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Case Report

Bilateral acute iris transillumination: Case report

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Abstract

Bilateral acute iris transillumination (BAIT) is a recently defined disease characterized with bilateral acute, severe pigment dispersion of iris and pupil sphincter paralysis. The etiopathogenesis of the disease is unknown, but antibiotics such as moxifloxacin, clarithromycin, viral infections, and fumigation therapies were considered as probable etiologic factors. A 33-year-old female was referred to our clinic for acute iridocyclitis refractory to azathioprine, colchicum and corticosteroid treatments. Ophthalmic examination revealed bilateral pigment dispersion, significant iris transillumination, heavy pigment deposition in iridocorneal angle, and elevated intraocular pressure. Upon systemic evaluation she was found to have bacterial urinary tract infection. BAIT is an important cause of pigment dispersion and clinicians must be vigilant for this condition to avoid unnecessary diagnostic tests and treatment.

Keywords: Uveitis, Iris transillumination, Masquerade syndrome, Pigment dispersion

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Introduction

Bilateral acute iris transillumination (BAIT) is a new clinical entity characterized by severe transillumination of the iris, acute onset of pigment dispersion in the anterior chamber, and a mydriatic pupil that is unresponsive or poorly responsive to light due to variable sphincter paralysis.[1] The circulating pigment particles in the anterior chamber may be confused with other conditions that also might lead to pigment dispersion (such as pigment dispersion syndrome, pseudoexfoliation syndrome, and iridocyclitis).[2–4]

Herein, we report a case with BAIT occurred after a bacterial urinary tract infection misdiagnosed as acute iridocyclitis.

Case report

A 33-year-old female who was diagnosed as having acute iridocyclitis refractory to systemic and topical treatment was referred to our clinic for second opinion. Upon arrival she had blurred vision, redness, photophobia and pain in both eyes. She was on topical prednisolone acetate (Pred forte, Allergan, Westport Co., Mayo, Ireland) (8 times a day), systemic methylprednisolone (Ultralan, Bayer Schering Pharma AG, Germany) (1 mg/kg/day), systemic azathioprine (Imuran, Glaxo Smithkline, United Kingdom) and colchicine (Colchium dispert, Dr. F. Frik, Istanbul, Turkey) for 2 weeks. Best corrected visual acuity was 20/25 in both eyes with intraocular pressures (IOPs) of 30 mmHg in the right and 32 mmHg in the left eye. Ophthalmic examination revealed bilaterally 4+ pigment dispersion in the anterior chamber, symmetrical diffuse iris transillumination, and pigment deposition on the corneal endothelium (Fig. 1A and B). Gonioscopy demonstrated dense pigment obscuring all iridocorneal angle components (Fig. 1C and D). Anterior segment optical coherence tomography (3D OCT-2000, Topcon Corporation, Tokyo, Japan) demonstrated no iris concavity (Fig. 1E and F). Posterior

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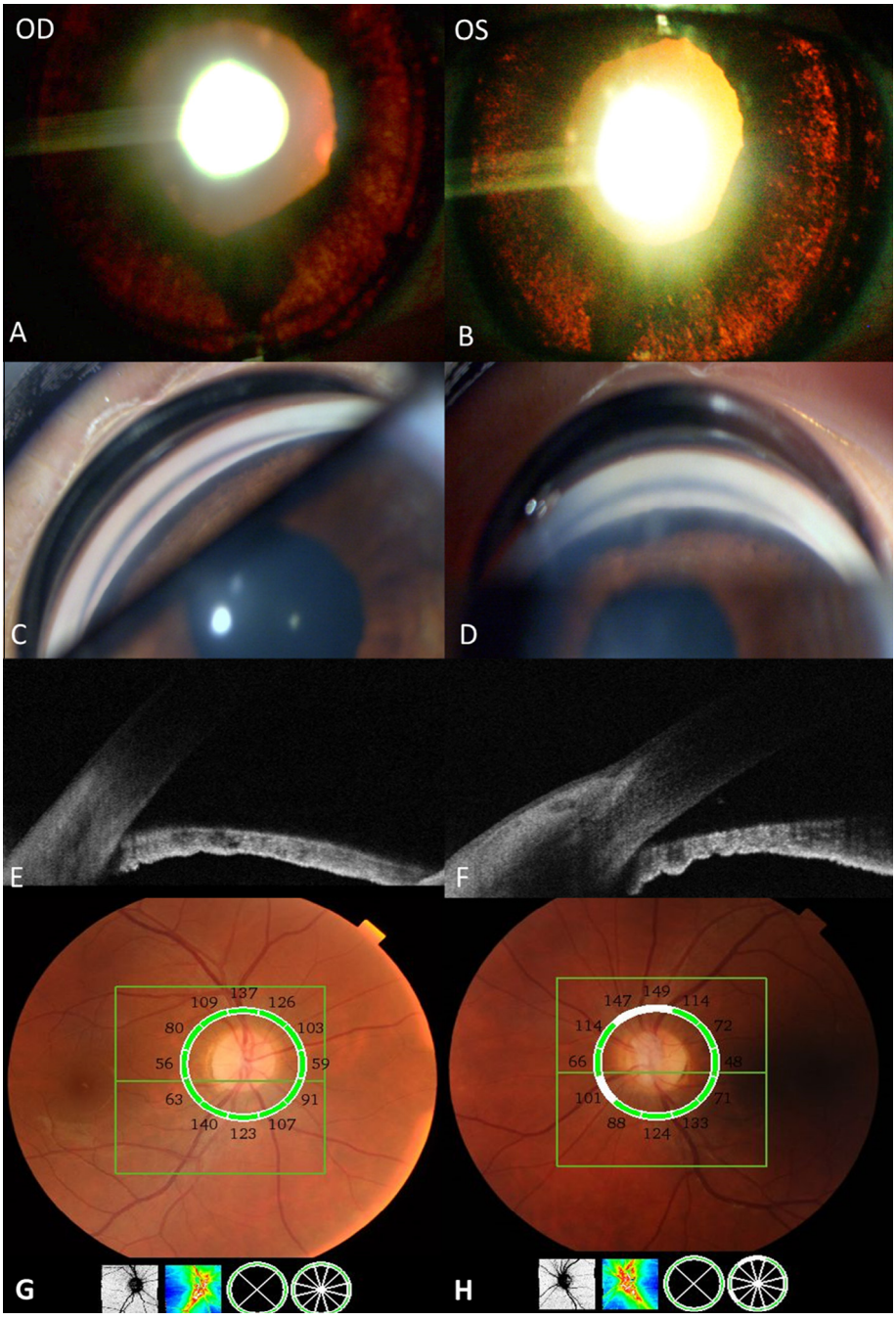

Figure 1. (A and B) Anterior segment photographs show significant iris transillumination, (C and D) gonioscopy reveals heavy pigment deposition in iridocorneal angle, (E and F) anterior segment OCT images demonstrated no backward bowing of the iris. (G and H) Retinal nerve fiber layer thickness and optic nerve photographs.

segment examination was unremarkable. In optic nerve analyses no glaucomatous defect was detected (Fig. 1G and H). Pupils were mydriatic and poorly responsive to light. The patient underwent a detailed laboratory evaluation including liver enzymes, blood urea nitrogen, creatinine, erythrocyte sedimentation rate, C-reactive protein, and viral serology. Serum IgG and IgM antibodies against Epstein-Barr virus, Cytomegalovirus, Herpes Simplex virus and Varicella Zoster virus were evaluated. The serological results showed that IgM antibodies were negative for all of these microorganisms and IgG antibodies were positive but not elevated for all. In complete blood count, neutrophilic leukocytosis was detected and urine analyses and urine microscopy supported bacterial urinary tract infection which was confirmed by an urologist. The etiological agent – confirmed by urine culture – was *Escherichia coli*. The remaining tests were within normal limits.

Systemic corticosteroid was discontinued and topical corticosteroid was tapered to 4 times a day. Bilateral topical dorzolamide/timolol fixed combination (Cosopt, Merck Sharp & Dohme, Paris, France) two times a day was started. With the decrement of IOP to 12/14 mmHg, pigment dispersion decreased gradually and disappeared completely within 2 weeks. Anti-glaucomatous treatment was stopped and IOPs remained within normal limits. At this point steroid

induced glaucoma was excluded. However, bilateral mydriatic pupillae poorly responsive to light and iris transillumination resisted. The patient was followed up for 20 months and in this period IOPs were within normal limits with no medication. Optic nerve analyses showed no glaucomatous defect. No other pigment dispersion attack was experienced. These findings suggested the diagnosis of BAIT.

Discussion

A large case series have recently been reported with an unusual bilateral acute iris transillumination, associated with symptomatic pigment showering after an upper respiratory tract infection.[1] This syndrome has been named as BAIT and demonstrates self-limiting features as in our case. Symptoms of our patient masqueraded acute iridocylitis and ocular findings overlapped pigment dispersion syndrome. Pigment deposition in the trabecular meshwork explained the early IOP rise.

BAIT and pigment dispersion syndrome resemble each other. Both of the diseases affect young people, and usually pigment deposition in the iridocorneal angle is dense and homogeneous. Also iris transillumination is symmetrical. But the onset of the disease in pigment dispersion syndrome is silent and disease progresses slowly in contrast to abrupt and symptomatic beginning in BAIT. And also transillumination defects are always in spoke like pattern because of the disease pathogenesis. Moreover pigment deposition on anterior surface of the lens and iris stroma might also be seen. Anterior segment examination and gonioscopy reveal iris concavity toward anterior surface of the lens. Pupil reactions to light in pigment dispersion syndrome are normal but in BAIT the presence of pupillary atony and distorted pupils are essential properties.[2]

The differential diagnosis of BAIT also includes acute iridocyclitis and pseudoexfoliation syndrome. Acute iridocyclitis is characterized by cellular extravasation and protein exudation in the anterior chamber. Posterior synechiae is also an important property of this entity. In the present case BAIT was mistaken for acute iridocyclitis and was tried to be treated with systemic steroids and immunomodulators. Pseudoexfoliation syndrome is also featured as transillumination defects around pupils; however, on slit lamp examination, fibrillar material accumulation on lens surface and iris sphincter is the hallmark of this entity. Additionally pseudoexfoliation syndrome is usually unilateral at the time of the diagnosis and is seen in older patients.[3,4]

Herpes simplex virus and cytomegalovirus also may cause iris atrophy and pupillary atony. Although IOP elevation and atonic pupils were seen as well, symmetrical and bilateral involvement of iris, and absence of keratic precipitates ensure us to think about BAIT for diagnosis. Furthermore a newly defined clinical entity by Tugal-Tutkun and Urgancioglu[5] Bilateral Depigmentation of Iris (BADI) should be considered in the differential diagnosis. The most important features to differentiate these diseases are the unaffected pupil and the absence of transillumination defect in BADI. The pathophysiology of BAIT still remains unclear. Recently, in a few reports, antibiotics such as moxifloxacin, clarithromycin and fumigation therapies have been implicated.[6–8] Tugal-Tutkun et al.[1] reported the largest series about this syndrome in 2011. According to the report, BAIT is a distinct disease and it is triggered by a viral infection not by antibiotic use. In our patient clinical findings were suitable with BAIT but there was not any medication use or viral infection. However, the patient had an ongoing bacterial urinary tract infection proven with laboratory testing. Probably, triggering factors are not only viral but also bacterial infections. However, more cases with further evaluations are needed to enlighten the exact etiology.

In conclusion, to avoid unnecessary diagnostic evaluation and treatment, clinicians should recall BAIT syndrome to mind in the differential diagnosis of iridocyclitis and pigment dispersion.

Conflict of interest

The authors declared that there is no conflict of interest.

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