



Ocular toxoplasmosis: Little is known, much less is true

Majority of cases no longer thought to be congenital; treatment and understanding evolving

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Figure 1 Active toxoplasmosis, with positive IgM circulating antibodies for toxoplasmosis. Whitening of the retina, vitreous cells disappeared in 1 week.

Baltimore—Many misconceptions exist about ocular toxoplasmosis, the most prevalent being that the majority of cases of toxoplasmosis are congenitally acquired.

According to Rubens Belfort, MD, PhD, MBA, the contrary is true, i.e., most cases are acquired. Dr. Belfort provided up-to-date information about the disease at the Current Concepts in Ophthalmology meeting, Baltimore.

Transmission

"For generations, we believed that all cases of ocular toxoplasmosis were congenitally acquired. That is not true. We started to see cases of acquired disease in 1985 and 1986. That was the first time that we had a chance to study siblings with bilateral ocular toxoplasmosis that was not congenitally acquired," Dr. Belfort said. He is head professor and president, Vision Institute, Department of Ophthalmology, Federal University of São Paulo, São Paulo, Brazil.

He and his colleagues have studied more than 500 families with more than one sibling with ocular toxoplasmosis.

"It is highly improbable that one mother would give birth to five or six children with congenital toxoplasmosis. We now have a great deal of evidence that indicates most cases were acquired after birth and they were not congenitally acquired," he said.

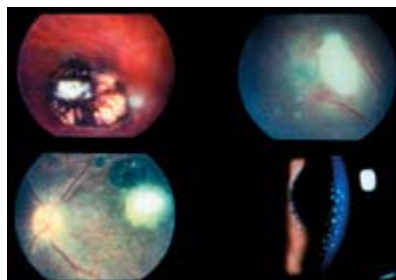


Figure 2 Pictured are examples of toxoplasmic active retinochoroiditis, satellite to old (hyperpigmented) scars and anterior uveitis secondary to the necrotic lesions in the retina.

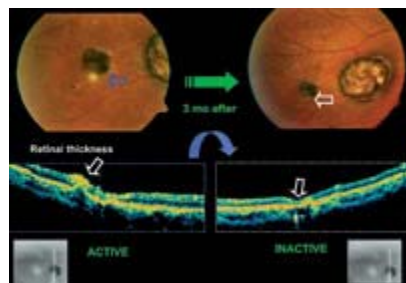


Figure 3 Optical coherence tomography imaging of ocular toxoplasmosis shows an active lesion and the lesion after 3 months.

Transmission of toxoplasmosis has been attributed to infected beef.

"That also is not true. There is no good scientific evidence that beef is the source of transmission of toxoplasmosis.

Undercooked lamb, pork, and chicken are the culprits in most cases, as well as the environment that is contaminated by the feces of infected cats," Dr. Belfort said. "Recently, water and air have been shown to be associated with some cases of transmission of ocular toxoplasmosis, with water responsible in many cases," he added. Other possible sources of the infection include organ transplantation and blood transfusion.

Dr. Belfort related a noteworthy case in which one domestic cat infected with the parasite gave birth and the feces of the animals contaminated a surface water reservoir. That contamination was responsible for transmission of the infection to more than 1,000 people in Brazil because the water was not filtered and the chlorination was inadequate to kill the oocysts. "That is probably the largest epidemic of systemic toxoplasmosis in the world," he



stated.

An examination of 408 patients infected in that outbreak showed that 10.2% of the patients developed ocular lesions during the acute stage of the infection.

The reason ocular toxoplasmosis is seen infrequently in acquired disease is that in 95% of cases, atypical self-limited and benign lesions—including retinal whitening, retinal vasculitis, anterior uveitis, and vitreous opacities—develop. Only in 4.4% of cases does typical necrotizing retinochoroiditis develop, according to Dr. Belfort.

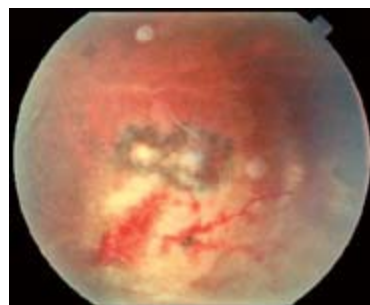


Figure 5 Severe vitreous toxoplasmic recurrence in a patient with recurrent toxoplasmosis.

Another prevalent misconception about the congenital form of the disease is the belief that a woman who has had IgG antibodies against *Toxoplasma* for years is protected against transmitting congenital toxoplasmosis to a fetus. A Brazilian study, however, showed that 10 women with IgG antibodies against *Toxoplasma* infected their children with the disease during pregnancy.

"As a rule, IgG antibodies to *Toxoplasma* protect the fetus, but that is not always the case," Dr. Belfort stated.

Detection

Ocular toxoplasmosis can be identified in the laboratory by serology, polymerase chain reaction, culturing, inoculation, and pathologic identification.

Molecular methods of detecting toxoplasmosis have limitations that include little automation, the lack of experienced technicians and appropriate laboratory design, and high costs, Dr. Belfort said.

Subtyping of the various *Toxoplasma* strains has been a great advance.

"We now know that there are many strains of *Toxoplasma*. Type I strain includes avian infections, type II strain two-thirds of all human isolates, and type III strain domestic animals," he explained, adding that equally important is that it seems variants also exist.

"In 2 to 3 years, we may be able to serotype our patients and be able to determine which patients will develop ocular toxoplasmosis and those who will be at high risk of having more recurrences than other patients," he said. "We are almost there, and it may be related to the genotype of the strains."

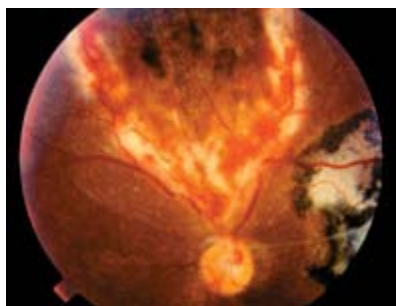
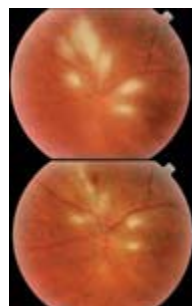


Figure 6 Active cytomegalovirus retinitis and an old, inactive scar secondary to ocular toxoplasmosis are seen in an HIV-positive patient with AIDS and a low CD4 count.

A consideration in patients with ocular toxoplasmosis is why some patients develop large lesions compared with other patients who do not. The natural history of the disease is not well known, Dr. Belfort said. Currently, with more advances in technology, it is possible to follow large cohorts of patients that are expected to provide clues to the clinical course in different patients.

Treatment



As many questions exist about the treatment of ocular toxoplasmosis as questions about the other features of the disease.

Prevention of toxoplasmosis is debatable, and it is unknown whether patients—specifically children—with the acquired benign systemic form of the disease should be treated in every case.

Treatment with pyrimethamine and sulfadiazine until now has been considered better, but Dr. Belfort questioned, "Better than what?" Trimethoprim and sulfamethoxazole (Bactrim, Roche) has fewer side effects and has better patient

Figure 7 Patient with toxoplasmic neoretinitis before (top) and after 1 week of anti-toxoplasmosis treatment. (Figures courtesy of Rubens Belfort, MD, PhD, MBA)

compliance while being probably equally effective as pyrimethamine and sulfadiazine, he said.

"As a rule in our clinic, we do not treat patients with pyrimethamine and sulfadiazine because of the advantages of trimethoprim and sulfamethoxazole," Dr. Belfort stated.

Steroid treatment is excellent when administered locally, systemically, periocularly, and even intraocularly in special cases, and always in combination with anti-toxoplasmic drugs, Dr. Belfort said.

The length of treatment, he noted, depends on the individual clinical picture. Retinal detachment is a potential complication and carries with it the risk of vision loss.

"That is especially true in children. Every eye with toxoplasmosis should be carefully examined for tears and treated to avoid retinal detachment," Dr. Belfort emphasized.

Disease recurrence has been studied in only one small clinical trial. A study of the long-term effects of intermittent trimethoprim (160 mg)/sulfamethoxazole (800 mg) on recurrent toxoplasmic retinochoroiditis showed that when medical therapy was administered every 3 days for 20 months, the disease recurred in only 6.6% of treated patients. Long-term intermittent treatment can reduce the rate of recurrent toxoplasmic retinochoroiditis.

"Recurrence is the cause of many eyes lost to ocular toxoplasmosis," Dr. Belfort said. "Little is known. Much less is true," he concluded.