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SHORT COMMUNICATION

Case report

Frosted branch angiitis and late peripheral retinochoroidal scar in a patient with acquired toxoplasmosis

D. DÍAZ-VALLE[1], E. DÍAZ-RODRÍGUEZ[2], T. DÍAZ-VALLE[1], J.M. BENÍTEZ DEL CASTILLO[3], N. TOLEDANO[1], M.J. FERNÁNDEZ ACEÑERO[4]

[1]Cornea and Ocular Inflammation Unit, Department of Ophthalmology, Hospital General de Móstoles, Madrid
[2]Vitreo-Retina Unit, Hospital Juan Ramón Jiménez, Huelva
[3]Cornea and Ocular Inflammation Unit, Department of Ophthalmology, Hospital Clínico San Carlos, Madrid
[4]Department of Pathology, Hospital General de Móstoles, Madrid - Spain

PURPOSE. *To report a case of acute frosted branch angiitis associated with acquired toxoplasmosis in which a late peripheral chorioretinal scar developed.*
METHOD. *Case report.*
RESULTS. *A 32-year-old man without systemic symptoms presented with sudden visual loss in his left eye. Examination demonstrated frosted branch angiitis without necrotizing chorioretinitis. Serologic tests showed elevated Toxoplasma gondii-specific immunoglobulin M antibody titers. Antitoxoplasmic therapy and oral steroids healed the ocular inflammation. In a follow-up visit one year later, a peripheral chorioretinal scar was noted.*
CONCLUSIONS. *Acute frosted branch angiitis without focal necrotizing chorioretinitis can be a manifestation of acquired toxoplasmosis. This could be an important, and sometimes forgotten, sign of the disease. (Eur J Ophthalmol 2003; 13: 726-8)*

KEY WORDS. *Acquired toxoplasmosis, Frosted branch angiitis, Retinochoroidal scar*

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INTRODUCTION

Ocular toxoplasmosis is one of the most frequently identified causes of posterior uveitis, and *Toxoplasma gondii* remains the most common pathogen to infect the retina in otherwise healthy individuals (1). Necrotizing retinochoroiditis is the hallmark of ocular toxoplasmosis. It is commonly associated with anterior uveitis, vitreous inflammation, and retinal vasculitis. Although periarteritis related to toxoplasmosis is common, diffuse sheathing of arteries and veins (frosted branch angiitis) has been infrequently described in patients with toxoplasmic retinochoroiditis (2). We report a case of acute frosted branch angiitis associated with acquired toxoplasmosis that developed a late peripheral chorioretinal scar.

Case report

A previously healthy 32-year-old man without any systemic symptoms complained of sudden loss of vision in his left eye. Visual acuity was 20/20 in the right eye and 20/80 in the left. Slit-lamp examination showed a 3+ cellular reaction in the left eye. Initial fundus examination disclosed a moderate (2+) vitre-

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ous haze and prominent and diffuse white sheathing of arteries and veins, consistent with frosted branch angiitis in the left eye (Fig. 1a). A detailed examination of the peripheral retina, including scleral depression, was performed and no retinochoroidal infiltrates were found. Ophthalmic evaluation of the right eye had normal results. Fluorescein angiography demonstrated prominent leakage of dye from the retinal vessels, but no vascular occlusions (Fig. 1b). Routine laboratory evaluation, including complete blood count, blood chemistry, urinalysis, PPD, chest x-ray, and serologies for toxoplasma, human immunodeficiency virus, and syphilis, was performed. Serologic tests for *T gondii*-specific immunoglobulin M antibodies by immunofluorescence antibody testing showed a titer of 1:128. All other laboratory studies were within normal limits. The patient was begun on antitoxoplasma therapy (sulfadiazine, pyrimethamine, and folinic acid) and oral prednisone, 0.5 mg/kg/day. The patient showed total resolution of the frosted branch angiitis within 4 weeks. One year after initial evaluation, there was a retinovascular narrowing, mild optic nerve pallor, and a new retinochoroidal scar in the upper periphery (Fig. 2). Visual acuity was 20/20 in the left eye.

DISCUSSION

Frosted branch angiitis was first reported in 1976 to describe the bilateral white perivascular retinal sheathing seen in a young immunocompetent boy. Subsequent reports have delineated the classic findings in frosted branch angiitis, such as decreased visual acuity, a preferential involvement of veins over arteries, and a rapid response to systemic corticosteroids (3). Toxoplasmic retinochoroiditis has been included on the list of differential diagnoses of frosted branch angiitis, but there have been few reports of this association (2). On the other hand, intraocular inflammatory reactions without focal necrotizing retinochoroiditis on initial examination have also been described in ocular toxoplasmosis, with development of inactive scars during follow-up examinations; this could be the result of live intraocular parasites producing the initial inflammation and lack of proliferation to evolve into necrotizing lesions in most of the patients with intact immune defenses (4). Retinal lesions associated

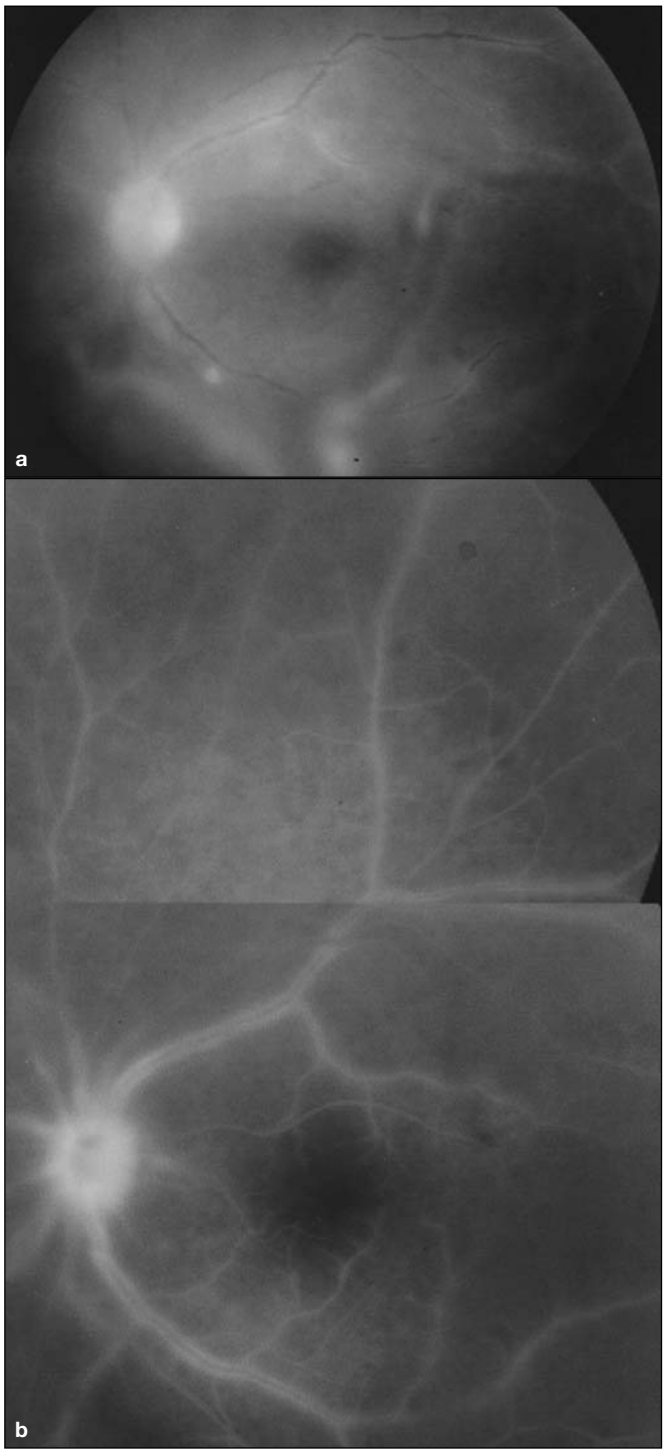

Fig. 1 - a) *Fundus photograph of the left eye shows diffuse sheathing of all retinal vessels and moderate vitreous haze, but no retinochoroidal scars.* **b)** *Fluorescein angiography demonstrates prominent leakage of dye from the retinal vessels and optic disk. These early phase photographs do not include the extreme periphery; peripheral ophthalmoscopic examination was irrelevant and there were no pathologic features to be shown.*

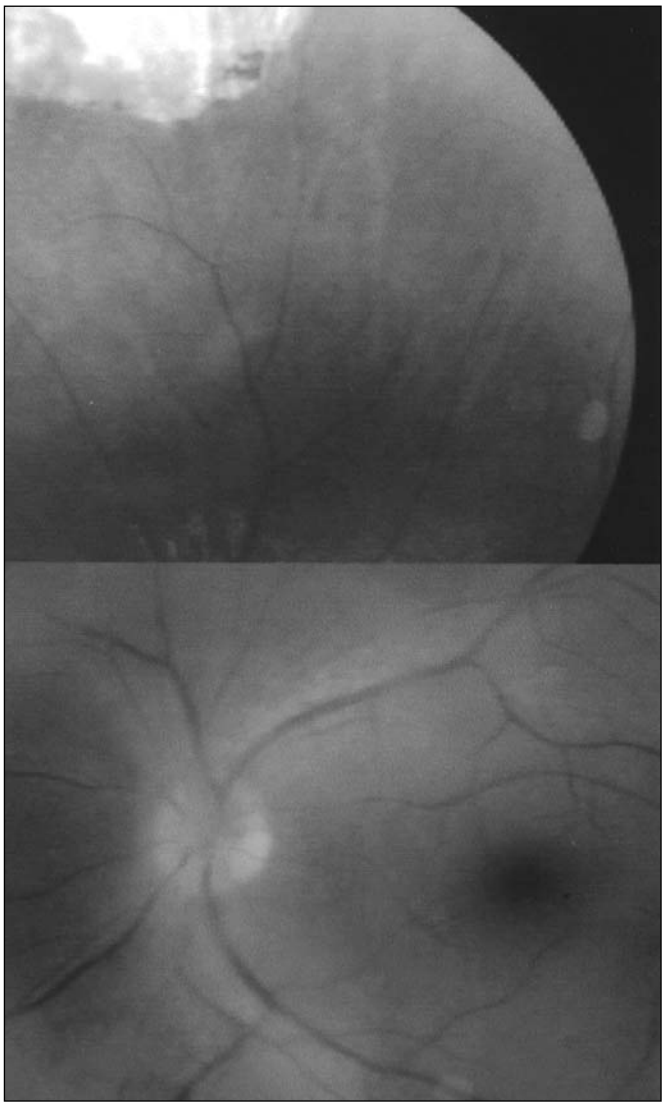

Fig. 2 - *One year after initial evaluation, there is retinovascular narrowing and a new chorioretinal scar in the upper periphery.*

with well-documented acquired infections can reactivate in a manner similar to that seen with congenital infections. It has been noted that postnatally acquired toxoplasmosis is characterized by frequent recurrences (5). The case presented here shows an atypical presentation of ocular toxoplasmosis as a frosted branch angiitis without the development of any retinochoroidal infiltrate. On the follow-up visit one year later, a peripheral inactive scar was noted and this could be the result of a subclinical reactivation due to the peripheral location of the inflammatory foci.

Although most cases of frosted branch angiitis are idiopathic, concomitant leukemia, lymphoma, autoimmune disorders, and active cytomegalovirus retinitis have been described (6). This case report demonstrates that frosted branch angiitis and the associated inflammatory reaction may be the only manifestation of a recently acquired *T gondii* infection. Thus, toxoplasma chorioretinitis should be remembered when considering the differential diagnosis of frosted branch angiitis. Further studies could examine recurrences or the development of a retinochoroidal scar.

Reprint requests to:
David Díaz-Valle, MD, PhD
Navahermosa 5, 2-A
28230 Las Rozas
Madrid, Spain
ddiazval@nacom.es

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