



## Toxoplasma Retinitis in Immunocompromised Host with Normal CD4 Count - A Case Report

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### Abstract

Toxoplasmosis is the most common opportunistic infection in patients with Human Immunodeficiency Virus (HIV) infection. A potentially blinding necrotizing retinitis can occur, with progressive deterioration of CD4+ T counts below 250 cells/cumm. This case highlights its unusual presentation in an HIV patient with normal CD4+ T counts. A 42-year-old gentleman presented with a week history of left eye diminution of vision. Clinical examination of the right eye was unremarkable while left eye showed anterior chamber and vitreous cells with a yellowish foveal lesion suspicious of toxoplasmic retinitis. He was seropositive for HIV, on therapy for ten years, with CD4+ T count 777 cells/cumm. Laboratory tests confirmed toxoplasmosis. He was started on oral anti-toxoplasma medication with tapering doses of oral and topical steroids. On subsequent follow-up, the patient had good visual and clinical improvement.

**Keywords:** Toxoplasmic retinitis; Human Immunodeficiency Virus; CD4+ T count

### Introduction

Toxoplasmosis is a zoonotic infection caused by the protozoal organism *Toxoplasma gondii*, an obligatory parasite of the cat. In an immunocompetent host, it is generally a benign self-limited infection; nevertheless, severe complications such as retinochoroidal involvement, have been known to occur. Ocular and Central nervous system toxoplasmosis is almost always seen in patients who are severely immunocompromised.

This case highlights the possibility of ocular toxoplasmosis in HIV positive individuals at normal CD4+ T counts.

### OPEN ACCESS

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Received Date: 01 Aug 2019

Accepted Date: 16 Aug 2019

Published Date: 21 Aug 2019

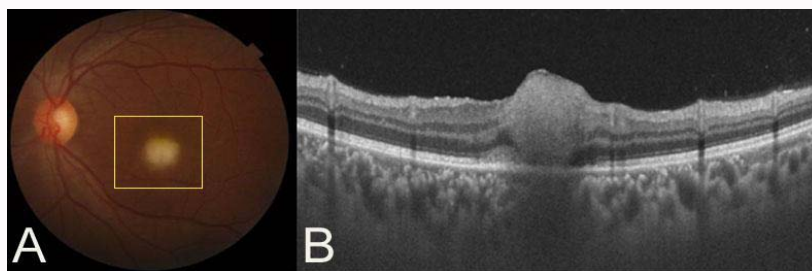
#### Citation:

Minija CK, Padmaja K, Dubey N. Toxoplasma Retinitis in Immunocompromised Host with Normal CD4 Count - A Case Report. Clin Case Rep Int. 2019; 3: 1116.

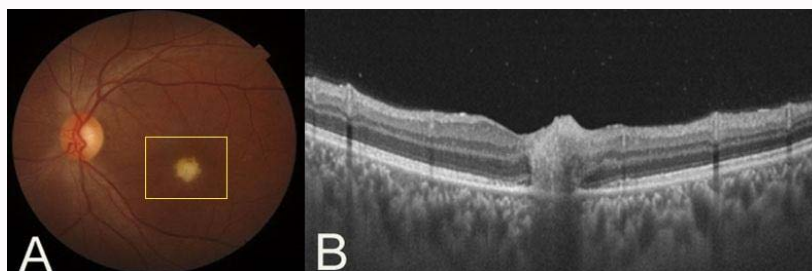
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### Case Presentation

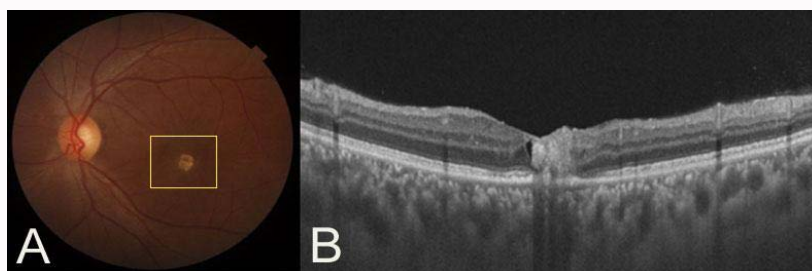
A 42-year-old gentleman presented with a history of diminution of vision of the left eye for a week. The Best-Corrected Visual Acuity (BCVA), recorded on Snellen's chart, was 6/6, N6 in the right eye and 6/36, N24 in the left eye. Clinically, the anterior segment examination of the right eye was normal, and the left eye showed anterior chamber cells of grade 1 (Standardized Uveitis Nomenclature grading: SUN). Posterior segment examination of the right eye was unremarkable, while the left eye revealed vitreous cells of grade 1+ (National eye institute system grading) with a dense yellowish lesion suggestive of retinitis, involving inferior half of fovea, suspicious of ocular toxoplasmosis (Figure 1A). Optical Coherence Tomography (OCT) of the left eye showed few hyper-reflective dots in the vitreous and loss of foveal contour with hyper-reflective full-thickness foveal lesion (Figure 1B). The patient was seropositive for HIV and on HAART medication for ten years with his recent (1 month since the time of presentation) CD4+ T count being 777 cells/cumm. Investigations revealed positive *Toxoplasma gondii* serology (IgG-453 IU/ml/IgM-9.4 IU/ml), positive Mantoux with induration of 23 mm. Systemic examination was normal. He was started on treatment with Tab. Septra DS (Trimethoprim 160 mg - sulfamethoxazole 800 mg) twice daily, along with Tab. Azithromycin 500 mg twice daily on day 1, followed by 500 mg once daily and topical prednisolone four times daily. On the follow-up visit after 3 days, consolidation of the lesion was noted, and oral wysolone 1 mg/kg body weight was added to the ongoing treatment. The consulting pulmonologist advised an additional INH prophylaxis. On the subsequent follow-ups, one week and one month later, the BCVA of left eye improved to 6/24, N8 and 6/9, N6 respectively. The posterior segment examination and OCT also showed a gross reduction in the size of the lesion in 2 visits (Figures 2A,2B and 3A,3B). During the course of disease he was treated with anti-toxoplasma medication for 8 weeks and a tapering dose of oral wysolone, 10 mg weekly taper for 7 weeks. Two months later, on completion of the course of treatment, BCVA of left eye had improved to 6/6,



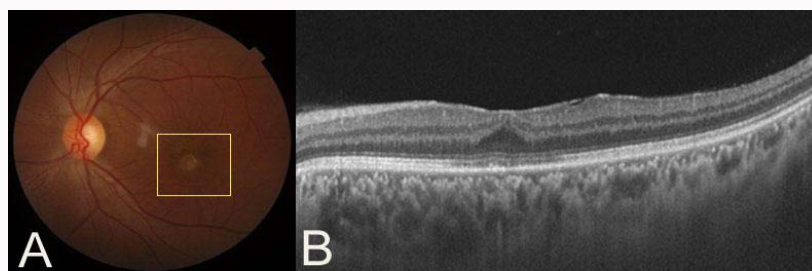
**Figure 1A, 1B:** Left eye showing yellowish lesion suggestive of retinitis involving inferior half of fovea. Swept-Source Optical Coherence Tomography (SS-OCT) showing vitreous cells and thickening of inner and outer layers of retina.



**Figure 2A, 2B:** Left eye showing shrink in size of retinitis lesion at 1 week. SS-OCT showing reduction in size of retinal thickening at 1 week.



**Figure 3A, 3B:** Left eye showing decrease in size of retinitis lesion at 1 month. SS-OCT showing reduction in vitreous cells and retinal thickening.



**Figure 4A, 4B:** Left eye showing gradual decrease in size of retinitis lesion with atrophic scar at 2 months. SS-OCT showing gradual reduction in retinal thickening with near normal foveal contour.

N6 and the inflammation had subsided completely, leaving behind an atrophic scar without pigmentation inferior to the fovea (Figure 4a). OCT showed improvement of foveal contour (Figure 4b).

**Discussion**

Approximately 70% to 80% of HIV-infected patients are going to be treated for associated eye disorder throughout the course of their disease. In general, the CD4+ T-lymphocyte count has been accustomed to predict the onset of bound ocular infections in patients who are seropositive. Patients with HIV infection are particularly susceptible to acute and reactive infections with *Toxoplasma gondii*, particularly when CD4+ T counts fall below 250 cells/cumm.

HIV patients with ocular toxoplasmosis must undergo a neurological evaluation; including imaging and lumbar puncture due to 30% to 50% of these patients have intracranial involvement. Toxoplasmic retinochoroiditis presents as areas of necrotizing retinitis, vitreous, and inflammation of the anterior segment, giving a classical picture of ‘headlight in the fog’ which usually resolves spontaneously, but often leaves pigmented chorioretinal scars [1]. The choroid and sclera may become involved secondarily [2]. In our case, however, the posterior segment manifestation in the left eye was in the form retinitis alone without the involvement of choroid. Following treatment, the lesion resolved to leave behind an atrophic scar without pigmentation.

Ocular toxoplasmosis in immunocompromised individuals frequently demonstrates a relatively fulminant course [3]. Potential presentations include multifocal disease in one eye, bilaterally active inflammation and/or extensive areas of necrotizing retinitis. Diagnostic confusion with viral retinitis may necessitate intraocular fluid analysis [4].

When the lesion is characteristic, the demonstration of IgG and IgM serum antibody gives a presumptive diagnosis and allows initiation of a specific therapy. In doubtful cases, it is possible to detect the parasite DNA by vitreous biopsy and PCR for *Toxoplasma gondii* [5].

Classical Triple regimen of anti-toxoplasma medications are given along with oral steroids to control inflammation. Steroids should be tapered and withdrawn a week prior to the completion of anti-toxoplasma medication. Intravitreal management option appears to be an effective treatment for immunocompetent patients, whereas systemic therapy is suggested in immunocompromised patients to prevent toxoplasmosis-related complications in the fellow eye or elsewhere in the CNS [6]. Prophylaxis with anti-toxic medications is effective against disseminated toxoplasmosis in immunocompromised patients when CD4+ T count less than 100 lymphocytes per cumm [7]. In our case, the eye was quite with normal CD4+ T count in the successive follow-ups without prophylactic treatment.

## Conclusion

Toxoplasmic retinochoroiditis need not necessarily manifest only in HIV positive individuals with low CD4+ T counts. It can

occur at normal CD4+ T counts as well. However, the severity of inflammation, systemic involvement, and need of prolonged anti-toxo therapy in the latter condition is less when compared to the former. Atypical presentation as toxoplasmic retinitis alone can occur without choroid involvement in HIV positive individuals.

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