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Case Report

A Case of Acute Progressive Central Vision Loss After Trauma

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Abstract

Toxoplasmic retinochoroiditis, an inflammatory eye disease caused by the parasite Toxoplasma gondii, manifests as focal necrotizing retinitis with features such as localized scarring, retinal edema, and optic nerve involvement. This report explores the case of a 49-year-old male experiencing progressive central vision loss in his right eye, initially diagnosed as a macular hole. Further results from spectral domain optical coherence tomography (SD-OCT), fluorescein angiography (FA), laboratory testing, and a comprehensive patient history suggested toxoplasmosis chorioretinitis. This case underscores the significance of a thorough history and diagnostics to accurately differentiate between macular holes and infectious etiologies.

Keywords: Toxoplasmosis; Chorioretinitis; Macular Hole; Spectral Domain Optical Coherence Tomography (SD-OCT); Fluorescein Angiography (FA); Trauma

Abbreviations

OD: Right Eye; OS: Left Eye; SD-OCT: Spectral Domain Optical Coherence Tomography; FA: Fluorescein Angiography; AC: Anterior Chamber

Case Report

A 49-year-old male with no significant ocular history presented with a progressively enlarging black dot in the center of his right eye vision, for the last three days. One day prior, the patient reported falling from a ladder but denied head strike. An external ophthalmologist diagnosed him with a macular hole.

The patient was born in Portugal and moved to Massachusetts when he was four years old. He had previously used intravenous drugs and was being treated for psoriasis with Humira at the time of presentation. His review of systems was otherwise negative including no ocular pain, fever or headache.

The ocular examination showed a visual acuity of hand motion in the right eye (OD) and 20/20 in the left eye (OS). Intraocular pressure was 14 OD and 16 OS. The right eye dilated fundus examination is seen in Figure 1. Autofluorescence revealed hyperautofluorescence of the foveola surrounded by relative hypofluorescence.

Spectral Domain Optical Coherence Tomography (SD-OCT) showed evidence of intraretinal edema, hyperreflectivity, thickening and no full thickness macular hole (Figure 2A). Fluorescein Angiography (FA) showed vasculitis with significant leakage in the macula. Given the evidence of vasculitis on FA, laboratory work up and anterior chamber (AC) tap was the next best step to elucidate the underlying etiology.

Laboratory testing revealed elevated white blood cells (11.47 WBCs/µl), C-reactive protein (8.2 mg/L), Erythrocyte sedimentation rate (23 mm/h) and was positive for Toxoplasma IgG, Toxoplasma IgM (>160.0 AU/mL), Bartonella Quintana IgM (1:20) and

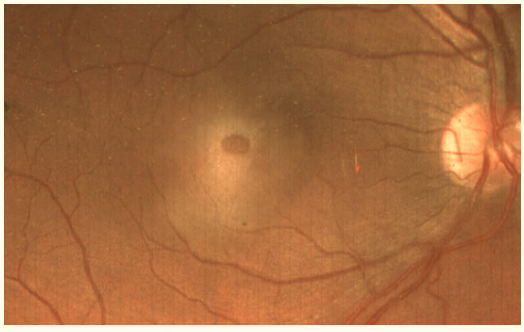

Figure 1: Fundus photography of the right macula of the patient.

Coxsackie B1-B6 (1:8-1:64). The patient was QuantTB Gold, Fluorescent Treponemal Antibody, VZV IgM (0.9 immune status ratio), CMV IgM (<30.0 AU/mL), HIV, HSV IgM, Lyme IgM negative and had normal Angiotensin converting enzyme (50 U/L). The AC tap was negative for Toxoplasma, CMV, VZV, and HSV.

The elevated but low Bartonella Quintana IgM titer was thought to be either a sign of early infection or a false positive. Further questioning also revealed that the patient has chronic and frequent exposure to multiple cats. Although the AC tap was negative for Toxoplasma, we suspected Toxoplasmosis chorioretinitis given the patient's history of immunosuppression, cat exposure and significantly elevated Toxoplasma IgM results. Azithromycin 500 mg daily was initiated due to effectiveness against both Bartonella and Toxoplasmosis infections.

Other courses of action were not indicated. Given the patient's rapidly worsening symptoms, observation would allow progression of the patient's infectious process causing more extensive macular scarring, leading to a worse visual outcome. Vitrectomy with macular hole repair would not be appropriate since no macular hole was present. Treatment with oral prednisone can be useful to control inflammation and immune hyperactivity; however, in this case, steroids would have caused worsening of the patient's infection by suppressing his immune system.

Toxoplasmic retinochoroiditis or 'ocular toxoplasmosis' is an inflammatory eye disease that follows intraocular infection from the parasite Toxoplasma gondii [1]. It is a common cause of posterior uveitis worldwide, with a higher prevalence reported in South America and Africa [5]. Routes of infection include oral or parenteral routes via oocysts in cat feces, uncooked meat, contaminated water, or through maternal-fetal transmission, and rarely from donated blood or organs [2-4]. Toxoplasma chorioretinitis typically presents as focal necrotizing retinitis with localized scarring and can be associated with retinal edema, optic nerve head edema, optic neuritis, iridocyclitis, and in rare cases, panuveitis [5]. Standard therapy for ocular toxoplasmosis combines pyrimethamine, sulfadiazine and corticosteroids, or alternatively, trimethoprim-sulfamethoxazole, azithromycin, clindamycin, or atovaquone can be used until there is resolution of the active lesion and a negative serological test [2,6].

Our patient was advised to follow up in three weeks to repeat Bartonella (1:20), Toxoplasma, VZV IgM (0.90 immune status ratio), and Coxsackie testing (1:8-1:32), which was stable. Toxoplasma IgM (87.40 AU/mL) was seen to be down trending.

Patient outcome

Given elevated Toxoplasma IgM titers, acute toxoplasmosis seemed most likely. The patient completed a six-week course of azithromycin 500 mg daily and sulfamethoxazole-trimethoprim 800-160mg twice a day, then reduced to prophylactic dosing, sulfamethoxazole-trimethoprim 800-160mg three times a week. Our patient responded well to therapy but has a guarded prognosis due to his macular scar. At two-year follow-up, best-corrected visual acuity was 20/800 OD and 20/20 OS. Slit lamp exam and SD-OCT showed a central pigmented round scar (Figure 2B).

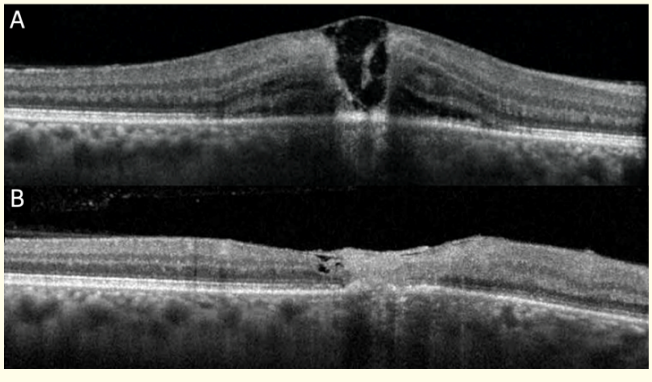

Figure 2: (A) SD-OCT one day after presentation showing intraretinal edema, hyperreflectivity, and thickening of the macula. (B) SD-OCT at two-year follow-up showing foveal scarring and atrophy. Spectral Domain Optical Coherence Tomography = SD-OCT.

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None.

Conflict of Interest

The authors report no financial interest or any conflict of interest.

Bibliography

1. Butler Nicholas J., *et al*. "Ocular toxoplasmosis II: clinical features, pathology and management". *Clinical and Experimental Ophthalmology* 41.1 (2013): 95-108.

2. Ryan Edward T., *et al*. "Toxoplasmosis". Hunter's Tropical Medicine and Emerging Infectious Diseases, Elsevier, New York, (2020): 95-108.

3. Tenter A M., *et al*. "Toxoplasma gondii: from animals to humans". *International Journal for Parasitology* 30.12-13 (2000): 1217-1258.

4. Holland Gary N. "Ocular toxoplasmosis: a global reassessment. Part I: epidemiology and course of disease". *American Journal of Ophthalmology* 136.6 (2003): 973-988.

5. Rai Mahendra and Kateryna Volodymyrivna Kon. "Chapter 15 - A Global Problem of Toxoplasmosi". The Microbiology of Central Nervous System Infections, Academic Press, (2018): 305-317.

6. Garweg Justus G and Uwe Pleyer. "Treatment Strategy in Human Ocular Toxoplasmosis: Why Antibiotics Have Failed". *Journal of Clinical Medicine* 10.5 (2021): 1090.