Definition of acquired toxoplasmosis infection: When is it congenital or acquired?*  
Definición de la adquisición de la infección toxoplásmica, ¿cuándo es congénita o adquirida?

Dear Sir,

We read with great interest the article by Álvarez et al. recently published in the Spanish Ophthalmological Society on the clinical characteristics of ocular toxoplasmosis in an immigrant population in Barcelona, Spain.1

It is noteworthy that the authors defined as congenital forms the presentations with a large macular atrophic scar and pigmented edges. Traditionally, the presence of lesions at the macular level has been considered a sign of congenital toxoplasmosis. However, as described by Holland in a recent review on the influence of age in ocular toxoplasmosis,2 more recent studies show that the presence of macular lesions does not differentiate reliably between congenital infections and those acquired after birth. A study carried out by ourselves3 describes the clinical characteristics of ocular toxoplasmosis in a Colombian cohort where the acquisition of the infection was determined in only 30% of patients (20% congenital, 10% acquired), with a large percentage (70%) remaining undetermined precisely because it is not possible to demonstrate whether the infection of a patient was acquired after birth if there is no evidence of seroconversion or serological demonstration of an acute infection (IgM+, IgG−) and subsequent positivization of the IgG antibodies, discarding natural IgM antibodies. Moreover, it is not possible to determine in an adult that the infection was congenital if there is no certainty about the infection being diagnosed at the prenatal level by means of the techniques described in literature, either immunological such as avid IgG, IgA, or through molecular biology with the PCR of gene B1 in amniotic liquid.

To illustrate the mistake of classifying the type of infection based on the morphological characteristics of the retinal choroiditis, we can observe case number 4, documented in tables 1 and 2 of the article by Álvarez et al. A 25 year-old Colombian male, with an active peripheral lesion of multiple foci without adjacent scars, IgM+ IgG+, but classified as congenital because he exhibited active macular lesions. In Columbia, IgM can be positive up to a maximum period of 2 years after the infection4 allowing us to assume that this patient exhibited an acquired and not congenital infection as suggested by the type of the lesion. On the other hand, although this is registered as a first episode, the fact of exhibiting peripheral inactive lesions (the article does not mention how many) gives rise to the assumption that the patient exhibited several activation episodes during the period of time since his first infection.

On the basis of our experience, identifying in a patient the type of infection (congenital or acquired), if the diagnostic is postnatal in many cases it is highly valuable information for the patient even though it would not modify the therapeutic approach. For this reason, we must be very careful when classifying the toxoplasmosis infection with ocular involvement.

We consider it very important to prepare guidelines to facilitate the diagnostic and treatment of patients with ocular toxoplasmosis. In the cases of patients from regions with high prevalence of this infection and with circulation of virulent parasite types such as in Colombia and Brazil, such guidelines would guarantee a reduction in impact on eyesight and on the quality of life of these patients.

REFERENCES


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Dear Sir,

We appreciate the interest in the comments related to our article by the group of de-la-Torre et al., who have a considerable experience in ocular toxoplasmosis. As they point out, the only way to determine conclusively the congenital or acquired origin of the toxoplasma infection are pre- and perinatal tests.1,2 Our study consists in the analysis of the clinical characteristics of 22 adult patients of different countries of whom we were unable to obtain any type of previous serological analysis. Accordingly, considering both factors, a probability classification was established based on the funduscopy aspect lacking absolute certainty as pointed out in the article (i.e., according to fundoscopic appearance of the lesions and the patient antecedents, the patients were classified in two groups: those with probable congenital involvement ...), following previously published criteria,3,4 although subsequently Holland,5 mainly through unpublished observations by Silveira, clarified that this is not an entirely reliable classification. In fact, other authors like Bosch-Driessen et al.6 also drew up clinical classifications in the absence of available serological tests (they defined as congenital children under 2 years of age exhibiting inactive cicatricial lesions, and in this subgroup they found a high rate of macular involvement). Consequently, and notwithstanding the classification by Holland5 as they adequately point out, this data should not be taken as a definitive classification.

Patient number 4 in our series is clearly a very interesting case for several reasons and perhaps the summary table could give rise to some confusion. This was a 25-year-old patient from Columbia who did not refer any similar clinical episode and was in fact diagnosed by us as having ocular toxoplasmosis. For this reason and even though he exhibited chorioretinal scars demonstrating that the toxoplasmosis was recurrent, he was classified as “no previous episodes” because the column in the table refers to episodes known by the patients and recorded in the anamnesis. The only antecedent provided by the patient was amblyopia in the right eye which exhibited a large and inactive macular scar (in contrast with what you commented), and for this reason at the clinical level the primary infection had taken place in childhood at the latest. In the other eye the patient exhibited 4 peripheral scars and 2 peripheral foci non-adjacent to said scars. Therefore, in the presence of bilateral and recurring toxoplasmosis which remained inactive for many years, we were surprised by the slightly high value of IgM because, as pointed out by Gómez-Marín et al.,7 the IgM values remain high up to 2 years after the first infection. Even so, the amnesia seemed to show that the patient had been infected many years earlier. In fact, Holland4 indicated that it is not possible to know if reinfections may arise, even more so considering that our patient could have been in contact with 2 different strains of toxoplasma (that of his country of origin and that of his country of residence). Finally, we should point out that it could have been a false positive but unfortunately we have no serologies to confirm this and, as you aptly comment in your letter, the classification of the patient does not influence the treatment. Therefore and considering the clinical context of the patient and the absence of pre- or perinatal data, it is not possible to determine whether this is both a congenital and acquired infection and we can only describe typical clinical characteristics, which is the aim of the text.

REFERENCES


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