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Ocular toxoplasmosis in an immunocompetent 8-year-old child: a new active lesion or a late manifestation of a congenital toxoplasmosis?

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PEER REVIEW

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Comments

This case depicts the ocular
toxoplasmosis in an immunocompetent
8-year-old child and the highlights
the importance of considering
T. gondii as a possible cause of
chorioretinitis in children living in
developed/developing countries.
Details on Page 327

ABSTRACT

Infection with the protozoan *Toxoplasma gondii* is one of the most frequent parasitic infections worldwide and the common infection of the retina in the general population. We describe a case report of a chorioretinitis in an immunocompetent 8-year-old patient as a consequence of a underdiagnosed neonatal toxoplasmosis. The boy was successfully managed with pyrimethamine and sulfadiazine. The present case we would like to empathize the importance of considering toxoplasma gondii as a possible cause of chorioretinitis in children living in developed countries and we provide a detailed reviewed of the literature about treatment of *Toxoplasma gondii* infection.

KEYWORDS

Chorioretinitis, *Toxoplasma gondii* infection, Children

1. Introduction

Infection with the protozoan *Toxoplasma gondii* (*T. gondii*) is one of the most frequent parasitic infections worldwide and a common infection of the retina in the general population. Approximately 25%–30% of the world human population is infected by *Toxoplasma*. The prevalence varies widely between countries: low seroprevalence (10%–30%) is observed in North America, South East Asia, Northern Europe, and Saharan countries of Africa, moderate prevalence (30%–50%) in the countries of Central and Southern Europe, and high prevalence in South America and in tropical African countries[1]. Today, ocular toxoplasmosis

in the USA and Western Europe is estimated to cause 35% of cases of chorioretinitis. The ocular toxoplasmosis is a frequent manifestation of the disease in immunodepressed patient or in congenital toxoplasmosis, but is very uncommon in immunocompetent children and occurs in 1% of cases. This disease typically affects the posterior pole of a single eye, lesions can be solitary, multiple or satellite to a pigmented retinal scar and may occur as a result of acute or recurrent infection[2]. Clinical manifestations of toxoplasmosis are highly variable and are influenced primarily by the host immunocompetence. Immunologically normal children who acquired infection postnatally generally remain asymptomatic or show lymphadenopathy

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in 10%–20% of cases; on the contrary, immunocompromised children often show fulminant and rapidly fatal infection. Congenital infection causes a wide variety of manifestations, most of all chorioretinitis and central nervous system lesions, and may also interests heart, lungs and gastrointestinal tract[3]. The severity depends on the time of infection during pregnancy: if the infection is acquired in the first trimester of pregnancy, the maternal–foetus transmission would be less probable (about 17%) but with a severe decorse and damage to several organs, such as visual or hearing impairment, learning disabilities or mental retardation; if acquired late in pregnancy, the transmission would be much more likely (rate transmission is about 65%) but it will be mild or asymptomatic at birth. Transmission of infection in weeks 10–24 results in the highest severity of clinical disease, whereas transmission in the period of 26–40 weeks results in subclinical disease which manifests latter in life. If left untreated, 85% of children with subclinical disease develop signs and symptoms of the disease including chorioretinitis or developmental delays. The transmission and severity of infection in the child may be modified by providing treatment to the mother during pregnancy. Treatment of children with congenital infection can also alter the course of disease, although relapses of chorioretinitis are still seen in treated children[4]. Despite a lack of published evidence for effectiveness of current therapies, most ophthalmologists elect to treat patients with ocular toxoplasmosis that reduces or threatens to impact vision. Classic therapy consists of oral pyrimethamine and sulfadiazine, plus systemic corticosteroid. Substantial toxicity of this drug combination has spurred interest in alternative antimicrobials, as well as local forms of drug delivery. At this time, however, no therapeutic approach is curative of ocular toxoplasmosis[5].

2. Case report

An 8-year-old child came at the attention of our Pediatric Infectious Disease Unit. During our evaluation obstetrical and perinatal history were investigated and maternal history suggested toxoplasmosis during late pregnancy. Serological tests documented maternal negative serology at 32nd week of pregnancy. Delivery occurred at 40th week of gestation without complications, except for the occurrence of maternal laterocervical lymphadenopathy. The newborn was in good clinical conditions. Work-up for congenital toxoplasmosis with serological tests, brain ultrasonography and funduscopy weren't performed at birth. During the first years of life he showed a regular growth at 50° centile and normal cognitive development. No symptoms of visual impairments were reported by parents. At 4 years of age,

an ophthalmologist evaluation was performed for suspected decrease in visual activity: visual acuity was 7/10 in the right eye and 8/10 in the left eye, and fundus examination showed a dystrophic macula with vitreal retraction in the right eye. Optical coherence tomography (OCT) didn't reveal any morphological or structural alteration of the retina, then no pharmacological treatment was proposed. Funduscopy was then performed yearly, without revealing more alterations. At 8 years of age, a macular lesion associated with mild visual impairment was identified at the right eye. The OCT exam confirmed an inactive macular lesion compatible with ocular toxoplasmosis (Figure 1). The child came then at the attention of our Pediatric Infectious Disease Unit for an assessment. Serological tests were performed using Chemiluminescence Immunoassay LIAISON® Toxo IgM and IgG test, DiaSorin Laboratories, Saluggia, Italy, and showed toxoplasma IgG 34.0 IU/mL (negative results <7.2 IU/mL, positive results >8.8 IU/mL) and toxoplasma IgM <3 AU/mL (negative results <6 AU/mL, positive results >8 AU/mL). At the same time, ophthalmological evaluation confirmed the macular lesion at the right eye and identified a small pigmented scar in the peripheral retina of the left eye. Given the clinical and serological data compatible with ocular toxoplasmosis, an antibiotic therapy was recommended, but refused by parents. Two years later, the child showed significant visual impairment at the right eye. OCT examination revealed a grey–white focus in the nasal retina of the right eye with vasculitis, haemorrhage and vitreitis, consistent with a newly active lesion. The antibiotic therapy consisting in pyrimethamine (25 mg/d), sulfadiazine (1.5 g twice/d) and dexamethasone (15 mg twice/d) was administered for 2 months, leading to a progressive resolution of inflammation and scarring of the lesion, with simultaneous improvement of visual acuity in 8 weeks.

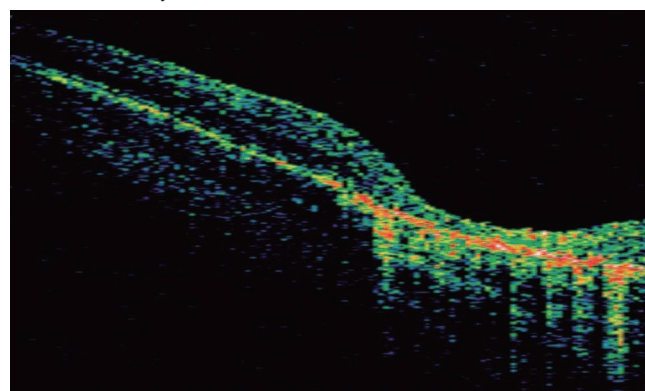


Figure 1. Optical coherence tomography image of toxoplasmic inactive lesion with cross-sectional excavation.

3. Discussion

The apicomplexan parasite *T. gondii* was discovered

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