# Acquired Immunodeficiency Syndrome Presented as Atypical Ocular Toxoplasmosis: a Case Report

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**Brief report**

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# Abstract

Background: The aim is to report an atypical presentation of ocular toxoplasmosis which led to the diagnosis of Acquired Immunodeficiency Syndrome (AIDS).

Case report: The 38-year-old woman was referred with metamorphopsia and reduced vision in the right eye in the past 3 weeks. Slit-lamp examination revealed granulomatous keratic precipitates (KPs), 2+ cells in the anterior chamber, and posterior synechiae. Fundus examination was remarkable for a white patch surrounding a scar, inferonasal to the optic disc with fibrous bands were emanating from the lesion and the retina around this region was detached with considerable extension towards periphery, while no breaks could be appreciated. She mentioned anorexia and losing 10 kg in the past three months and bilateral angular cheilitis was observed on systemic evaluation. The results of the patient's complete blood count revealed bicytopenia and CD4 lymphocytes: 79 cells/µl. Enzyme-linked immunosorbent assays (ELISA) for HIV antibodies came back positive which was later confirmed with the Western-blot test. Brain Magnetic Resonance Imaging (MRI) showed multiple ring-enhancing lesions in both cerebral cortices. Patient underwent Anti-Toxoplasmosis and Anti-HIV treatment and serous retinal detachment was completely resolved.

Conclusion: This report highlights the fact that sometimes the eyes are the site of the first presentation of a systemic life-threatening condition and emphasizes the role of ophthalmologists in such cases. In cases of atypical presentation, appropriate laboratory tests and CNS imaging should be requested. Systemic treatment with anti-toxoplasmosis regimens and HAART is mandatory in AIDs patients with ocular toxoplasmosis.


# Background:

Since the 1950s, ocular toxoplasmosis has been reported as the most common cause of infectious uveitis worldwide. Toxoplasma gondii is a ubiquitous obligate intracellular microorganism, which is estimated to affect one-third of the world’s population.[1–3]

Active ocular toxoplasmosis is characterized by a necrotizing retinochoroiditis lesion accompanying with localized or diffuse vitritis. In immunocompetent patients, lesions are usually self-limited and scarified within two months. Atypical forms of ocular toxoplasmosis such as diffuse outer retinitis, which can mimic acute retinal necrosis (ARN), punctuate outer retinal toxoplasmosis, occlusive retinal vasculitis, neuroretinitis, scleritis, and exudative retinal detachment have been reported.[4] Atypical lesions may occur bilaterally and have been reported in the immunocompromised, neonate and elderly patients.

This parasite can be fatal in immunocompromised individuals, such as HIV/AIDS patients especially those with CD4 T lymphocyte cell counts less than 200 cells/µL. In such a setting, reactivation of opportunistic organisms usually occurs with multiorgan involvement, especially neurological and ocular manifestations.

The central nervous system, as the most common site of Toxoplasma reactivation, can be involved in about 30% of patients with AIDS, especially in those who are treatment-naive and Toxoplasma seropositive with CD4 < 100 cells/μL. [5–8]

Here, we report an atypical presentation of ocular toxoplasmosis which led to the diagnosis of AIDS. The serous retinal detachment was resolved with anti-toxoplasmosis treatments and HAART.

# Case Report:

The 38-year-old woman was referred to the retina department of Farabi Eye Hospital with metamorphopsia and reduced vision in the right eye in the past 3 weeks. At the time of the presentation, bilateral angular cheilitis was observed (figure-1-A). Uncorrected visual acuity in the right eye was counting fingers at two meters. Media opacity and posterior synechia (PS) precluded refraction in this eye. Slit-lamp examination revealed granulomatous keratic precipitates (KPs) distributed in Arlt's triangle, 2 + cells in the anterior chamber, and a relatively broad-based PS causing a keyhole appearance in the pupil (figure-1B). The crystalline lens was clear and 2 + cells were present in anterior vitreous. Fundus examination was remarkable for a white patch surrounding a scar, inferonasal to the optic disc with the size of three-disc diameter. Some fibrous bands were emanating from the lesion and the retina around this region was detached with considerable extension towards the superior, nasal, and inferior periphery, while no breaks could be appreciated (figure-1C). The vision in the left eye was 20/20 and ophthalmic examination was unremarkable in this eye.

Spectral-domain optical coherence tomography (SD-OCT) (Spectralis Heidelburg Germany) disclosed evidence of vitritis, a detached posterior hyaloid face, and an internal limiting membrane (ILM) wrinkling, a fine epiretinal membrane (ERM) nasal to the fovea in the right eye. (figure-1D). Scanning the lesion from superior through inferior failed to show any break responsible for retinal detachment.

On further questioning, she mentioned anorexia and losing 10 kg in the past three months. She denied using any medications or having other illnesses. Considering systemic symptoms and signs and due to involuntary weight loss, the patient underwent a comprehensive infectious, neoplastic, and rheumatologic workup. The results of the patient's complete blood count (CBC) are summarized in Table 1. Based on the ocular findings, additional tests were ordered for Toxoplasma gondii antibodies which revealed the presence of IgG antibodies while IgMs were absent.

Table 1
The results of the patient's CBC-diff

| Blood component | | Reference range |
|---|---|---|
| White blood cells | 3400 | 4500-11,000/$mm^3$ |
| Red blood cells | 4.19 | Male: 4.3–5.9 million/$mm^3$<br>Female: 3.5–5.5 million/$mm^3$ |
| Hemoglobin | 8.1 | Male: 13.5–17.5 g/dL<br>Female: 12.0–16.0 g/dL |
| Hematocrit | 26.8 | Male: 41%-53%<br>Female: 36%-46% |
| Mean corpuscular volume | 64.0 | 80–100 $\mu m^3$ |
| Mean corpuscular hemoglobin | 19.3 | 25.4–34.6 pg/cell |
| Mean corpuscular hemoglobin concentration | 30.2 | 31%-36% Hb/cell |
| Platelets | 236000 | 150,000-400,000/$mm^3$ |
| Neutrophil | 61.7 | 40–60 % |
| Lymphocytes | 27.9 | 20–40 % |
| Monocytes | 7.8 | 4–8 % |
| Eosinophil | 2.6 | 1–3 % |
| Basophil | 0 | 0–1 % |
| CD4 lymphocytes | 79 cells/µl | 500–1400 cells/µl |

Imaging studies including chest-X-Ray, abdominal sonography, and age-related neoplastic workup including mammography, pap smear, and colonoscopy were unremarkable except for non-specific polyps in the patient's large intestine.

Enzyme-linked immunosorbent assays (ELISA) for HIV antibodies came back positive which was later confirmed with the Western-blot test.

Once the diagnosis of toxoplasmic retinochoroiditis in an immunosuppressed patient was established, treatment with Trimethoprim/sulfamethoxazole (TMP/SMX) 960 mg tablets twice daily was commenced. Brain Magnetic Resonance Imaging (MRI) showed multiple ring-enhancing lesions in both cerebral cortices. (Fig. 2). Meanwhile, the patient developed a right hemiparesis.

Highly active antiretroviral therapy (HAART) was added to her anti-toxoplasmosis treatment and dose of Trimethoprim/sulfamethoxazole (TMP/SMX) increased to 960 mg /three times a day and Azithromycin 250mg/daily was added to antitoxoplasmic regimen. During the follow-up visits, vitreous inflammation decreased substantially, subretinal fluid gradually resolved and motor deficit improved. Yellow subretinal hard exudates appeared in the perifoveal area with complete reattachment of the retina in three months.

## Discussion And Conclusions:

The presence of cells in the anterior chamber and vitreous cavity, large Kps, PS, and a patch of retinitis adjacent to a pigmented retinochoroidal scar are clinical clues to the diagnosis of toxoplasmic retinochoroiditis. Positive Toxoplasma IgG antibody along with negative IgM indicates infection with the organism at some time.[2, 9, 10] However, this case shows some ocular and systemic features that are not commonly expected in the typical case of toxoplasmic retinochoroiditis. In this case, pursuing these atypical features, led us to the diagnosis of AIDS. The first red flag was the presence of an extensive retinal detachment. Although tractional, exudative and rhegmatogenous retinal detachments can occur in ocular toxoplasmosis, such reports are limited; the reported prevalence of retinal detachment in acquired toxoplasmosis varies between 2.5 to 11 percent which is mostly of tractional or rhegmatogenous type. [10–13]

Angular cheilitis and significant weight loss, are not expected to occur in typical ocular toxoplasmosis. The presence of angular cheilitis calls for investigating the possibility of anemia and its underlying etiology. In our case, CBC revealed bicytopenia (anemia and leukopenia) which necessitates a thorough investigation for immunodeficiency.

Increased energy expenditure happens in the setting of opportunistic infections. Weight loss in patients with HIV is associated with poor prognosis, increased risk of disease progression and opportunistic pathology.[14]

In a study by Neves et al on 37 immunocompetent patients with acute acquired toxoplasmosis the frequency of systemic manifestations was inspected. Weight loss, anemia, and leukopenia were seen in 62.2%, 10.8% 16.2% of patients, respectively.[9]

The possibility of toxoplasmic encephalitis should be investigated in every patient with HIV and retinochoroidal toxoplasmosis. The study of choice for this purpose is brain MRI with contrast that can illustrate characteristic enhancing ring lesions. While not pathognomonic, the presence of several brain abscesses is the most characteristic feature of T. gondii infection in AIDS patients. Autopsy usually shows a global involvement of both hemispheres, though the basal ganglia and the corticomedullary junction are the most common sites of involvement.[6–8]

There are several reports on the management of retinal detachment in the setting of ocular toxoplasmosis. Among the 193 patients with ocular toxoplasmosis, Kianersi et al reported five patients with RD: three rhegmatogenous and two tractional. All five patients underwent vitrectomy or scleral

buckling surgery. It is well known that severe intraocular inflammation can lead to tractional retinal detachment. With the progression of traction, a retinal hole and subsequent rhegmatogenous detachment may develop. Another possible mechanism of hole formation is the occurrence of full-thickness necrosis in the retinitis patch. [13]

In another study, Al-Zahrani et al reported a 24-year-old man with exudative RD secondary to reactivation of a toxoplasmosis lesion, which was successfully managed with anti-toxoplasmosis treatment.[11]

In a study by Faridi et al, on thirty-five eyes of 28 patients with ocular toxoplasmosis and sufficient follow-up, they identified 4 eyes (11 percent) with secondary RD, which was either rhegmatogenous, tractional, or a combination of the two. All eyes with retinal detachment underwent surgical repair of which 50% developed recurrent RDs (two of 4 patients). They concluded that handling ocular toxoplasmosis and related RDs may necessitate several measures, including surgical management, systemic therapy, and intravitreal injections. formation of new breaks or PVR-related complications may necessitate multiple surgeries in cases with recurrent RD.[12] Our case represents the exudative type of detachment with no distinct retinal break. Observation of a retinal fold may advocate the presence of a tractional component in this case. The resolution of RD following an anti-toxoplasmic regimen and HAART and without surgical intervention may suggest that decision upon surgery should not be rushed in such patients and a period close followup visits for ocular examination combined with appropriate systemic treatment may be warranted.

This report highlights the fact that sometimes the eyes are the site of the first presentation of a systemic life-threatening condition and emphasizes the role of ophthalmologists in such cases. Judicious ocular and systemic evaluation of patients with ocular toxoplasmosis are of utmost importance. In cases of atypical presentation, appropriate laboratory tests and CNS imaging should be requested. Systemic treatment with anti-toxoplasmosis regimens and HAART is mandatory in AIDs patients with ocular toxoplasmosis. Treatment options for retinal detachment in this setting should be meticulously approached.

# Abbreviations:

- AIDS: acquired immune deficiency syndrome
- KP: Keratic Precipitate
- ELISA: enzyme-linked immunosorbent assay
- HIV: human immunodeficiency viruses
- MRI: Magnetic resonance imaging
- CNS: Central Nervous System
- HAART: highly active antiretroviral therapy
- OCT: Optical Coherence Tomography

# Declarations:

### Authors Contributions

All authors managed the patient clinically. E.K. and Z.M. wrote the manuscript. F.G and N.E and H.R revised draft. All authors discussed the results and approved the final manuscript.

### Availability of data and materials

The data generated during the present study is available upon request from the corresponding author.


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### Ethics declarations

### Ethics approval and consent to participate

Not applicable. The authors declare that they adhered to the CARE guidelines/methodology.

### Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

### Competing interests

The authors declare that they have no competing interests.

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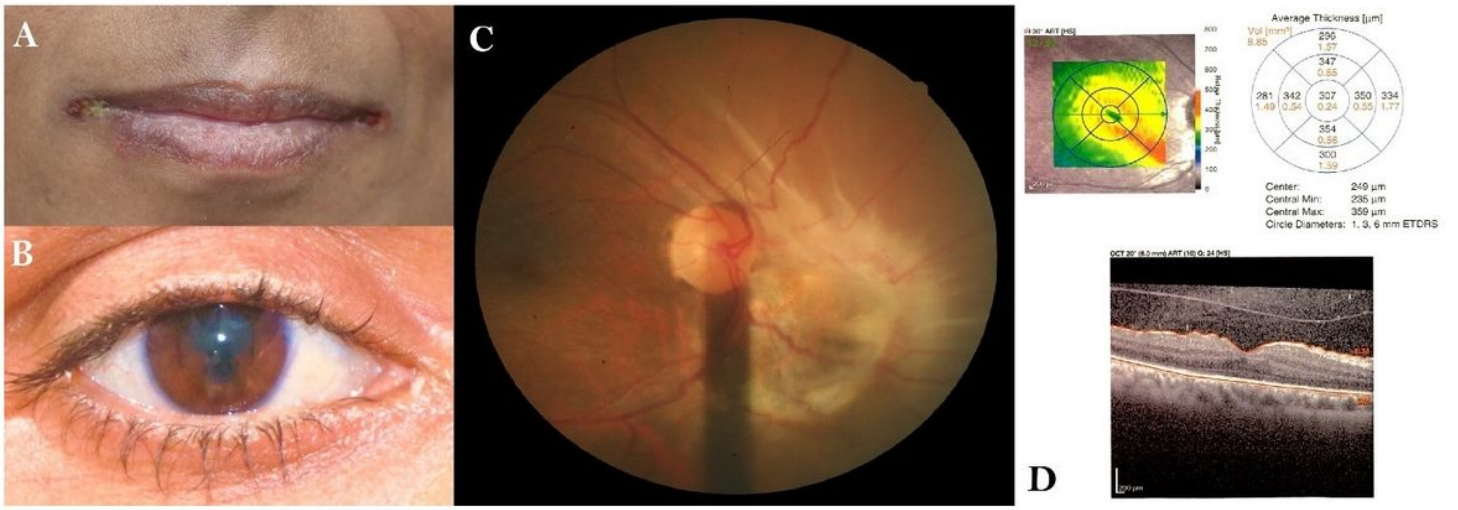
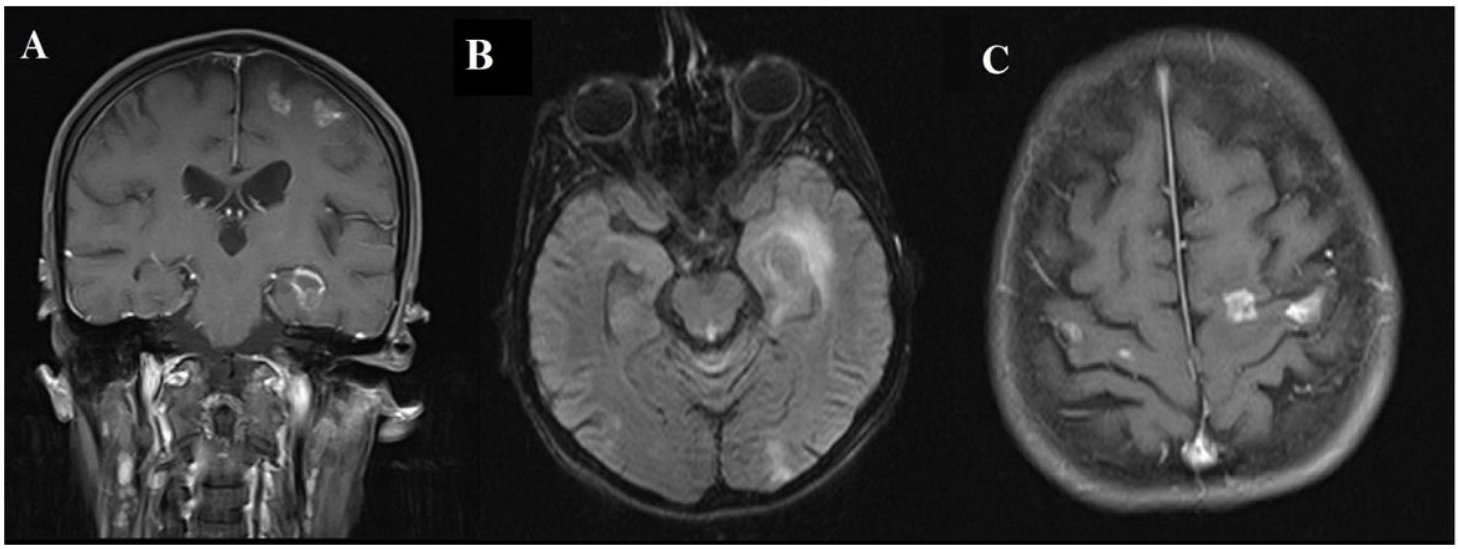
# Figures



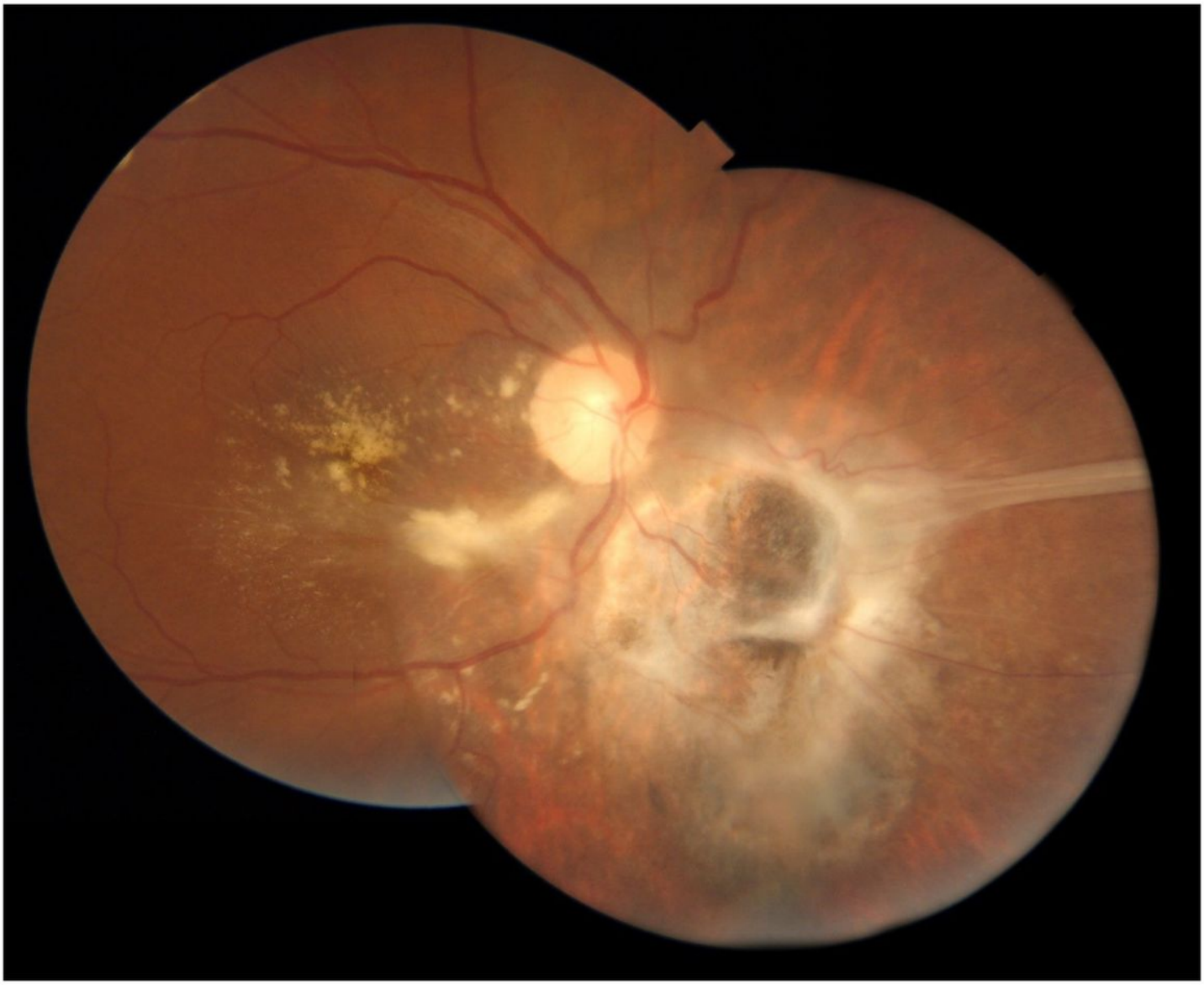

## Figure 1

Clinical features of the patient at the time of presentation. Bilateral angular cheilitis (A), posterior synechiae, and keyhole-shaped pupil (B), In the right eye inferonasal to the optic disc an annular white patch centered with a pigmented scar is seen. White subretinal bands are extending from the lesion. The size of the lesion is about three-disc diameter and adjacent retina is detached (C). Optical Coherence Tomography of the right eye demonstrates evidence of vitritis, detached posterior hyaloid, internal limiting membrane wrinkling, and a fine epiretinal membrane nasal to the fovea. (D)




## Figure 2

Magnetic resonance imaging (MRI): T1 with contrast in coronal (A) and axial planes (B and C): multiple ring-enhancing lesions in the left and right cerebral cortices.



## Figure 3

Montage fundus photography of the patient's right eye three months after initiation of anti-toxoplasmosis treatment and HAART, shows complete retinal reattachment. A retinal fold extends from the lesion to the nasal periphery. Subretinal exudates are seen in the inferior area of the disc and around the macula.