Case report

Transient mydriasis in toxoplasmosis retinochoroiditis

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ABSTRACT

Keywords: Mydriasis Anisocoria Toxoplasmosis Retinochoroiditis

Purpose: To report a case of transient anisocoria and mydriasis in a 14 year old boy with toxoplasmosis retinochoroiditis. Observation: The patient presented with panuveitis and mydriasis which persisted for 18 days and spontaneously resolved. Conclusions and importance: Mydriasis is a rare potential neurological manifestation of toxoplasmosis retinochoroiditis. Clinicians should be aware of this rare cause of anisocoria.

1. Introduction

Toxoplasma gondii is an intracellular protozoan parasite which may cause ocular and systemic manifestations in the affected host. Congenital ocular toxoplasmosis is frequently bilateral and acquired toxoplasmosis lesions are typically unilateral although bilateral presentation may occur in approximately 30% of cases.1 The most common finding in ocular toxoplasmosis is retinochoroiditis, a disease in which necrosis of the retinal fiber layer leaves a pigmented scar that can result in decreased vision and potential recurrence. The diagnosis of ocular toxoplasmosis is made by testing for IgG serology in addition to clinical findings. Polymerase chain reaction testing of aqueous specimens can may useful in establishing the diagnosis of undifferentiated retinitis. Intraocular inflammation typically resolves within 6 weeks in immunocompetent individuals. Neurological manifestations of both congenital and acquired toxoplasmosis include but are not limited to seizures, paresis, hemianopia, ataxia or cranial nerve palsies.2,3 Pupil miosis is frequently observed in eyes with panuveitis if anterior chamber inflammation is present. Herein we report a case in which transient mydriasis developed in a symptomatic patient with toxoplasmosis retinochoroiditis.

2. Case report

A 14 year old boy with a history of pica as a child was admitted to the neurology service with decreased vision, headache and right sided mydriasis, greatest under bright illumination conditions (Fig. 1). No pupil constriction could be induced with light or accommodation. There was no history of prior exposure to medications with mydriatic or cycloplegic effects. Examination of old photographs did not reveal any evidence of prior anisocoria. Uncorrected visual acuity was 20/60 in the right eye without pinhole improvement. Visual motility was full and the eyes were orthotropic. Eye pain did not occur with eye movement and there were no signs of orbital inflammation. Examination of the right eye revealed intraocular pressure of 30 mmHg. Small keratic precipitates were present on the endothelium inferiorly, the cornea was otherwise normal in appearance. One plus cell was present both in the anterior chamber and vitreous. A focus of necrotizing retinitis in the temporal midperiphery, with numerous satellite lesions along both arterioles and venules, centered approximately 15mm from the optic disc (Fig. 2). The anterior chamber was deep and the crystalline lens was clear. The left eye was normal. Normal evoked potentials, magnetic resonance imaging and lumbar puncture tests were normal. A complete blood cell count, complete metabolic panel, erythrocyte sedimentation rate, antinuclear antibody, double stranded DNA, and rheumatoid factor were all normal or negative. There was serologic evidence of prior exposure to toxoplasma gondii and cytomegalovirus, the remaining of the serologies for infectious causes of necrotizing retinitis were negative (Table 1). Because the clinical presentation was most consistent with toxoplasmosis retinochoroiditis and was not typical of cytomegalovirus retinitis he was treated with trimethoprim 160mg/sulfamethoxizole 800mg twice daily for 6 weeks, prednisolone acetate 1% four times daily and timolol maleate 0.5% once daily. No pilocarpine or other miotic, mydriatic or cycloplegic agents were instilled into either eye during the first 2 months after presentation. One week after discharge...
from the hospital the intraocular pressure was normal, the anterior chamber was quiet and the headache had resolved. Mydriasis was still present at the next outpatient visit, 18 days following onset, but had completely resolved without sequelae by the time of an outpatient visit 2 months after presentation. Uncorrected visual acuity in the right eye recovered to 20/20, transillumination defects of the iris did not develop and the area of retinitis evolved into a chorioretinal scar.

3. Discussion

Toxoplasmosis is a rare cause of anisocoria. There are two reports in the literature discussing the findings of anisocoria in patients with toxoplasmosis infection. In one case, a 23 year old Caucasian woman with a macular scar presumed secondary to toxoplasmic retinochoroiditis was reported to have an afferent pupillary defect and anisocoria.4 In a series of 43 patients with anisocoria, 16 were reported to have active toxoplasma gondii cervical adenitis, two of which presented symptomatically with headaches and decreased visual fixation.5 Pupil miosis occurs when anterior uveitis results in breakdown of the blood ocular barrier with subsequent release of prostaglandins into the aqueous fluid. Pupil mydriasis may occur in anterior uveitis associated with herpetic viral ocular infection or when the intraocular pressure rises to levels high enough to cause ischemia of the iris sphincter. In this case, mydriasis persisted for 18 days despite normalization of IOP and the intraocular pressure was not elevated to levels which are known to cause ischemia of the pupil sphincter. Although we cannot exclude the possibility that the intraocular pressure may have been elevated to higher levels prior to presentation, there was no corneal edema nor did the patient report vision worse than at presentation to suggest greater elevation of the intraocular pressure. No palpable cervical adenopathy was present and the cervical lymph nodes did not appear enlarged on magnetic resonance angiography images of the neck. We speculate that diffuse involvement of the temporal fundus along with inflammation overlying the long ciliary nerve may have been causative factors in this case.

4. Conclusions

Mydriasis in toxoplasmosis is a rare yet potential neurological manifestation of toxoplasmosis retinochoroiditis. Clinicians should be aware of this rare cause of anisocoria.

Patient consent

Consent to publish this case has been obtained from the patient and legal guardians in writing. Institutional review board waiver was granted.

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Declaration of competing interest

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Appendix A. Supplementary data

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