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Short Communication

Usefulness of vitrectomy in the treatment of ocular toxoplasmosis

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Although not useful in acute ocular toxoplasmosis, vitrectomy can be indicated at a later stage for the management of *complications* such as persistent vitreous opacification, secondary epimacular membrane, and retinal detachment.

In some cases, vitreous opacification can persist despite intensive antibiotic and steroid treatment. After 2 or 3 months of treatment, we must ask the following questions: is the vitreous opacification due to inflammatory cells or to vitreous degeneration? Is it still possible to reduce it with steroids ? If opacification is due to large vitreous cords and secondary vitreous degeneration, steroid treatment is no longer indicated and vitrectomy could be proposed in order to clear the posterior cavity, to improve the visual acuity and to avoid overtreatment with steroids.

Epimacular membranes secondary to toxoplasmosis are not much different from idiopathic ones, and the postoperative prognosis is good.

Retinal detachment is rare. It can be either rhegmatogenous and/or tractional.

In some rare cases, vitrectomy is indicated in the management of the acute phase, especially when the diagnosis is not clear. Vitrectomy can help rule out ocular lymphoma and viral retinitis. At the beginning, vitrectomy should be performed, with the infusion turned off, until the eye collapses. Generally, we can obtain 1 cc of undiluted vitreous. Once the infusion is turned on, other syringes are used for the collection of peripheral vitreous. With this material, it is possible to examine the lymphocytes and perform PCR. Viruses can be detected as well by PCR.

Vitrectomy increases the clearance of the posterior cavity. Some specialists believe that it can favorably influence the evolution of uveitis, although this point is debatable. In our series of eight cases of vitrectomy performed for toxoplasmosis, one case is interesting in this respect. A 40 year old woman visited us for the 7th recurrence of toxoplasmosis. On fundus examination, we could see the consecutive scars of previous infection along the upper temporal arcade. At the end of this chain, there was a large peripheral active retinochoroiditis, measuring 2 disc diameters. This lesion was treated with antibiotics and steroids, but despite several months of treatment, a dense network of vitreous cords persisted. Because of decreased visual acuity and vitreous debris, a vitrectomy was performed. No further recurrence was observed during a follow-up of twenty years. One can ask if vitrectomy had not removed factors that could be implicated in the recurrence process.