A Case Report of Longstanding Toxoplasmosis Chorioretinitis

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Abstract: Introduction: Congenital toxoplasmosis (CT) can elicit severe damage to several organs, especially the eye, and may be manifested at birth or later. The diagnosis of ocular toxoplasmic infection is based primarily on the characteristic of ocular findings and supportive serologic evidence. It is generally well agreed that macular lesions, involving the optic nerve, and cases with intense inflammation should be treated. Objective: To describe a clinical presentation and reviews the current management options of reactivation of ocular inflammation, typical of a ocular toxoplasmosis with evidence of congenital infection, a satellite scar or an inactive lesion in the other eye, in which antibodies titer remain as a chronic infection. Material and methods This is a retrospective case report of a 13 years old boy presented with complaints of decreased vision in his left eye for 4 weeks and did not refer clearly for the vision in the right eye. Examination of the fundus oculi noticed cikatrical chorioretinal macular area in the right eye with visual acuity 1/10, and active chorioretinitis, preretinal and intraretinal bleeding in the left eye with visual acuity 1/10. The serology resulted positive for Toxoplasma gondii. He was treated at our Pediatric Department of Infectious Disease Service with classic therapy: Pyrimethamine, Sulfadiazine with supplemental leucovorin (folinic acid) to minimize pyrimethamine associated hematologic toxicity and oral steroids for 4 weeks. Result: The fact that our patient presented initially with such severe anterior inflammation suggests as that this was a longstanding infection. This is further supported by the results of his blood work, IgG for Toxoplasma gondii resulted positive, 650. The patient had presumed ocular toxoplasmosis from his mother, which also resulted IgG positive for Toxoplasma The therapeutic outcome showed improvement in acute case, treatment resulted in reduced retinal area of left eye, without cikatrical signs and no vitreal inflammation with a visual acuity of left eye 10 / 10, and no change in visual acuity of right eye 1/10 with cikatrical central area. Monitoring of blood counts showed no bone marrow suppression after therapy. Conclusion: This case shows a severe presentation of a relatively wellknown clinical entity. Although ocular toxoplasmosis may be self-limiting in immunocompetent individuals, prompt diagnosis and proper management can improve visual outcome. Suddenly blurred vision in the quiet eye in the young adult could be chorioretinal inflammation and scar caused by Toxoplasma gondii. In a case such as this, it is important to utilize the current management options to fight the infection and as well to control inflammation and minimize long-term ocular damage. It is necessary to suspect this pathology in every children attending consultation with vision disorder. Diagnosis should be confirmed by characteristic lesions of fundus oculis and any serological value of Toxoplasma IF1 and treatment should be indicate as fast as possible in active lesions.

Keywords: ocular toxoplasmosis, chorioretinitis, pyrimethamine, sulfadiazine, leucovorin

1. Introduction

Toxoplasmosis gondii is an obligate intracellular parasite that finds its host in mammals and birds. Humans may become infected by ingesting the parasite’s eggs or cysts. Once infected, the parasite may be passed across the placenta from a pregnant mother to her fetus resulting in a congenital form of the disease. Although it is generally accepted that half of all cases are caused by acquired toxoplasmosis and the other half are caused by congenital infections, the prevalence of T. gondii infection varies widely across geographic locations and age demographics.1 Patients with ocular toxoplasmosis most commonly present with complaints of decreased vision, floaters, and sometimes ocular discomfort. Pediatric patients may also present with strabismus, nystagmus, or leukokoria.2 The classic clinical finding of ocular toxoplasmosis is a focal chorioretinitis with overlying vitritis.3 The patient may also have other associated findings such as serous retinal detachments,4,5 vasculitis,6 severe chorioretinitis mimicking acute retinal necrosis,7,8 papillitis,9 mild granulomatous uveitis,2 and scleritis.2,4 Ocular toxoplasmosis is usually a self-limiting condition in immunocompetent individuals.10 The need for treatment is determined based on the location of the lesion and severity of the inflammation. Classical treatment of ocular toxoplasmosis is with pyrimethamine, sulfadiazine, and prednisone.11

2. Objective

We want to describe a clinical presentation and reviews the current management options of reactivation of ocular inflammation, typical of a ocular toxoplasmosis with evidence of congenital infection, a satellite scar or an inactive lesion in the other eye, in which antibodies titer remain as a chronic infection.

3. Material and Methods

This is a retrospective case report of a 13 years old boy presented with complaints of decreased vision in his left eye for 4 weeks and did not refer well for the vision in the right eye. Examination of the fundus oculi noticed cikatkarial chorioretinal macular area in the right eye with visual acuity 1/10, and active chorioretinitis, preretinal and intraretinal hemorrhage in the left eye with visual acuity 1/10. (Fig 1) The serology resulted positive for Toxoplasma gondii. He was treated at our Pediatric Department of Infectious Disease Service with classic therapy: Pyrimethamine, Sulfadiazine with supplemental leucovorin (folinic acid) to minimize pyrimethamine associated hematologic toxicity and oral steroids for 4 weeks. (Fig 2)
3.1 Figures

Figure 1: OS (oculus sinister) Active superotemporal toxoplasmosis chorioretinitis. Preretinale and intraretinale bleeding. OD (Oculus dexter) Old cicatrical corioretinal toxoplasmosis area.

Figure 2: OS Active toxoplasmosis area toward remission. OD Old cicatrical toxoplasmosis area.

Figure 3: OS Vitritis significantly reduced, as well as bleeding. Toxoplasmosis area still active.

Figure 4: OS Toxoplasmosis area still active, no significant inflammatory activity in vitreous.

4. Discussion

T. gondii is an obligate intracellular parasite that finds it host in mammals and birds. Cats are thought to be the classic host because they are the only host that can shed environmentally resistant eggs called oocytes. Humans may become infected when they ingest the oocytes in feline fecal matter. This may occur through direct exposure to cats or by consuming contaminated fruits or vegetables. Humans are also susceptible to T. gondii cysts, or bradyzoites, by ingesting undercooked meat. Once infected, the replicating form of the parasite, or tachyzoite, may be passed across the placenta from a pregnant mother to her fetus, resulting in a congenital form of the disease. The prevalence of T. gondii infection varies widely across geographic locations and age demographics. A total of 22.5% of the United States population has been infected, and it is estimated that 2% of the population has had an episode of ocular toxoplasmosis. Diagnosis of ocular toxoplasmosis is based largely on the clinical examination. Serologic testing for antitoxoplasmosis antibodies, polymerase chain reaction analysis of aqueous humor and vitreous, and culturing aqueous humor and vitreous fluid may be used to further strengthen or prove the diagnosis. The classic presentation of toxoplasmosis chorioretinitis is a bright chorioretinal lesion with an overlying vitritis. There have been reports in the literature of atypical clinical presentations of a mild granulomatous anterior uveitis. The fact that our patient presented initially with such severe anterior inflammation suggests that this was a longstanding infection. This is further supported by the results of her blood work. Immunoglobulin M (IgM) is the first immunoglobulin to appear in the immune response. Immunoglobulin G (IgG) is the predominant antibody in the secondary immune response, which is most commonly seen with recurrent exposure to a specific antigen. A recently published study found that IgM remained positive in patients with T. gondii infections for 12 to 18 months, and the IgG avidity remained low for a maximum of 4 months. Our patient had no clinical signs of prior infection in the retina or radiologic evidence of prior infection in the central nervous system. Therefore, this primary lesion had likely been active for several months before treatment. Ocular toxoplasmosis usually is a self-limiting condition in immunocompetent individuals. The need for treatment is determined based on the location of the lesion and severity of the inflammation. Classic treatment of ocular toxoplasmosis has been with pyrimethamine, sulfadiazine, and prednisone for 4 to 12 weeks. Prognosis depends on the location and severity of the infection. Visual outcome is usually less favorable when the lesion is close to, or involving, the macula. One study reports 40% of patients with ocular toxoplasmosis had a final visual acuity of 20/100 or worse, and 16% of patients had a visual acuity between 20/40 and 20/80. Ocular toxoplasmosis may reactivate in up to 79% of patients.

5. Results

The fact that our patient presented initially with such severe anterior inflammation suggests us that this was a longstanding infection. This is further supported by the results of his blood work. IgG for Toxoplasma gondii resulted positive, 650. The patient had presumed ocular toxoplasmosis from his mother, which also resulted IgG positive for Toxoplasma. The therapeutic outcome showed improvement in acute case, treatment resulted in reduced retinal area of left eye, without cicatrical signs and no vitreal inflammation with a visual acuity of 10/10. The patient had no clinical signs of prior infection in the retina or radiologic evidence of prior infection in the central nervous system. Therefore, this primary lesion had likely been active for several months before treatment. Ocular toxoplasmosis usually is a self-limiting condition in immunocompetent individuals. The need for treatment is determined based on the location of the lesion and severity of the inflammation. Classic treatment of ocular toxoplasmosis has been with pyrimethamine, sulfadiazine, and prednisone for 4 to 12 weeks. Prognosis depends on the location and severity of the infection. Visual outcome is usually less favorable when the lesion is close to, or involving, the macula. One study reports 40% of patients with ocular toxoplasmosis had a final visual acuity of 20/100 or worse, and 16% of patients had a visual acuity between 20/40 and 20/80. Ocular toxoplasmosis may reactivate in up to 79% of patients.

6. Conclusion

This case shows a severe presentation of a relatively wellknown clinical entity. Although ocular toxoplasmosis may be self-limiting in immunocompetent individuals,
prompt diagnosis and proper management can improve visual outcome. In a case such as this, it is important to utilize both topical and oral medications to fight the infection and as well to control inflammation and minimize long-term ocular damage.

References


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