Infectious chorioretinitis in an immunocompetent patient: A diagnostic dilemma

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A 44-year-old male presented with a history of defective vision in the right eye for the past 5 months with the previous history of tubercular cervical lymphadenitis. On examination, right eye revealed panuveitis with dense vitritis and chorioretinitis in the superotemporal quadrant. His Mantoux test was positive (25 mm × 25 mm induration), QuantiFERON-TB Gold was test positive, aqueous aspirate was positive for Mycobacterium tuberculosis genome, negative for viruses and toxoplasma, and he was initiated on four-drug antitubercular therapy (ATT) with oral steroids. On follow-up, he had worsening of vitritis and intravenous methylprednisolone was given suspecting paradoxical reaction to ATT; however, a repeat AC tap was positive for toxoplasma B1 genome, IgG antitoxoplasma antibody was also positive in serum and aqueous; hence, we switched to systemic antitoxoplasma therapy. He underwent a therapeutic vitrectomy along with intravitreal clindamycin and dexamethasone for persistent vitreous membranes and vitritis. The patient responded well to the treatment with a reduction in vitritis and scarring of the lesion.

Key words: Chorioretinitis, Goldmann-Witmer coefficient, polymerase chain reaction, toxoplasmosis

Ocular toxoplasmosis usually presents as a focal necrotizing retinochoroiditis with adjacent scar associated with significant inflammation in typical cases and diagnosed mainly on clinical grounds in typical cases.[3] In areas that are highly endemic for both tuberculosis (TB) and toxoplasmosis, patients may present immunologic evidence of both latent infections, and in the absence of characteristic features, the differential diagnosis depends on polymerase chain reaction (PCR) of intraocular fluids.[2] Seropositivity for toxoplasmosis is common worldwide and hence IgG positivity is useful in supporting the diagnosis and not confirming it.[8] The various conditions which mimic ocular toxoplasmosis are TB, necrotizing retinitis, Bartonella-associated neuroretinitis, intraocular lymphoma, punctate inner choroidopathy, and multiple evanescent white dot syndrome.[3] We present a case of ocular toxoplasmosis which mimicked TB and was diagnosed based on PCR of aqueous humor, intraocular antibody production, and clinical judgment.

Case Report

A 44-year-old male presented with complaints of defective vision in the right eye for the past 5 months, was diagnosed previously as posterior uveitis, and treated with two courses of oral steroids and systemic antiviral for 2 weeks by his local ophthalmologist. He had a history of childhood TB in the form of cervical lymphadenitis and had taken antitubercular therapy (ATT) 25 years back. Previous investigations showed Mantoux positivity with induration of 25 × 25 mm with 5 tuberculin units. High-resolution computed tomography was within normal limits, and QuantiFERON-TB Gold test was positive. His HIV status was negative. His best-corrected visual acuity (BCVA) was 20/40 and 20/20 in the right and left eye, respectively.

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Two months later, his BCVA was 20/63 and 20/20 in the right and left eye, respectively. Slit lamp examination showed 2+ vitreous cells and fundus showed persistent chorioretinitis with dense vitritis [Fig. 1b]. Paradoxical reaction to ATT was suspected, and the patient was started on intravenous methylprednisolone (IVMP) 1 g for 3 days. AC tap was repeated which showed positivity for toxoplasma B1 genome by nested PCR [Fig. 3b] and IgG for toxoplasma was positive in aqueous. Following IVMP inflammation regressed, lesions on the retina appeared flat [Fig. 1c]. The patient was started on systemic antitoxoplasma therapy and ATT was discontinued.

One month later, his BCVA was 20/63 and 20/20 in the right and left eye, respectively. Fundus examination revealed vitreous membranes persistent chorioretinitis [Fig. 1d]. The patient was continued on systemic antitoxoplasma therapy and steroids. Due to the persistent vitritis and plenty of vitreous membranes and nonresolving lesions, patient underwent therapeutic vitrectomy along with intravitreal clindamycin (1 mg/0.1 ml) and dexamethasone (400 µg) along with the vitreous biopsy. PCR of the biopsied specimen showed negativity for viruses, toxoplasma, and MTB genome. The patient was advised to continue the oral antitoxoplasma therapy. The patient responded well to the treatment and showed clearance of vitritis along with scarring of lesions [Fig. 1e]. One month later, at the last visit lesions resolved, his BCVA was 20/32 and 20/20 in the right and left eye, respectively [Fig. 1f].

Discussion

As classified by Gupta et al., our patient showed positivity for both tuberculin skin test and QTB assay, RT-PCR of aqueous showed positivity for MTB genome, and fundus showed peripheral chorioretinitis with previous evidence of extrapulmonary TB (biopsy-proven cervical lymph node TB), and hence we considered a diagnosis of presumed ocular TB and started the patient on ATT with systemic steroids.[2] The patient, however, did not respond well to the treatment and had worsening vitritis, a paradoxical reaction to ATT was
suspected, and the patient was given IVMP 1 g for 3 days. After IVMP, lesions persisted while the vitritis appeared to regress. Hence, a repeat AC tap was done which showed nested PCR positivity for toxoplasma B1 genome, IgG anti-toxoplasma antibody positivity in aqueous and serum, and hence systemic antitoxoplasma therapy was added. Although patient responded well to the antitoxoplasma therapy, he had persistent vitreous membranes obscuring vision. Thus, we advised a therapeutic vitrectomy along with intravitreal clindamycin and dexamethasone. However, the vitreous biopsy was negative for toxoplasma, MTB, and viruses. The patient responded very well to the treatment with a reduction in inflammation and lesions scarred. The patient did not require multiple courses of intravitreal clindamycin as systemic antitoxoplasma therapy was continued. Kishore et al. published the first evidence of the use of intravitreal clindamycin might be effective, in the form of a case report in 1998 and a retrospective case series including four patients in 2001. Soheilian et al. found no statistical difference between intravitreal clindamycin with dexamethasone and triple therapy in a study of 68 patients. Papadopoulo et al. demonstrated the efficacy of therapeutic vitrectomy in severe toxoplasmic retinochoroiditis associated with dense vitritis. De Groot-Mijnes et al. showed that with PCR alone, diagnosis was missed in 65% of cases of ocular toxoplasmosis, whereas intraocular antibody production contributes more toward the diagnosis of toxoplasmosis. In a study by Gupta et al., sampling and quantitative analysis of MTB DNA by RT-PCR, they correlated the MTB DNA genome with the site of analysis with less numbers of 4.53 × 10^4 copies in active subretinal mass lesion (vitreous sample) compared to 1.76 × 10^6 copies in retinal pigment epithelium cells by Vasconcelos-Santos et al. They justify the need to correlate the DNA load with clinical presentation in terms of morphology and severity of tubercular uveitis in a larger set of patients.

We believe that in our case, systemic TB in the form of cervical lymphadenitis, treated 25 years back, created a dilemma in the diagnosis. Repeat PCR of the aqueous after 2 months of ATT showed positivity for both toxoplasma B1 genome as well as intraocular anti-toxoplasma antibody production, which contributed more toward the diagnosis of toxoplasmosis. Our patient responded initially well to systemic antitoxoplasma therapy. Due to the presence of persistent vitreous membranes, we attempted therapeutic vitrectomy. We believe, probably the systemic antitoxoplasma therapy, along with intravitreal clindamycin and the therapeutic vitrectomy helped in the resolution of lesions. Our case evoked a diagnostic challenge and demonstrated the importance and limitations of PCR of ocular fluids, especially in areas endemic for TB.

**Conclusion**

Ocular toxoplasmosis manifesting as infectious chorioretinitis in an immunocompetent patient may simulate TB. PCR of aqueous humor, intraocular antibody production, and clinical judgment may help in the diagnosis.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Da Mata AP, Oréfice F. Toxoplasmosis. In: Foster CS, Vitale AT, editors. Diagnosis and Treatment of Uveitis. Philadelphia: W.B. Saunders Company; 2002. p. 385-410.
2. Vasconcelos-Santos DV, Zierhut M, Rao NA. Strengths and weaknesses of diagnostic tools for tuberculous uveitis. Ocul Immunol Inflamm 2009;17:351-5.
3. Holland GN. Ocular toxoplasmosis: A global reassessment. Part I: Epidemiology and course of disease. Am J Ophthalmol 2003;136:973-88.
4. Vasconcelos-Santos DV, Dodds EM, Oréfice F. Review for disease of the year: Differential diagnosis of ocular toxoplasmosis. Ocul Immunol Inflamm 2011;19:171-9.
5. Kishore K, Conway MD, Peyman GA. Intravitreal clindamycin and dexamethasone for toxoplasmic retinochoroiditis. Ophthalmic Surg Lasers 2001;32:183-92.
6. Soheilian M, Ramezani A, Azimzadeh A, Sadooghii MM, Dehghan MH, Shahghadami R et al. Randomized trial of intravitreal clindamycin and dexamethasone versus pyrimethamine, sulfadiazine, and prednisolone in treatment of...
Vitrectomy for epiretinal membrane

A diagnosis of adult-onset Coat’s disease with ERM was made. In view of the poor long-term outcome, the main focus of treatment in Coats’ disease in the past has been the management of telangiectatic vessels in the form of laser photocoagulation or transscleral cryotherapy. Vitreoretinal surgery is commonly used to manage the retinal detachment secondary to exudation or cryopexy.

However, vitrectomy for the macular involvement in Coats’ disease has been limited to a few case reports. Limited reports are also present regarding complications related to vitrectomy for the management of epiretinal membrane (ERM). Early pars plana vitrectomy resulted in resolution of the macular edema and subretinal exudation. A relatively benign rationale of early vitrectomy in such cases are discussed.

Utility of ultra-wide-field imaging and complement for the diagnosis of infectious uveitis. Am J Ophthalmol 2017;118:134-41.

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ocular toxoplasmosis. Ophthalmology 2011;118:134-41.

Papadopoulou DN, Petropoulos IK, Mangioris G, Pharmakakis NM, Pournaras CJ. Pars plana vitrectomy in the treatment of severe complicated toxoplasmic retinochoroiditis. Eur J Ophthalmol 2011;21:83-8.

De Groot-Mijnes JD, Rothova A, Van Loon AM, Schuller M, Ten Dam-Van Loon NH, De Boer JH, et al. Polymerase chain reaction and Goldmann-Witmer coefficient analysis are complimentary for the diagnosis of infectious uveitis. Am J Ophthalmol 2006;141:313-8.

Gupta A, Bansal R, Gupta V, Sharma A, Bambery P. Ocular signs predictive of tubercular uveitis. Am J Ophthalmol 2010;149:562-70.

Sharma P, Bansal R, Gupta V, Gupta A. Diagnosis of tubercular uveitis by quantitative polymerase chain reaction. J Ophthalmic Inflamm Infect 2010;1:23-7.