Diffuse toxoplasmic retinochoroiditis as an initial manifestation of AIDS.

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Abstract

Ocular toxoplasmosis commonly presents with typical clinical features of focal retinitis patch over the posterior pole, often at the border of a preexisting retinochoroidal scar, with an overlying vitreous haze. Although the disease is known to present with atypical clinical features like papillitis, neuro-retinitis, diffuse necrotizing retinochoroiditis etc. in patients with Acquired Immunodeficiency Syndrome (AIDS), it is very rare for such features to present as an initial manifestation of AIDS. Such atypical clinical features also pose a great diagnostic challenge, especially when the immune status of the patient is not known. We hereby report a case where diffuse toxoplasmic retinochoroiditis presented as an initial manifestation of AIDS. The patient was initially diagnosed to have viral retinitis but polymerase chain reaction from anterior chamber tap confirmed toxoplasmosis. Serum ELISA was positive for HIV 1 with a viral load of 22,067 copies/ml. The patient was successfully treated with systemic and intravitreal anti-toxoplasma medication along with Highly Active Anti-Retroviral Therapy (HAART). The importance of early diagnosis of HIV with early initiation of HAART cannot be understated. We recommend that *Toxoplasma* infection should be kept in the differential diagnosis of diffuse retinochoroiditis. We also recommend HIV screening of adults with newly diagnosed ocular toxoplasmosis.

Keywords: Toxoplasmosis, Retinochoroiditis, HIV, AIDS, Atypical.

Abbreviations: AIDS: Acquired Immuno Deficiency Syndrome; ELISA: Enzyme-Linked Immunosorbent Assay; HIV: Human Immunodeficiency Virus; HAART: Highly Active Anti-Retroviral Therapy; PCR: Polymerase Chain Reaction; HSV: Herpes Simplex Virus; CMV: Cytomegalovirus; VZV: Varicella Zoster Virus; SUN: Standardization of Uveitis Nomenclature; VDRL: Venereal Disease Research Laboratory; AC: Anterior Chamber; HBV: Hepatitis B Virus; HCV: Hepatitis C Virus; ARN: Acute Retinal Necrosis; PORN: Progressive Outer Retinal Necrosis; TRC: Toxoplasmic Retinochoroiditis

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Introduction

Toxoplasmic retinochoroiditis is the most common cause of posterior uveitis in immunocompetent individuals [1-3]. The infection is caused by the protozoa, Toxoplasma gondii and is transmitted via ingestion of undercooked meat, contaminated water, and trans-placentally [4]. The disease is known to be more aggressive in immunocompromised patients and often presents with atypical clinical features. Toxoplasmic papillitis, neuro-retinitis, pars planitis, retinal necrosis, scleritis, periarteritis and vascular occlusions with bilateral or multifocal involvement have all been reported in patients with AIDS [4-7]. Although atypical features are known to occur in immunocompromised individuals, it is very rare for such ocular features of toxoplasmosis to present as the initial manifestation of AIDS [8]. We hereby report a case of diffuse toxoplasmic retinochoroiditis presenting as the initial manifestation of acquired immunodeficiency syndrome. To the best of our knowledge, such a presentation has not been reported from the Indian sub-continent.

Case Reports

A 57-year-old, Indian female presented to the Uvea clinic of a tertiary care eye hospital with chief complaints of sudden onset, painless, progressive diminution of vision in her right eye for the past 3 weeks. The patient was diagnosed to have viral retinitis elsewhere and was started on oral antiviral drugs along with oral steroids. Her serum Polymerase Chain Reaction (PCR) was negative for Herpes Simplex Virus (HSV) I and II, Cytomegalovirus (CMV) and Varicella-Zoster Virus (VZV). The patient was not responding to the treatment and hence was referred for further management. The patient was a known case of systemic hypertension and type II diabetes mellitus, which were under control with medications. On examination, the patient was well built and well-nourished. Visual acuity in the right eye was 6/48 for distance and N24 for a near while in the left eye it was 6/6 and N6 on Snellen Chart. Slit-lamp examination of the right eye revealed fine keratic precipitates, anterior chamber cells and flare 1+ with a clear lens and 1+ cells in the anterior vitreous (SUN Classification). On fundus examination by indirect ophthalmoscope, there was grade I vitreous haze, the optic disc was hyperemic and edematous, a diffuse yellowish-white retinal lesion was noted from 1 to 5

o'clock meridian extending from the optic disc up to the periphery from 1 to 3 o'clock and up to the equator from 3 to 5 o'clock. Similar retinal lesions were also noted from 9 to 11 o'clock meridian near the arcade with a small lesion over the macula. Sparse retinal hemorrhages were noted along with the lesions (Figure 1a). The left eye anterior and posterior segment examination was unremarkable. A presumptive diagnosis of viral retinitis was made and the patient was started on intravenous acyclovir therapy (500 mg 3 times a day) along with oral steroids (1 mg/kg). Anterior Chamber (AC) tap was performed for PCR for HSV I and II, CMV and VZV. PCR from the AC tap report came negative for these viruses. The patient was kept under close observation. As the lesions were not responding, the patient was investigated further for serum HIV, IgG, and IgM for toxoplasmosis and VDRL. Serum Elisa for HIV I and IgG for toxoplasma came positive, while serum VDRL was negative. The previous AC tap sample was then subjected to PCR for toxoplasmosis, which came out positive (Figure 1c).

A diagnosis of diffuse toxoplasmic retinochoroiditis with AIDS was made. The patient was referred to a physician for complete systemic evaluation and to rule out other opportunistic infections. Her presenting CD4 cell count was 47 and the viral load was 22,067 copies/ml. She was negative for HBV and HCV. Her MRI brain showed cortical atrophic changes only; there were no signs of encephalitis. The thorough systemic evaluation was done and all other opportunistic infections were ruled out. The patient was started on oral clindamycin 300 mg 4 times a day with trimethoprim and sulfamethoxazole (160/800) combination 2 times per day along with HAART (Tenofovir+Lamivudine+Efavirenz). She was also treated with intravitreal clindamycin (1 mg in 0.1 ml) +dexamethasone (400 µg). Intravitreal injections were given twice at 2 weeks interval. The patient subsequently reported to the clinic 1 month after the initiation of therapy with near-total resolution of retinochoroiditis with early scarring of the lesions (Figure 1b). Her CD 4 count also improved to 199 cells/ml. She was advised to continue systemic anti-toxoplasma medication along with HAART and to review after 1 month.

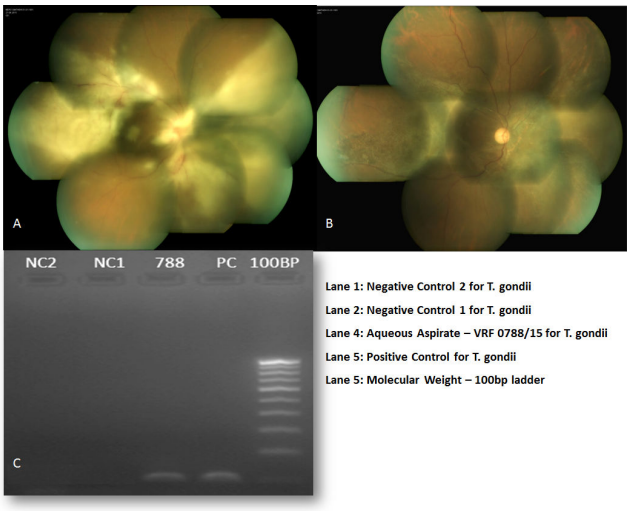

Figure 1. (a): *Fundus photo montage showing disc edema with diffuse retinochoroiditis patch nasally and a large patch temporally along with sparse retinal hemorrhages and vasculitis;* ***(b):*** *Fundus photo montage after 1 month of therapy showing complete resolution of disc edema with healed retinochoroiditis patches;* ***(c):*** *Positive Polymerase chain reaction report for Toxoplasma gondii from aqueous tap.*

Discussion

Toxoplasma gondii affects nearly 10% of patients with AIDS. Central nervous system manifestation is seen in nearly 55% of the cases, while toxoplasmic retinochoroiditis is a rare manifestation of the disease seen in about 2%-8% [8]. Ocular opportunistic infections seen in AIDS include CMV retinitis, HSV I and II retinitis, ARN and PORN due to HSV and VZV and toxoplasmic retinochoroiditis. TRC in AIDS can have atypical clinical presentations. Typical TRC presents as solitary or multifocal retinitis lesions located posteriorly and measuring 2-3 disc diameter with intense overlying vitreous haze giving the classical appearance of 'headlight's in fog'. In patients with AIDS, the disease more often presents as diffuse necrotizing retinal lesions with a lack of intense vitreous inflammation mimicking viral retinitis. Other atypical presentations include neuro-retinitis, hemorrhagic vasculitis and serous retinal detachment [9,10].

In the present case, the patient presented with diffuse necrotizing retinal lesion due to toxoplasma with sparse hemorrhages mimicking ARN. Previously only 4 cases have been reported to present with diffuse toxoplasmic necrotizing retinochoroiditis, all of them in immunocompromised states. In our case, the immune status of the patient was unknown and the patient was quite healthy otherwise hence we did not suspect TRC initially.

Conclusion

This case highlights that TRC can be present as an initial manifestation of AIDS without any other systemic opportunistic infections. We recommend that toxoplasmosis should be kept as an important differential in all cases presenting with diffuse retinitis and all such patients should be screened for HIV.

Declaration

Ethical approval

Ethics approval and consent to participate: not applicable.

Consent for publication

Consent to publish the case report has been taken from the patient concerned and does not disclose the identity or infringe the privacy of the patient.

Availability of data and material

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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