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Case Report

Ocular toxoplasmosis related macular traction: A case report and review of the literature

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Abstract

This is a case of toxoplasmosis retinochoroiditis which has resulted in the formation of vitreomacular traction upon resolution which is rarely associated with ocular toxoplasmosis. A 39-year-old male came with an active toxoplasmosis retinochoroiditis. Best-corrected visual acuity, full ophthalmic slitlamp examination, colour fundus photography, spectral domain optical coherence tomography (SD-OCT), and fluorescein angiography were performed. Presumed ocular toxoplasmosis diagnosis was supported by serological tests. The patient was treated medically for 45 days and on his follow up he developed macular traction which was shown in SD-OCT with a good visual acuity. Vitreoretinal traction is a rare complication of ocular toxoplasmosis and ranges from mild to severe traction which might require surgery. We suggest a close follow up for patients with toxoplasmosis retinochoroiditis and early recognition could avoid exposing patients to surgery.

Keywords: Ocular toxoplasmosis, Toxoplasma retinochoroiditis, Vitreomacular traction

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Introduction

Ocular toxoplasmosis is the most common cause of posterior uveitis worldwide[1] and one of the most common causes of uveitis in Saudi Arabia.[2] The most common ocular manifestation is retinochoroiditis, frequently localized at the posterior pole and may involve the macula[3,4] or come with vitritis. Resolution of active lesions results in hyperpigmented chorioretinal scars with areas of chorioretinal atrophy. Spectral Domain Optical Coherence Tomography (SD-OCT) of active toxoplasmosis lesions show increased retinal reflectivity and RPE-choriocapillaris/choroidal optical shadowing, while toxoplasmosis scars show disorganised retinal signalling and retinal thinning, as well as focal choriocapillaris/choroidal relative hyperreflectivity.[5–7] Uncommon presentations of ocular toxoplasmosis include punctate outer retinal toxoplasmosis, retinal vasculitis, retinal vascular occlusions, rhegmatogenous and serous retinal detachments, a unilateral pigmentary retinopathy mimicking retinitis pigmentosa, neuroretinitis, optic neuritis and scleritis.[8–10] We report an unusual case of toxoplasma retinochoroiditis which has resulted in vitreomacular traction on follow up visits.

Case report

A 39-year-old male presented to the Emergency Room complaining of progressive blurring of vision in the right eye. The patient underwent best-corrected visual acuity

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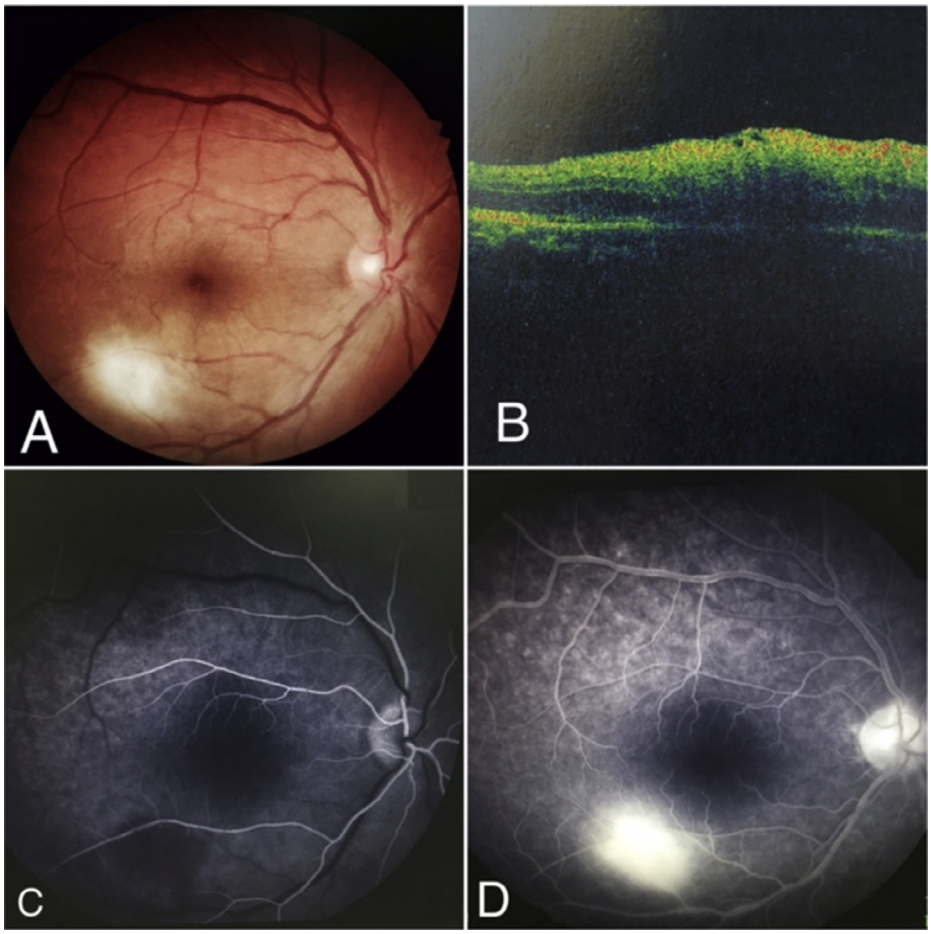

Fig. 1. (A) shows presumed ocular toxoplasmosis-related retinitis inferotemporal to the macula on fundus examination. (B) shows SD-OCT with accomulation of subretinal fluid under the chorioretinitis lesion. (C) shows fundus fluorescine angiography with early hypofluoresence at the chorioretinal lesion site. (D) shows fundus fluorescine angiography with late leakage at the site of chorioretinal lesion.

testing, full ophthalmic slit-lamp examination of the anterior and posterior segments, (SD-OCT), and fluorescein angiography. Visual acuity was 20/22 in the right eye and 20/20 in the left eye, without correction. Anterior segment examination in both eyes was unremarkable and intraocular pressure was 18 mmHg. Fundus examination of the right eye showed 1 disc diameter whitish yellow inflammatory lesion inferotemporal to the macula not involving the fovea with focal vitritis. The fundus examination of the left eye was normal. SD-OCT in the right eye showed subertinal fluid and retinal thichening indicating an active retinochoroiditis. Fluorescein angiography showed early hypofluorescence of the lesion and involved vessels with progressive hyperfluorescence due to leakage of the dye in addition to leakage from the involved blood vessels indicating vasculitis (Fig. 1). A serologic search for Toxoplasma gondii infection was positive for immunoglobulin G (133 UI/mL, reference value, 10 UI/mL), whereas immunoglobulin M levels were normal, indicating prior exposure to Toxoplasma. A syphilis screen and other tests, including for tuberculosis, toxocariasis, sarcoidosis and autoimmune diseases were all negative. A presumptive diagnosis of toxoplasma retinochoroiditis was made, and a regimen of spiramycin 1gm 3 times daily was started and planned for 6 weeks. Gradual reduction of the retinal edema was noted after 2 weeks of presentation and the patient was started on oral prednsolone 1 mg/kg/day with weekly tapering dose. 2 weeks later (1 month from presentation), mild reduction of visaul acuity in the left eye to 20/28 was noted and on fundoscopic examination, there was flattening and pigmentation of the lesion which indicate scarring, with the development superficial retinal folds involving the macula indicating traction at superficial macular layers. SD-OCT showed Vitreoretinal traction involving the superficial layers of the macula (Fig. 2). Patient was being closely followed up to detect any progression of traction. 2 months after complete resolution, the patient has developed a chorioretinitis lesion on the border of the previous scar which and on fundus fluorescine angiography it has shown a characteristic early hypofluoresence with late leakage (Fig. 3) indicating a toxoplasmosis satellite chorioretinal lesion for which another cycle of spiramycin has started for 45 days.

Discussion

In the case reported here, the primary ocular toxoplasmosis infection was treated promptly with with resolution of retinochoroiditis, chorioretinal scar formation and vitreo-

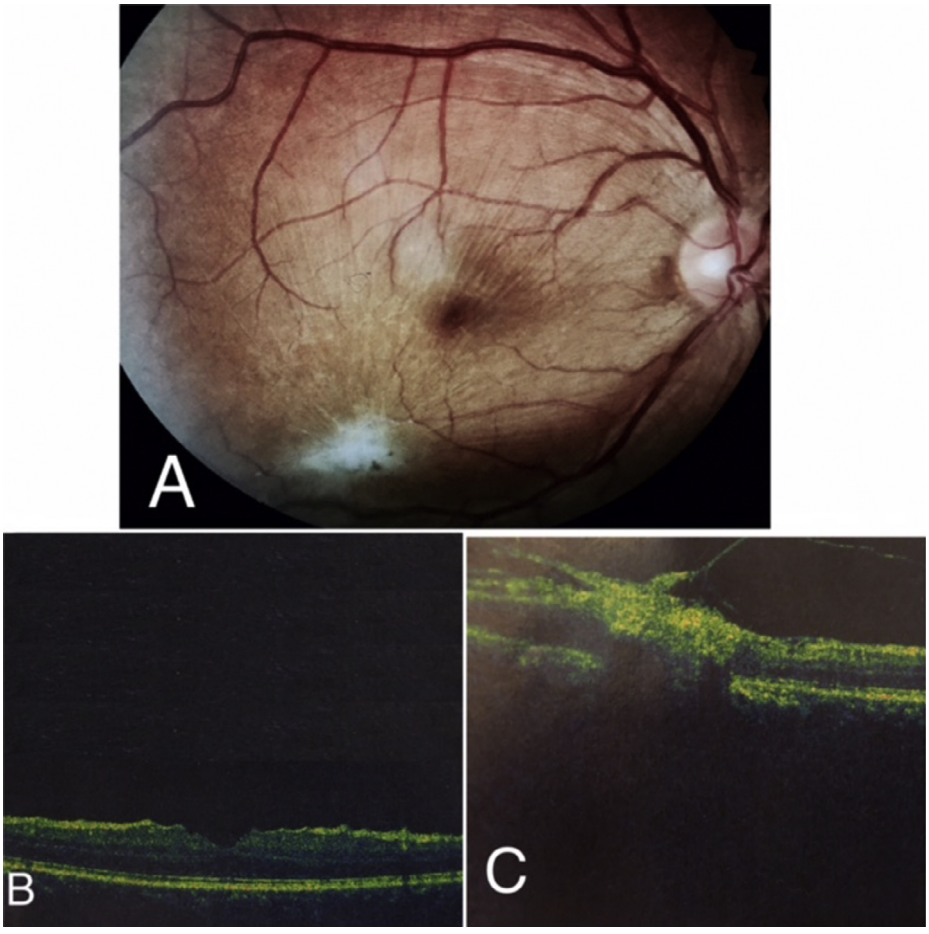

Fig. 2. (A) shows scarring of the chorioretinal lesion upon resolution with the formation of vitreomacular traction. (B) shows SD-OCT of the macula with superficial traction involving the fovea. (C) shows SD-OCT with vitreoretinal traction at the site of scar.

macular traction. This complication probably happened as a consequence of vitreous inflammation.[11] Vitreomacular traction has been reported to occur as a result of toxoplasmosis chorioretinitis resolution and scar formation.[5,11,12] Oréfice et al.[5] have found residual tractional maculopathy in 2 out of 15 patients which was not seen by fundoscopy but only using SD-OCT. Both of these 2 cases represent milder forms than our case which had superficial retinal folds involving the macula. Alfredo Adán et al.[12] found macular traction by fundus examination only in 2 of 15 patients with ocular toxoplasmosis which was severe and late resulted in pars plana vitrectomy. For both patients, vision improved from 20/400 to 20/40 in one patient and 20/100 to 20/30 in the other. This suggests that toxoplasmosis related macular traction has a spectrum which starts from changes detected only by SD-OCT and ends with severe traction which might need surgery. Giuseppe Scarpa et al had a case of superotemporal toxoplasmosis chorioretinitis which was properly treated with pyremethamine and sulfadiazine for 45 days. 2 months after initial presentation, the patient was having an acceptable vision with superficial macular corrugation which was interpreted as epiretinal membrane. The patient came back with a profound macular traction that happened 8 months after resolution of toxoplasmosis chorioretinitis for which a pars plana vitrectomy was done and vision improved from 20/200 to 20/20. It is possible that the earlier superficial macular lesion was an early vitreomacular traction which has resulted in profound vitreomacular traction indicating progression of traction which might result from episodes of reactivation. Therefore, we suggest that toxoplasmosis related vitreomacular traction is a progressive disease through its spectrum most likely due to episodes of reactivation and needs regular follow ups with serial fundus examination and SD-OCT, with fundus fluorescine angiography if there was any suspicion. It is also important to council the patients that a upon any change in vision, they need to visit the Emergency Room to rule out recurrence of toxoplasmosis retinochoroiditis or progression of vitreomacular traction.

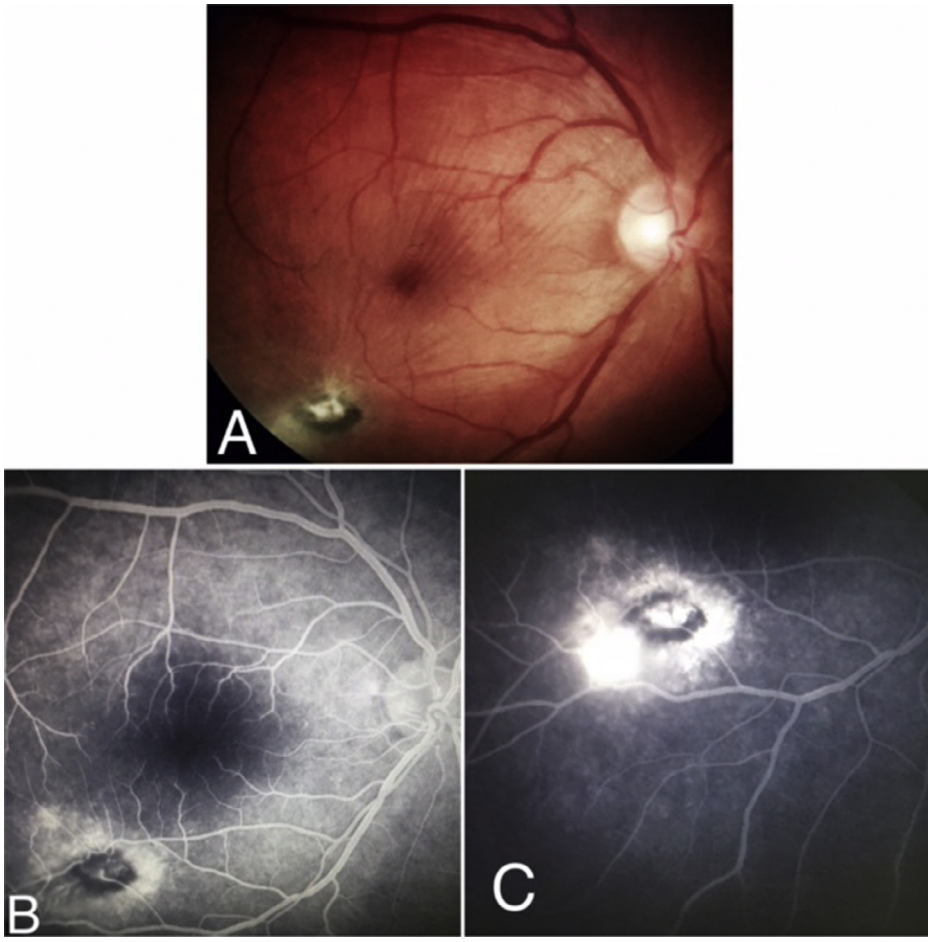

Fig. 3. (A) shows scarring at the area of previous chorioretinal lesion with peripheral suspecious area of chorioretinitis. (B) shows fundus fluorescine angiography with early hyperfluorescence peripheral to the scar. (C) shows fundus fluorescine angiography with late leakage peripheral to the scar indicating an active satellite lesion.

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