

Ocular toxoplasmosis associated with scleritis

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We report an atypical presentation of *Toxoplasma* retinochoroiditis with associated scleritis in a young and immunocompetent patient. The diagnosis was done on the basis of Polymerase chain reaction of vitreous sample, and the clinical response to specific treatment. This case highlights the unusual presentation of ocular toxoplasmosis as scleritis.

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Ocular toxoplasmosis presents as a localized retinochoroiditis in typical cases.^[1] Atypical presentations include variations in the pattern of presentation in the same structure, such as isolated retinal vasculitis, multifocal or outer retinal involvement, or involvement of adjacent structures, such as the optic nerve.

Such atypical presentations are usually seen in elderly or immunocompromised individuals.^[1]

We report a case of scleritis along with retinochoroiditis in a young, immunocompetent woman, as an atypical presentation of Toxoplasmosis.

Case Report

A 26-year-old woman presented with complaints of sudden onset of severe pain, redness, and defective vision in her right eye since 10 days. There was no history of trauma or any prior symptoms in the affected eye. There was no history suggestive of any other systemic illness. She denied any history of intravenous or other drug use.

She gave history of being treated with oral medications, for decreased vision in her left eye, 5 years earlier.

Her best corrected visual acuity was 20/200 in OD and 20/20 in OS. Intraocular pressure was normal in both eyes. Anterior segment examination of her right eye showed a diffuse non-necrotizing scleritis, localized inferiorly [Fig. 1]. There were few fresh granulomatous keratic precipitates on the

inferior cornea and 2+ cells and flare in the anterior chamber. Lens was clear. The anterior vitreous had 3+ cells. Anterior segment examination of the left eye was within normal limits.

Ocular fundus examination of the right eye revealed a 3+ vitreous haze suggestive of intense vitritis. Through the haze the disc was barely seen, but the characteristic "Headlight in Fog" appearance of macular retinochoroiditis was lacking. The inferior periphery, however, showed a circumferentially oriented large yellow lesion involving the inferonasal quadrant, with the posterior extent till the equator. Another similar lesion was noted in the superonasal periphery, discontinuous with the main lesion. Her left ocular fundus showed multiple confluent pigmented chorioretinal scars, starting in the periphery of the nasal to the inferotemporal quadrants, with tongue-shaped extensions posteriorly, beyond the equator. There were two round scars around one disc diameter in size above the superotemporal arcade [Figs. 2 and 3].

B scan Ultrasonography of right eye revealed associated posterior scleritis, but without any evidence of retinal detachment [Fig. 4].

In view of intense vitritis with confluent peripheral retinitis in one eye of an otherwise healthy woman with similar location of scars in the other eye, a provisional diagnosis of acute retinal necrosis with scleritis was made. A vitreous tap was performed under aseptic precautions through the inferotemporal quadrant, avoiding the area of scleritis. She was empirically started on oral valacyclovir in the recommended dose. Topical Dexamethasone drops were started 6th hourly along with Homatropine eye drops instilled 12th hourly in the right eye. Oral steroids were added after 48 h of starting oral antivirals. The complete blood picture was normal; a purified protein derivative (PPD) test dose given on presentation was negative. Her chest X-ray was normal and systemic examination by a physician did not reveal any evidence of systemic tuberculosis or Diabetes mellitus.

Serology for syphilis and Human immunodeficiency virus were negative.

The vitreous sample subjected to multiplex polymerase chain reaction (PCR) test, revealed the presence of *Toxoplasma*. The sample was negative for Herpes simplex virus, Herpes zoster virus, and *Mycobacterium tuberculosis*. She was started



Figure 1: Inferior scleritis

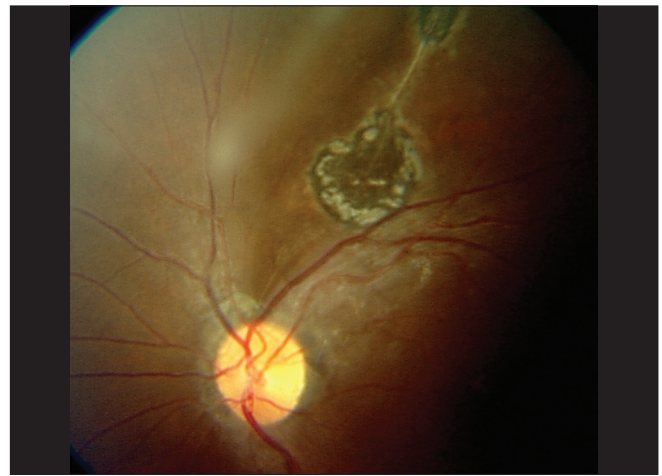


Figure 2: Fundus photo (OS) depicting chorioretinal scars above superotemporal arcade

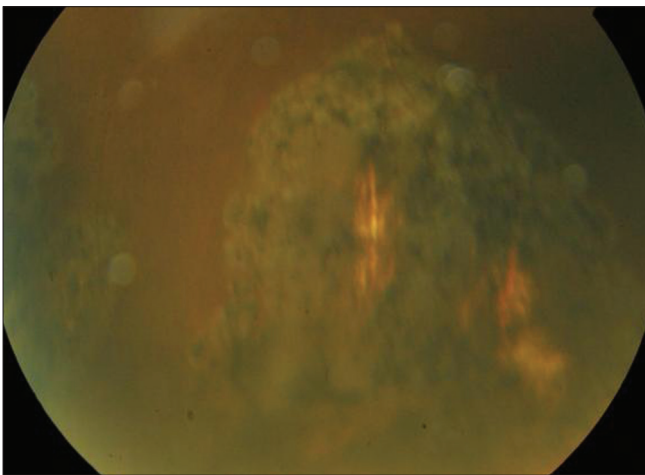


Figure 3: Fundus photo (OS) depicting inferior peripheral chorioretinal scars

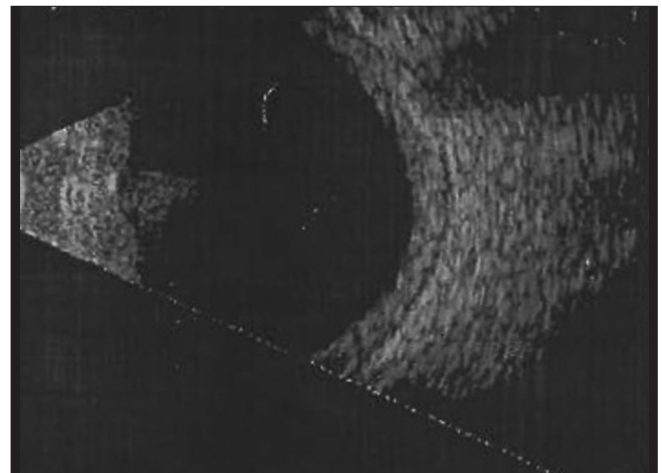


Figure 4: B scan (OD) depicting posterior scleritis

on oral trimethoprim/sulfamethoxazole (160 mg/800 mg b.i.d.) and Azithromycin (500 mg o.d.), along with oral steroids in the recommended doses. The antiviral, which had shown no response for 3 days, was stopped.

On review after a week, she was tolerating her treatment well and showing signs of improvement. At the end of 3 weeks, her visual acuity had improved to 20/30 in the affected eye.

The scleritis had completely resolved, and the areas of peripheral retinochoroiditis were scarring with pigmentation. She completed the course of antitoxoplasma treatment, and is currently asymptomatic.

Discussion

Ocular toxoplasmosis is the most common identifiable cause of posterior uveitis in the world.^[2] It typically presents as a macular or peripheral unifocal retinochoroiditis, which may be adjacent to an old scar of a congenital or previous infection. Atypical presentations are seen in immunosuppressed or elderly individuals. They include punctuate outer retinal toxoplasmosis, retinal vasculitis, retinal vascular occlusions, serous or rhegmatogenous retinal detachments, retinitis pigmentosa resembling retinopathy, and neuroretinitis.^[1]

However, in our young, immunocompetent patient, the presence of peripherally located confluent, circumferentially oriented lesions with intense vitritis closely resembled acute retinal necrosis, which has a poor visual prognosis if not promptly treated. She also presented with scleritis, which has been reported in association with acute retinal necrosis.^[3] Tuberculosis was also considered as a differential diagnosis, as we reside in an endemic area, and the pigmented choroiditis scars in the other eye could be attributed to previous infection. Infectious scleritis is commonly associated with tuberculosis and Herpes zoster infection, among others.^[4] Both of these agents could have resulted in a similar presentation as seen in our case. However, PCR analysis, negative PPD test, and clinical response to specific treatment, favored Toxoplasmosis.

In their case series, Schuman *et al.* reported only 2 of the 5 patients with clinical toxoplasmic scleritis to be young and immunocompetent.^[5] Histopathological evaluation in their other 3 cases revealed the presence of a layer of uninflamed sclera in between the superficial zone of scleritis, and deeper zone adjacent to the toxoplasma retinochoroiditis lesions.^[5] This questioned the earlier theory of reactionary granulomatous inflammation of the sclera; solely due to adjacent retinochoroiditis.

Hence clinicians dealing with retinochoroiditis with scleritis should have toxoplasmosis as a differential in their etiological diagnosis. In atypical cases, such as ours, ocular fluid analysis would help in early identification of the causative agent against which treatment may be directed. The role of PCR in the diagnosis of atypical presentation of toxoplasmosis, along with specific antibody titers from ocular fluids is well established.^[6] Enzyme-linked immunosorbent assay (ELISA) is used to detect anti-toxoplasma IgG in serum as well as in ocular fluid. A rising titer of antibodies is diagnostic in serum. The relative increase in local specific IgG in ocular fluid compared to antibodies in serum can be calculated using the Goldmann – Witmer coefficient (GWC). The combination of GWC with PCR is reported to be more sensitive in diagnosing ocular Toxoplasmosis.^[7]

This case highlights the unusual manifestation of scleritis in Toxoplasmosis, which is a common cause of infectious uveitis, as well as the role of PCR in the diagnosis of atypical retinochoroiditis.

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