International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2014): 5.611

A Case Report of Longstanding Toxoplasmosis Chorioretinitis

Raida Petrela[1], Emarjola Brahimllari[2]

[1,2]Infectious Disease Service, Pediatric Department, University Hospital Center "Mother Theresa", Tirana, Albania,

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 primarly 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 cikatricial 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. 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 reducted retinal area of left eye, without cikatricial 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 cikatricial 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 charachteristic lesions of fundus oculis and any serological value of Toxoplasma IFI 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 leukocoria.[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 o 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 cikatricial 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

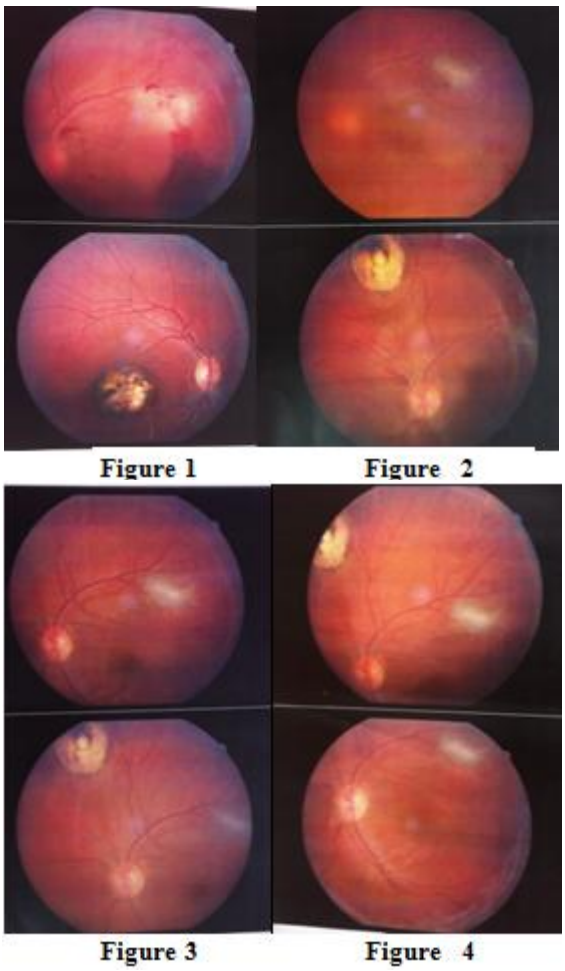

Figure 1: OS (oculus sinister) Active superotemporale. toxoplasmosis chorioretinitis. Preretinale and intraretinale bleeding.OD (Oculus dexter) Old cikatricial corioretinal toxoplasmosis area

Figure 2: OS Active toxoplasmosis area toward remission OD Old cikatricial 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.[13] 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.[14] 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.[15] 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.[16] 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.[2] 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.[17] Immunoglobulin G (IgG) is the predominant antibody in the secondary immune response, which is most commonly seen with recurrent exposure to a specific antigen.[18] 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.[19] 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.[10] 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.[11]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.[23] Ocular toxoplasmosis may reactivate in up to 79% of patients.[3]

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 reducted retinal area of left eye, without cikatricial 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 cikatricial central area.(Fig 3, 4) Monitoring of blood counts showed no bone marrow suppression after therapy.

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

[1] Stanford MR, Tan HK, Gilbert RE. Toxoplasmic retinochoroiditis presenting in childhood: clinical findings in a UK survey. *Br J Ophthalmol* 2006;90(12):1464-7.
[2] Bonfioli AA, Orefice F. Toxoplasmosis. Semin Ophthalmol 2005; 20(3):129-41.
[3] Bosch-Driessen LE, Berendschot TT, Ongkosuwito JV, et al. Ocular toxoplasmosis: clinical features and prognosis of 154 patients. *Ophthalmology* 2002;109(5):869-78.
[4] Smith JR, Cunningham ET Jr. Atypical presentations of ocular toxoplasmosis. *Curr Opin Ophthalmol* 2002;13(6):387-92.
[5] Kraushar MF, Gluck SB, Pass S. Toxoplasmic retinochoroiditis presenting as serous detachment of the macula. *Ann Ophthalmol* 1979;11(10):1513-4.
[6] Diaz-Valle D, Diaz-Rodriguez E, Diaz-Valle T, et al. Frosted branch angiitis and late peripheral retinochoroidal scar in a patient with acquired toxoplasmosis. *Eur J Ophthalmol* 2003;13(8):726-8.
[7] Balansard B, Bodaghi B, Cassoux N, et al. Necrotising retinopathies simulating acute retinal necrosis syndrome. *Br J Ophthalmol* 2005; 89(1):96-101.
[8] Moshfeghi DM, Dodds EM, Couto CA, et al. Diagnostic approaches to severe, atypical toxoplasmosis mimicking acute retinal necrosis. *Ophthalmology* 2004;111(4):716-25.
[9] Song A, Scott IU, Davis JL, et al. Atypical anterior optic neuropathy caused by ocular toxoplasmosis. *Am J Ophthalmol* 2002;133(1): 162-4.
[10] Norose K, Fumie A, Hye-Seong M, et al. Effects of sulfamethoxazole on murine ocular toxoplasmosis in interferon-g knockout mice. *Invest Ophthalmol Vis Sci* 2006;4(1):265-71.
[11] Rothova A, Meenken C, Buitenhuis HJ, et al. Therapy for ocular toxoplasmosis. *Am J Ophthalmol* 1993;115:517-23.
[12] Soheilian M, Sadoughi MM, Ghajarnia M, et al. Prospective randomized trial of trimethoprim/sulfamethoxazole versus pyrimethamine and sulfadiazine in the treatment of ocular toxoplasmosis. *Ophthalmology* 2005;112(11):1876-82.
[13] Dubey JP, Bhaiyat MI, Macpherson CN, et al. Prevalence of toxoplasma gondii in rats (Rattus norvegicus) in Grenada, West Indies. J Parasitol 2006;92(5):1107-8. Figure 10 Chorioretinal scarring with mild activity at center.
[14] Lopez A, Dietz VJ, Wilson M, et al. Preventing congenital toxoplasmosis.*MMWR Recomm Rep* 2000;49(RR-2):59-68.
[15] Hollad GN. Ocular toxoplasmosis: a global reassessment. Part I: epidemiology and course of disease. *Am J Ophthalmol* 2003;136(6):973-88.
[16] Moshfeghi DM, Dodds EM, Couto CA, et al. Diagnostic approaches to severe, atypical toxoplasmosis mimicking acute retinal necrosis. *Ophthalmology* 2004;111(4):716-25.
[17] Pier GB, Lyczak JB, Wetzler LM. Immunology, infection, and immunity. Washington, DC: *ASM Press*; 2004.
[18] Goodman JW. Immunoglobulin structure and function. In: Stites DP, Stobo JD, Fudenber HH, et al, eds. Basic and clinical immunology. *Los Altos, CA: Appleton and Lange*; 1987:27.
[19] Kodym P, Machala L, Rohacova H, et al. Evaluation of a commercial IgE ELISA in comparison with IgA and IgM ELISAs, IgG avidity assay and complement fixation for the diagnosis of acute toxoplasmosis. *Clin Microbiol Infect* 2007;13(1):40-7.
[20] Nicholson DH,Wolchok EB. Ocular toxoplasmosis in an adult receiving long-term corticosteroid therapy. *Arch Ophthalmol* 1976;94(2):248-54.
[21] Sabates R, Pruett RC, Brockhurst RJ. Fulminant ocular toxoplasmosis.*Am J Ophthalmol* 1981;92(4):497-503.
[22] BactrimProduct Information (package insert). Approved 1 August 2005, Amended 27 November 2006.
[23] Atmaca LS, Simsek T, Batioglu F. Clinical features and prognosis in ocular toxoplasmosis. *Jpn J Ophthalmol* 2004;48(4):386-91.

Author Profile

Prof. Dr. Raida Petrela, is graduated in the Faculty of the Medicine with excellent grades. She is professor at Faculty of the Medicine, University of Tirana. Currently, works at Infectious Disease Service, Pediatric Department, University Hospital Center "Mother Theresa", Tirana, Albania.

Emarjola Brahimllari, is graduated in the Faculty of Medicine University of Tirana. She has equivalented her Medicine Laurea in the University of Milan, Italy. Actually, Resident of Pediatrics at the University Hospital Center "Mother Theresa", Tirana, Albania.