Acute Neuro-Retinitis Secondary to a Recently Acquired Ocular Toxoplasmosis in a Young Immunocompetent Patient

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ABSTRACT
A 27-year-old male patient with no medical history presented with a painless unilateral vision loss in the right eye, counting fingers at 5 meters, with active vitreous inflammation, stage II papilledema, whitish lesion on the superior-temporal border of the optic disc and a poor macular reflex, without any active or scarring chorioretinal lesion in the periphery. Multimodal imaging showed intra- and subretinal macular edema with early hypo-fluorescence followed by progressive centripetal filling of the peripapillary lesion and finally papillary diffusion. The visual field revealed an inferotemporal defect. Blood work showed hyperleukocytosis with positive toxoplasma serology, the rest of the workup was negative, especially no immunosuppression. The diagnosis of toxoplasmic anterior optic neuropathy was made due to the papillary and peripapillary involvement without a distant active or scarring lesion, with positive toxoplasma IgG and IgM serology. Trimethoprim/sulfamethoxazole antibiotic treatment with systemic corticosteroid therapy was initiated in our patient with an excellent outcome, including restoration of visual acuity to 10/10, complete vitreous clearance, complete resolution of the papillary and macular edema, and the peripapillary lesion without visible scarring, but leaving a persistent defect of the corresponding retinal nerve fibers. This clinical case thus illustrates a toxoplasmic anterior optic neuropathy with direct papillary involvement complicated by macular edema, a rare and atypical clinical entity of ocular toxoplasmosis, especially in a young immunocompetent subject, reflecting the clinical polymorphism of this pathology.

Keywords: Acquired Ocular Toxoplasmosis, Anterior optic neuropathy, Neuro-retinitis

1. INTRODUCTION
Acquired or congenital ocular toxoplasmosis [1], is a major cause of infectious posterior uveitis in immunocompetent patients [2], [3], whose severity is correlated with associated complications: macular edema, retinal detachment, retinal vascular occlusions and choroidal neovascularization [4].

Involvement of the optic disc during ocular toxoplasmosis is uncommon and usually associated with a clinical polymorphism that may raise several differential diagnoses [2].

The aim of this work is to illustrate a clinical case of a rare papillary involvement during ocular toxoplasmosis, with an atypical evolution.

2. CASE REPORT
A 27-year-old male patient with no significant past medical history presented with a ten-day history of painless loss of vision in the right eye (RE).

Initial examination of the RE showed limited visual acuity to counting fingers at 5 meters, a relative afferent pupillary defect, with normal anterior segment and intra-ocular pressure. The posterior segment showed active vitreous inflammation with an exudative whitish retinal infiltrate extending from the superior-temporal rim of the disc, associated with stage II papillary edema and poor macular reflex. Examination of the peripheral retina revealed no active retinal lesions or associated scarring (Fig. 1).
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Examination of the left eye (LE) was normal. Macular optical coherence tomography (OCT) revealed macular intraretinal and subretinal edema in the RE (Fig. 2), and fluorescein retinal angiography showed hypo fluorescence of the peripapillary lesion in the early stage (Fig. 3a), associated to a progressive centripetal filling, and perilesional vasculitis with papillary diffusion in the late stages (Fig. 3b). Automated static visual field showed a lower altitudinal defect (Fig. 4).

Blood tests revealed hyperleukocytosis with positive toxoplasma IgG and IgM serologies; all other serologies were negative including Syphilis, Bartonella, Sarcoidosis, Lyme disease; Tuberculosis and immunodeficiency virus.

The treatment combined antibiotic therapy with trimethoprim/sulfamethoxazole (TMP/SMX) 800/160 mg at a dose of 1 tablet twice daily for 6 weeks, combined with intravenous boluses of methylprednisolone 1 g per day for 3 days, 48 hours after starting antibiotic treatment, and followed by oral prednisolone 1 mg/kg/day with gradual tapering.

5 days after initiating treatment, there was an improvement in visual acuity up to 3/10 and a decrease in macular edema.

Due to the papillo-macular involvement with significant visual impact, the antibiotic treatment (TMP/SMX) was maintained beyond 6 weeks for prophylactic purposes.

Clinical evaluation at 6 months showed a restored visual acuity to 10/10 with complete vitreous cleaning, complete resolution of papilledema and retinal infiltrate with no visible retinal scarring, and no residual macular edema. However, there was still a defect in the superior temporal retinal nerve fibers (Fig. 5), with a persistent arcuate defect in the visual field.

3. Discussion

Involvement of the optic disc in ocular toxoplasmosis is most often secondary to a distant active chorioretinal lesion [2], but can sometimes result from primary involvement of the optic disc, giving the aspect of toxoplasmic anterior optic neuropathy [2], [5].

Toxoplasmic anterior optic neuropathy is a rare manifestation of ocular toxoplasmosis, characterized by a persistent vertical, arcuate, or central visual field defect [5], and whose visual prognosis is not correlated with papillary involvement itself, but with the associated macular involvement [2].

It may result from a juxta-papillary focal chorioretinal lesion, also known as Jensen's juxta-papillary retinochoroiditis, leaving a visible retinal scar, or from direct focal optic disc involvement, which has an excellent visual prognosis with minimal or no peripapillary scarring and persistent vertical or arcuate visual field loss [2].

The papillitis illustrated in this case, associated with focal thickening of the retinal nerve fibers without visible scarring at a later stage but leaving an arcuate defect in the retinal nerve fibers, is consistent with a toxoplasmic anterior optic neuropathy with direct papillary involvement [5], associated with mixed, cystoid and subretinal macular edema, giving the aspect of a toxoplasmic neuro-retinitis.

Besides the rarity of this clinical entity, its occurrence in the context of acquired ocular toxoplasmosis in a young immunocompetent patient highlights the peculiarity of this clinical case, since direct papillary involvement is often described in recurrent congenital toxoplasmosis or an immunocompromised subject [5].

Treatment of ocular toxoplasmosis is not systematic and the condition may resolve spontaneously; however, optic disc damage is one of the indications for anti-toxoplasma treatment [6], [5].
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Fig. 3. Fluorescein retinal angiography: (a) Hypofluorescent peripapillary lesion with peri-lesional and peripapillary flaming hemorrhagic spots creating a mask effect; (b) Centripetal hyperfluorescence of the retinal infiltrate with fluorescein uptake of the vessel wall adjacent to the infiltrate, giving the appearance of associated vasculitis. There is also papillary diffusion.

The classic first-line treatment consists of an association of pyrimethamine- and sulfadiazine-based antibiotics with folinic acid supplementation [6], and requires hematologic monitoring due to numerous side effects [7]. One of the alternative treatments consists of a combination of pyrimethamine and azithromycin [6], with less risk of side effects.

The use of trimethoprim-sulfamethoxazole in our patient was mainly motivated by the unavailability of pyrimethamine in Morocco and by the proven efficacy of this antibiotic therapy in numerous studies demonstrating a non-inferiority to conventional treatment with a similar risk of side effects [8]–[10]. The use of azithromycin alone remains controversial, given its efficacy and especially its lower toxicity.

The risk of recurrence of ocular toxoplasmosis is highest in the first two years after the infectious episode [11], although preventive treatment is not systematically required, but is reserved for forms in which recurrence may lead to visual threat or visual field loss [12]. Papillary or peripapillary involvement is, therefore, one of the indications for long-term prophylactic treatment, especially TMP-SMX for 1 year [11], [13], with regular clinical and hematologic monitoring [12].
Fig. 5. Color fundus photography at 6 months complete resolution of papilledema, macular edema and retinal infiltrate without visible retinal scarring, but with loss of superior temporal retinal nerve fibers.

4. Conclusion

The diagnosis of ocular toxoplasmosis should not be limited to the presence of a typical chorioretinal focal lesion, but should also include findings of anterior optic neuropathy or neuro retinitis, demonstrating the clinical polymorphism of this entity.

Conflict of Interest

Authors declare that they do not have any conflict of interest.

References