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CASE REPORT

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Ann-Pascale Guagnini
Patrick De Potter
Laurent Levecq
Alexandra Kozyreff

Atypical spherical deposition on vitreoretinal interface associated with toxoplasmic chorioretinitis

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A.-P. Guagnini (✉) · P. De Potter · L. Levecq · A. Kozyreff
Department of Ophthalmology,
Université Catholique de Louvain,
Cliniques Universitaires St-Luc,
Avenue Hippocrate,
1200 Brussels, Belgium
e-mail: ann-pascale.guagnini@ofta.ucl.ac.be

Abstract *Purpose*: To report the clinical and optical coherence tomography features of spherical deposits associated with recurrent toxoplasmic chorioretinitis. *Methods*: Case report. *Results*: Atypical, 100 μm to 150 μm, greyish deposits appeared along retinal arteries and veins as well as on the vitreoretinal interface in the macula of a 44-year-old Caucasian woman while she was being treated for recurrent toxoplasmic chorioretinitis with antiparasitic drugs and subconjunctival injection of betamethasone. They disappeared progressively with the systemic use of corticosteroids. Their clinical course was nicely documented by optical coherence tomography. *Conclusions*: Greyish spherical deposits on the vitreoretinal interface have been previously described as inflammatory reactions in asymptomatic human T-lymphotropic virus type I (HTLV-1) carriers and in patients with HTLV-1-associated uveitis. For the first time, optical coherence tomography (OCT) provided clinical correlation of this unusual presentation of toxoplasmic chorioretinitis.

Keywords Retinochoroiditis · Ocular toxoplasmosis · Deposits · Vitreoretinal interface · Posterior uveitis · Human T-lymphotropic virus type I

Introduction

Ocular toxoplasmosis has triggered a lot of interest in the past 50 years. Nowadays, new aspects of the disease are still being discovered. Apart from the characteristic focal retinochoroiditis adjacent to a nearby retinochoroidal scar and vitritis, we find neuroretinitis, papillitis, Jensen's retinochoroiditis. Less frequently, we find outer retinal toxoplasmosis, multifocal retinochoroidal lesions, scleritis, isolated retinal vasculitis and pigmentary retinitis [5].

We report and document tiny, greyish, spherical deposits found on the retinal arteries and veins as well as on the vitreoretinal interface in the posterior pole of a 44-year-old white Caucasian woman with recurrent toxoplasmic retinochoroiditis.

Materials and methods

We report the case of one patient who presented with a recurrence of a toxoplasmic chorioretinitis. Visual acuity, fluorescein angiography, optical coherence tomography, blood serology and treatment were evaluated.

Results

A 44-year-old Caucasian woman was seen for a progressive decrease in her visual acuity OS of 3 weeks' duration. On presentation, visual acuity was 20/20 in the right eye and 20/50 in the left eye. Anterior and posterior segment examination of the right eye was unremarkable. The left

eye presented with moderate inflammation in the anterior chamber and the vitreous. Fundus examination showed inflammatory reactivation of an old toxoplasmic retinochoroidal scar located in the superonasal quadrant (Fig. 1a). Systemic work-up consisted of examination of blood serology and chest radiography. Results of tests for syphilis, *Borrelia* spp. and *Bartonella* spp. were negative; toxoplasmosis IgM test results were negative, while those for toxoplasmosis IgG were positive. Lysozyme and angiotensin-converting enzyme (ACE) levels and the chest radiography findings were normal. Systemic treatment with pyrimethamine and sulfadiazine was started, and, 3 days later, a subconjunctival injection of betamethasone was given because the patient refused systemic corticosteroids. Corticosteroids were decided upon because of the proximity between the focus and the optic nerve. The parasitic focus showed progressive scarring, but visual acuity decreased to 20/60 after 1 month of treatment. Fundus examination revealed the presence of multiple, 100 μm to 150 μm, grey deposits along the retinal veins and arteries and on the vitreoretinal interface in the macular region (Fig. 1b), confirmed by optical coherence tomography (Fig. 2). The deposits blocked the underlying fluorescence on angiography. Oral systemic treatment with methylprednisolone 0.5 mg/kg per day was then given, with slow tapering over a 2-month period. The patient's response was satisfactory. Four months after the initial presentation, visual acuity returned to 20/20. The inflammatory retinochoroidal focus was transformed into an inactive retinochoroidal scar. While the spherical greyish deposits disappeared on the vitreoretinal interface in the macular region (Fig. 2), some deposits persisted along retinal veins.

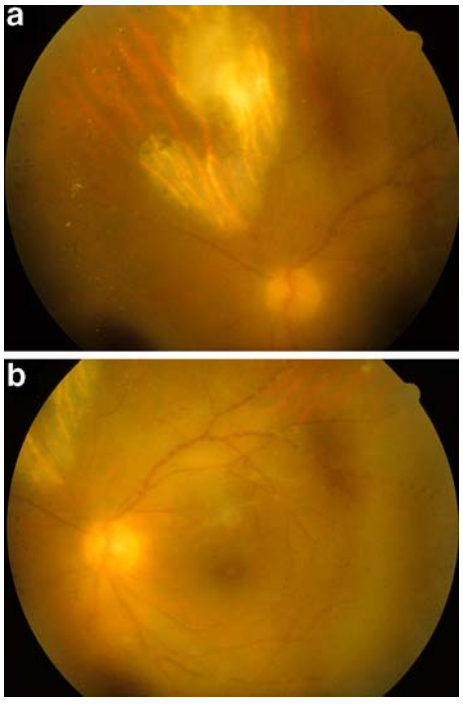

Fig. 1 **a** Toxoplasmic scar and reactivation focus in the superonasal quadrant. **b** Small grey deposits are situated along arterioles, veins and macular region

Discussion

In his textbook J.M.D. Gass initially described the possible presence of multiple, small, granular deposits that may develop along the inner retinal surface in the vicinity of the acute toxoplasmic retinitis [2]. Among other features seen in toxoplasmic retinitis, peri-arteriolar plaques (Kyrieleis' arteriolitis) simulating arterial emboli may occur either in the vicinity of the acute retinitis or remote from it [5]. The atypical, granular, inflammatory deposits seen in our patient were different from those described in Kyrieleis' arteriolitis as they were located along the retinal veins and arteries and on the vitreoretinal interface in the macular region.

Similar spherical greyish deposits were also reported in 14.5% of human T-lymphotropic virus type I-associated uveitis [3] and in one case of ocular syphilis [1]. Nakao and associates described granular inflammatory deposits in a female patient with acute retinal necrosis due to varicella zoster virus, and in a male patient with pre-proliferative diabetic retinopathy [4]. Both patients were found to be otherwise asymptomatic carriers of HTLV-I. Post-vitrectomy electron microscopy examination of those deposits showed debris of disrupted cellular components with

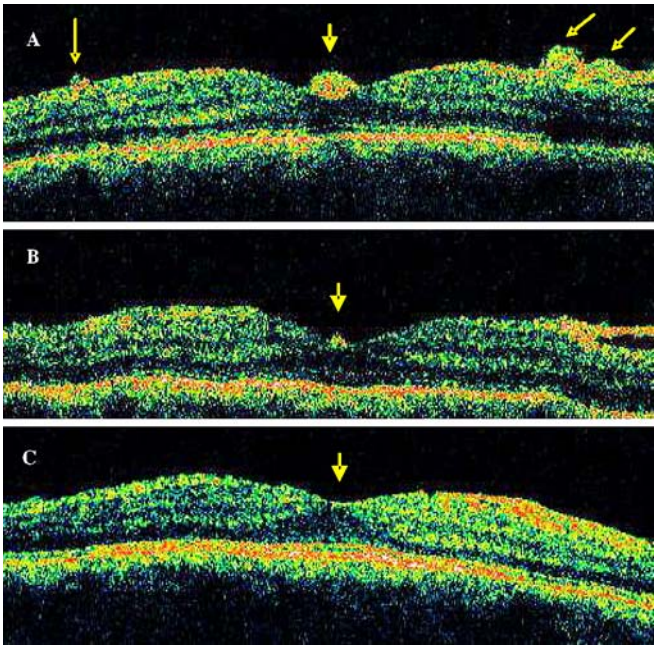

Fig. 2 Horizontal OCT scans crossing the foveola. Hyper-reflective deposits on the foveolar and the perifoveolar region progressively fade after systemic antiparasitic and corticosteroid treatment (*arrows*)

materials resembling thickened basement membrane, but it did not explain the pathophysiological mechanism [4]. The result of an HTLV-I blood serology test on our patient was negative.

Our patient illustrates the multiple grey deposits found on vitreoretinal interface described by Gass in ocular toxoplasmosis. Our case shows that such deposits may appear away from the active toxoplasmic focus. We did not find other similar cases in the literature in association with ocular toxoplasmosis, but these granular deposits are a common feature in HTLV-I carriers. They disappeared with combined, systemic, antiparasitic and corticosteroid treatments. OCT was helpful in following the resolution of the lesions.

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