Short communication

Toxoplasmic retinochoroiditis: relapse vs choroidal neovascular membrane

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ABSTRACT

Clinical case: We report four patients with both decreased visual acuity and retinochoroidal lesions compatible with ocular toxoplasmosis in which a diagnosis of active toxoplasmic retinochoroiditis or choroidal neovascular membrane was made based on a specifically designed diagnostic screening.

Discussion: In the context of a compatible clinical picture, with retinochoroidal scars and low grade or absence of inflammation, choroidal neovascular membranes may mimic active toxoplasmic retinochoroiditis and vice-versa. A thorough ophthalmic, serological, and immunological examination (in ocular fluids) may help in the differential diagnosis allowing for proper therapeutic decision-making.

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Retinocoroiditis toxoplásmica: ¿recidiva o membrana neovascular coroidea?

ABSTRACT

Caso clínico: Se presentan cuatro casos de pacientes con deterioro de agudeza visual y lesiones retinocoroideas compatibles con toxoplasmosis ocular en los que el diagnóstico de retinocoroiditis toxoplásica activa o membrana neovascular coroidea se basó en un cribaje diagnóstico específicamente diseñado para ello.

Discusión: En el contexto de un cuadro clínico compatible, con cicatrices coriorretinianas sugestivas e inflamación escasa o ausente, las membranas coroideas neovasculares pueden simular cuadros de retinocoroiditis toxoplasmica activa y viceversa. Un exhaustivo estudio oftalmológico, serológico e inmunológico (del humor acuoso) puede facilitar el diagnóstico, permitiendo una adecuada toma de decisión terapéutica.

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Introduction

There are symptoms, as is the case of choroidal neovascularization (CNV), capable of triggering an acute deterioration of visual acuity (VA) in the context of a previous toxoplasmic retinochoroiditis and very similar in appearance to this disease that make differential diagnosis more difficult. Four patients with chorioretinal lesions resulting in a deterioration of VA are described herein. In these cases, the therapeutic decision was based on a protocol that included different criteria (clinical, immunological and microbiological). The use of this protocol allowed for resolution of symptoms in all cases.

Clinical case

Four patients, all of them female, with ages ranging from 19 to 44 years (mean age: 30 years), came to our inflammation unit reporting sudden loss of VA. They all had a documented history of previous toxoplasmic retinochoroiditis, although only two of them (patients 1 and 2) had received antitoxoplasma treatment. In all patients the presence of macular retinal scars and evidence of activity around the lesion area were observed.

Patient 1 showed macular scars with one whitish retinal lesion adjacent to one of the scars and the absence of vitritis (fig. 1A). She had received antitoxoplasma treatment in the past due to a relapse of inflammatory symptoms (fig. 1B).

Patient 2 showed macular scars with whitish lesion adjacent to one of the scars and the absence of vitritis (fig. 1A). In this case, diagnosed with toxoplasmosis relapse. Patient 2 showed macular scars with whitish lesion adjacent to one of the scars and the absence of vitritis (fig. 2A). She had received anti-VEGF treatment (bevacizumab) due to CNV secondary to previous toxoplasmosis (fig. 2B).

Patient 3 showed grayish scar lesion (fig. 3A). Angiography (FAG) revealed late hyperfluorescence and slight adjacent vasculitis (fig. 3B).

Patient 4 showed macular scars with a whitish retinal lesion and associated macular edema.
All patients were explored using optical coherence tomography (OCT) and fluorescein FAG. Findings for both tests are shown in table 1. Furthermore, the following supplementary tests were performed:

1. IgG and IgM anti-toxoplasma Gondii antibodies in serum.
2. IgG and IgM anti-toxoplasma Gondii antibodies in aqueous humor.
3. Infectious screening of other possible causes of necrotizing retinitis (herpes virus 1, 2, CMV, varicella zoster virus, tuberculosis, syphilis, toxochariasis).

Antibody tests were carried out simultaneously. The Goldmann-Witmer coefficient (GW) analysis was performed using the resulting determinations.

Treatment was prescribed based on clinical, immunological and microbiological findings.


Symptoms in all four cases were brought under control and no relapses were observed later on after at least a six-month follow-up.

**Discussion**

Even though in most cases the diagnosis of toxoplasmic retinochoroiditis is based on clinical findings, on certain occasions these do not suffice to establish diagnosis. As illustrated in the images (fig. 1A and fig. 2B), clinical presentation is virtually identical in both patients (patients 1 and 2) although their etiology is very different. Being aware of this possibility may prove essential for the visual prognosis of the patient, since delayed diagnosis...
could result in irreversible anatomical and functional sequelae.

On the other hand, as illustrated in patient 3 (fig. 3A), the clinical debut of toxoplasmic retinochoroiditis can be very similar to that of CNV (which, on the other hand, is a relatively common sequela and is therefore expected in these patients) and only a few small details observed during exploration (see vasculitis adjacent to the lesion area in fig. 3B), combined with other findings during screening, can point to the real origin of the lesion. The characteristic image of the relapse in the OCT (hyperreflectivity of the internal retinal layers due to retinal thickening/edema and posterior shadow) sets it a priori clearly apart from that of CNV (hyperreflectivity of the external retinal layers due to neovascular complex and subretinal liquid), although in patient 3 this difference was difficult to assess.

In spite of previous studies that demonstrated the efficacy of photodynamic therapy, the therapeutic approach to choroidal neovessels associated with toxoplasmosis seems to be limited to the use of intravitreal anti-VEFGs. In our case (patients 1 and 4), bevacizumab was prescribed in 3-injection patterns (1.25 mg/0.1 ml) during a four-month period with full resolution of symptoms.

Due to the high prevalence of seroconversion, Toxoplasma gondii IgG positivity has little diagnostic value. Thus, in the case of toxoplasmosis, and specially during the initial stages (less than one week of evolution), based on the patocronia of the inflammatory process, we advocate the use of GW coefficient versus the polymerase chain reaction (PCR) for the detection of toxoplasma (in those probable cases where both tests cannot be performed, which would be the ideal scenario, due to scarce aqueous humor samples): retinal cysts formed after primo-infection contain bradyzoites turning into tachyzoites (the pathogenic form of toxoplasma) which are released when the cyst ruptures (relapse) as a response to stimuli that have not been completely defined yet. However, release takes place at an extremely low pace and these can be observed at late stages, when inflammatory phenomena have already become evident, so that PCR sensitivity at this stage is considerably lower than GW’s. In patients with immunodeficiencies (i.e. AIDS) we recommend nevertheless performing a PCR, as their ability to produce antibodies could be compromised.

Finally, other diseases whose clinical features resemble those of toxoplasmic retinochoroiditis should be taken into consideration. Thus, and particularly in dubious cases as the ones described herein, we recommend performing thorough studies in order to discard them and increase at the same time the chances for diagnostic and therapeutic success.

Conflict of interest

None of the authors has declared any conflict of interest.

References


