OCULAR TOXOPLASMOSIS: SHOULD ALL PATIENTS BE TREATED?

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Ocular toxoplasmosis is a retinochoroiditis due to *Toxoplasma gondii*, an obligate intracellular protozoan. Toxoplasmic retinochoroiditis is the main cause of posterior uveitis in several geographic areas. Ocular infection occurs in 2% and 18% of seropositive individuals in the US and Southern Brazil, respectively. The parasite establishes a love-hate relationship with the eye, manipulating the immune response and inducing variable initial lesions and further relapses. The severity of the disease is mainly due to the parasite genotype and the host immune status. Even though the parasite was identified more than a century ago, infection remains a potential blinding condition in all age groups, starting before birth. Diagnosis is usually easy to obtain even though laboratory investigations may be necessary. Mimicking a trench war, toxoplasmic lesions may progress from the periphery towards the posterior pole with an end-stage blinding condition.

Therapeutic management is the most controversial issue. Until recently, because of the rare but well-documented allergic and life-threatening reactions that can be associated with antibiotics, and because of the usually self-limited nature of the disease, treatment used to be proposed to patients with lesions within the vascular arcades of the posterior pole (zone 1), dense vitritis or severe ocular inflammation requiring corticosteroids, retinal vein occlusion, papillitis with visual field defects and retinal detachment. It is also highly recommended in immunodeficient patients.

Even though evidence-based data on the efficacy of anti-parasitic drugs are still missing, new strategies with a good safety profile are available and may be proposed earlier during the course of the disease, but also in selected cases, in order to reduce sight-threatening relapses. Therefore, indications and strategies must be adapted to each situation. The ideal drug for ocular toxoplasmosis must be parasitocidal, destroying cysts, reaching optimal concentrations in the posterior segment and having an acceptable tolerance profile. None of the current available drugs fulfill all these criteria. The classic treatment of ocular toxoplasmosis consists in pyrimethamine (100mg loading dose the first day followed by 50 mg daily), sulfadiazine (1g every 6 or 8 hours), folinic acid (5mg daily) and systemic corticosteroids (iv methylprednisolone in sight-threatening cases and/or oral prednisone 1mg/kg daily). The mean duration of treatment is 6 weeks but longer regimens may be needed in immunocompromised hosts or more virulent strains.

Other alternatives such as intravitreal clindamycin and dexamethasone seem to be equivalent to oral pyrimethamine, sulfadiazine and prednisolone. The intravitreal regimen may be an option during pregnancy or in patients with systemic intolerance to antibiotics.[1] It is important to consider that an increasing number of ophthalmologists choose to propose well-tolerated molecules such as azithromycin to nearly all patients with ocular toxoplamsosis. Secondary prophylaxis is another major issue in one-eye patients and those with frequent recurrences or macular threat. Silveira et al. have shown that the rate of relapse was 6.6% in a group of patients treated with trimethoprim (160mg) / sulfamethoxazole (800mg) once every 3 days, and 23.8%.in a group of patients without prophylaxis. The study was performed during a period of 20 months.[2] Revisiting the therapeutic options and indications may be an important step towards long-term maintenance of the visual function and avoidance of major complications.

References

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2- Silveira C et al. The effect of long-term intermittent trimethoprim/sulfamethoxazole treatment on recurrences of toxoplasmic retinochoroiditis. Am J Ophthalmol. 2002;134(1):41-6.