucoma, or optic nerve pathology were observed. On detailed anamnesis, she reported systemic moxifloxacin use 1 month earlier for an upper respiratory tract infection. Clinical findings and drug history supported a diagnosis of BAIT. BAIT should be considered in the differential diagnosis of anisocoria, especially when neurological imaging is normal. Early recognition may prevent unnecessary investigations and optimize patient care.

Keywords: Anisocoria; moxifloxacin; pupil disorders; uveitis.

Anisocoria is commonly defined as a difference of 0.4 mm or more in pupil diameter between the two eyes. [1] It can result from a wide spectrum of etiologies, ranging from benign physiological variations to potentially life-threatening conditions, and may be of either neurological or ophthalmological origin.

Among ophthalmological entities, bilateral acute iris transillumination (BAIT) and bilateral acute depigmentation of the iris (BADI) are recently described clinical syndromes. Both conditions are characterized by acute-onset, bilateral, and marked pigment dispersion from the iris. However, a distinguishing feature of BAIT is the presence of sphincter paralysis, which is absent in BADI. [2]

Herein, we present a case of BAIT associated with anisocoria that developed following oral moxifloxacin use for an upper respiratory tract infection. Written informed consent was obtained from the patient for publication of this case.

Case Report

A 54-year-old female presented to the emergency department after noticing unequal pupil sizes while looking in the mirror. She also complained of photophobia. Clinical examination confirmed anisocoria, and she was referred to the neurology department for further evaluation.

Neurological assessment revealed no ptosis, ophthalmoplegia, or cranial nerve deficits. Motor and



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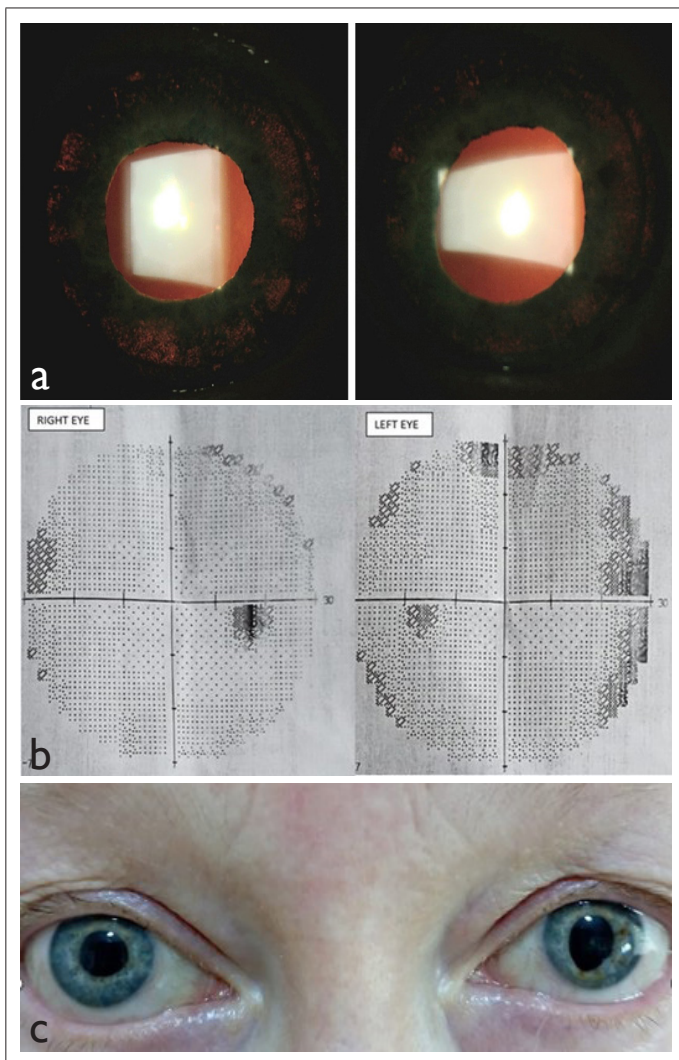


Fig. 1. (a1 and a2) Biomicroscopic photograph of iris transillumination deficiency (right eye is shown in the right side, left eye is shown in the left side). (b) Grey scale of Humphrey 30-2 SITA standard visual field test results with no abnormality other than rim defects, (left to right, consecutive right eye and left eye). (c) Photograph of anisocoria due to pupillary sphincter defect.

sensory examinations were within normal limits. Deep tendon reflexes were preserved, and there were no signs of cerebellar or extrapyramidal involvement.

Contrast-enhanced brain and cervical MRI showed no abnormalities. The patient denied any history of ocular trauma, chronic systemic disease, or prior intraocular surgery. Given the absence of neurological pathology, she was referred to the ophthalmology clinic for further evaluation.

Ophthalmic examination demonstrated best-corrected visual acuity of 0.0 logMAR along with mild impairment in color vision. Pupillary evaluation showed bilateral mydriasis with anisocoria; the right pupil measured 6 mm and the left 4 mm, both with poor reactivity to light. Slit-lamp

examination revealed marked bilateral iris transillumination defects suggestive of pigment epithelial loss (Fig. a1, a2). Fundoscopic evaluation was unremarkable except for thin retinal architecture attributed to prior refractive surgery. Optical coherence tomography and Humphrey visual field testing revealed no structural or functional abnormalities (Fig. 1b).

Further anamnesis revealed that the patient had been prescribed oral moxifloxacin 400 mg once daily for 1 week to treat an upper respiratory tract infection approximately 1 month before presentation. Following completion of the antibiotic course, she developed bilateral ocular redness and was seen at an outside ophthalmology clinic, where she was treated with topical 0.1% nepafenac and a combination of moxifloxacin/dexamethasone eye drops. After resolution of the redness, she noticed persistent anisocoria and photophobia, prompting her emergency department visit (Fig. 1c).

In the absence of any neurological or intraocular pathology that could explain the anisocoria, the constellation of clinical findings – particularly the bilateral iris transillumination, mydriasis, poor pupillary reaction, and recent moxifloxacin use – supported a diagnosis of BAIT.

DISCUSSION

BAIT is a rare but increasingly recognized syndrome characterized by acute, bilateral loss of iris pigment epithelium, iris transillumination defects, and pupillary sphincter paralysis resulting in fixed or poorly reactive dilated pupils.^[2,3] It has been strongly associated with systemic fluoroquinolone exposure, particularly moxifloxacin, and typically follows upper respiratory tract infections.

Although the precise pathophysiology remains unclear, immune-mediated toxicity to the iris pigment epithelium has been postulated. Pigment liberated into the anterior chamber can accumulate in the trabecular meshwork, increasing the risk for secondary ocular hypertension or glaucoma.^[3] Therefore, close monitoring of intraocular pressure (IOP) is essential in these patients.

Anisocoria itself has a broad differential diagnosis, ranging from physiological variants to serious neurological conditions. Physiological anisocoria is generally <1 mm, remains constant under different lighting conditions, and is not associated with other neurological signs.^[4,5]

In the context of BAIT, anisocoria results from iris sphincter paralysis due to pigment epithelial damage, leading to mydriatic and poorly reactive pupils. This

condition can closely mimic other causes of anisocoria with neurological implications, such as Horner syndrome, Adie's tonic pupil, or oculomotor nerve palsy.^[3]

Horner syndrome presents with ptosis, miosis, and anhidrosis resulting from disruption of the sympathetic pathway. Anisocoria in Horner syndrome is typically more pronounced in dim lighting due to the affected pupil's inability to dilate.^[4]

Adie's tonic pupil results from postganglionic parasympathetic denervation and is characterized by a dilated pupil that reacts poorly to light but constricts during accommodation. It is often unilateral and may be associated with diminished deep tendon reflexes (Adie's syndrome).^[4]

Oculomotor nerve palsy usually manifests with ptosis, ophthalmoplegia, and a dilated, non-reactive pupil due to parasympathetic fiber involvement. It is most often caused by compressive lesions or ischemia.^[4]

BAIT can be differentiated from these entities by the presence of bilateral iris transillumination, a history of recent fluoroquinolone exposure, absence of neurological signs, and symmetrical or asymmetrical mydriasis with poorly reactive pupils.

Recognition of BAIT is important to avoid misdiagnosis and prevent unnecessary diagnostic procedures, including neuroimaging and invasive testing. Treatment primarily involves the use of topical corticosteroids to reduce inflammation and pigment dispersion. Monitoring for elevated IOP is essential, as secondary glaucoma may develop and may be resistant to medical therapy, occasionally requiring surgical intervention.^[6]

CONCLUSION

This case highlights the importance of thorough history taking – including recent drug exposure – and a detailed ophthalmologic examination in the differential diagnosis

of anisocoria. In patients with recent systemic antibiotic use, especially fluoroquinolones, and characteristic iris changes, BAIT should be strongly considered.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

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