## Answer to Ophthaproblem *continued from* **page 1415**

### 3. *Toxoplasma* retinochoroiditis

The protozoan *Toxoplasma* is a coccidian, obligate, intracellular parasite of worldwide distribution responsible for zoonotic infection in humans and other mammals. *Toxoplasma* can cause severe life-threatening or destructive cerebral and ocular disease in newborns, and it is an important cause of ocular disease in both immunocompetent and immunosuppressed individuals.[1]

*Toxoplasma* can affect the eye, producing retinochoroiditis, and is a frequent cause of retinal disease. Ocular toxoplasmosis might be congenital or acquired.[2] When a susceptible pregnant woman acquires primary toxoplasmosis, transplacental transmission of the parasite to the fetus might occur. It is endemic in South America and occidental Africa. Ocular toxoplasmosis has been reported to manifest most commonly between the second and fourth decades of life. It is a necrotizing retinitis that can cause blindness and that might have a long-term evolution with several clinical relapses. Recurrence of the infection is attributed to multiplication of the parasites from retinal cysts located at the border of the scars.[3] What triggers this reactivation of the cysts remains unknown.

*Toxoplasma* is the most frequent cause of infectious retinitis in immunocompetent patients. Most cases are caused by reactivation of prenatal infections. The active form of retinitis is usually associated with granulomatous or nongranulomatous uveitis. Symptoms of ocular toxoplasmosis include blurry vision, pain, floaters, and redness of the eye. It can be remarkably atypical in situations of evident immunosuppression such as AIDS, malignancy, and use of chronic immunosuppressive drug therapy.[4]

Secondary potential complications of ocular toxoplasmosis are cataracts, glaucoma, cystoid macular edema, retinochoroidal shunts, papillitis, subretinal neovascularization, and even blindness.

Diagnosis is usually based on the clinical appearance of the fundus lesions. Serologic confirmation of exposure to *Toxoplasma* organisms serves as supportive evidence. In fact, there is no correlation between inflammatory activity and serum antibody titres. Atypical cases might require either a vitreous or an aqueous sample. Polymerase chain reaction is capable of detecting *Toxoplasma gondii* DNA in either an aqueous or a vitreous sample in only one-third of patients with ocular toxoplasmosis. Anti-*Toxoplasma* immunoglobulin G or A antibodies can be detected in either an aqueous or a vitreous sample.

*Toxoplasma* retinochoroiditis is highly characteristic and can be diagnosed only based on the typical fundoscopy findings: a white-to-yellow creamy lesion located near a pigmented chorioretinal scar is the most characteristic feature. It must initially be differentiated from cytomegalovirus retinitis; however, cytomegalovirus retinitis is almost exclusive to severely immunocompromised patients. The pigmented scar is not present. Cytomegalovirus retinitis evolves rapidly to the "cheese pizza" typical appearance. Fungal endogenous endophthalmitis usually appears after serious surgery or when venous or arterial catheters are present (*Candida* spp are the most common pathogens). *Toxocara* retinochoroiditis is another ocular parasite infection, typically found in children as a single granuloma, either in posterior pole or midperiphery, accompanied by mild to severe vitritis and epiretinal fibrosis.

Treatment is mandatory in all immunosuppressed patients with active lesions. However, not all active lesions of retinal toxoplasmosis need to be treated in immunocompetent patients. The mere presence of a focus of retinitis is not always an indication for treatment. Generally, small peripheral lesions heal spontaneously and can be followed conservatively. Indications for therapy include severe vitritis, which causes moderate vision loss, or location of the lesion on the macula, threatening the optic nerve head (Jensen papillitis) or main blood vessel.

Therapy includes antimicrobial drugs with or without corticosteroids. Several drugs have been proposed, including pyrimethamine, sulfadiazine, spiramycin, clindamycin, and trimethoprim-sulfamethoxazole. The most common combination is pyrimethamine, sulfadiazine, and corticosteroids, but trimethoprim-sulfamethoxazole and corticosteroids are currently being used owing to their safety and effectiveness. Traditional short-term treatment of active lesions does not prevent recurrences.[5] The most common side effects are associated with pyrimethamine and include hematologic complications, such as thrombocytopenia and leucopenia; therefore, supplementation with folinic acid is recommended. Corticosteroids must be used judiciously when the lesion is threatening the optic nerve head or is in the macula, or when severe vitritis is present: usually between 30 and 60 mg/d of prednisone, started 24 hours after anti-*Toxoplasma* therapy and gradually withdrawn before finishing the anti-*Toxoplasma* treatment.

*Toxoplasma* retinochoroiditis is a frequent retinal disease characterized by the onset of blurry vision, pain, and redness of the eye. It can be diagnosed on the basis of the typical fundoscopic findings: a white-to-yellow creamy lesion located near a pigmented chorioretinal scar.

**Dr Moreno-Martin** is an ophthalmologist at the Hospital del Henares, **Dr Vallejo-Garcia** is an attending ophthalmologist at the Hospital Universitario La Paz, and **Dr Rayward** is an ophthalmology resident at the Hospital Clínico San Carlos, and **Dr Vano-Galvan** is an attending dermatologist at the Hospital Universitario Ramón y Cajal, all in Madrid, Spain.

**Competing interests**
None declared

**References**

1. Balasundaram MB, Andavar R, Palaniswamy M, Venkatapathy N. Outbreak of acquired ocular toxoplasmosis involving 248 patients. *Arch Ophthalmol* 2010;128(1):28-32.
2. Burnett AJ, Shortt SG, Isaac-Renton J, King A, Werker D, Bowie WR. Multiple cases of acquired toxoplasmosis retinitis presenting in an outbreak. *Ophthalmology* 1998;105(6):1032-7.
3. Roberts F, Mets MB, Ferguson DJ, O'Grady R, O'Grady C, Thulliez P, et al. Histopathological features of ocular toxoplasmosis in the fetus and infant. *Arch Ophthalmol* 2001;119(1):51-8.
4. Holland GN. Ocular toxoplasmosis in the immunocompromised host. *Int Ophthalmol* 1989;13(6):399-402.
5. Commodaro AG, Belfort RN, Rizzo LV, Muccioli C, Silveira C, Burnier MN Jr, et al. Ocular toxoplasmosis: an update and review of the literature. *Mem Inst Oswaldo Cruz* 2009;104(2):345-50.