

# Congenital toxoplasmosis: when a screening is missed

## Abstract

Congenital toxoplasmosis occurs in 1:1000 to 1:10000 live births and the spectrum of manifestations is wide. We report a case of male infant with confirmed congenital toxoplasmosis infection after seroconversion between the 2nd and 3rd trimester. Despite having started adequate treatment after birth, ophthalmological lesions were permanent. The benefit of maternal treatment is not well established and prevention should be the key.

**Introduction:** Transplacental transmission of *Toxoplasma gondii* is responsible for congenital toxoplasmosis. The variant parasite prevalence throughout the world, along with different national maternal screening programs, explains that the estimated incidence of this infection is of 1:1000 to 1:10000 live births.

The risk of infection depends on the timing of maternal infection, increasing during the course of pregnancy and reaching a 65-71% risk in the third trimester.<sup>1,2</sup> The gestational age at the time of maternal infection also accounts for the ample clinical outcome, being more severe in the early stages of fetus development, when it can lead to spontaneous abortion or stillbirth. Although congenital toxoplasmosis is subclinical in 75% of infected newborns<sup>1</sup>, the spectrum of manifestations is wide, going from hearing loss and ophthalmological lesions to severe compromise of central nervous system.

**Keywords:** gestational, congenital toxoplasmosis, ophthalmological, hearing loss, pregnancy

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## Case report

We report the case of a male infant, admitted to the neonatal care unit after birth due to maternal toxoplasmosis seroconversion. It was an otherwise uneventful and supervised pregnancy, with three ultrasounds described as normal and remaining negative serologies (rubella, syphilis, HIV and HBV). Seroconversion was verified at 36 weeks (IgM 4.68IU/mL, IgG >200IU/mL, avidity 49%), following absence of screening for toxoplasmosis during 2nd trimester, and spiramycin was initiated. He was born full-term to a 30-year-old woman, gravida 2, para 1, by vaginal delivery, with Apgar score 9 and 10 at one and five minutes respectively. His physical examination was unremarkable, BW 3595 (P50), L 48cm (P50), HC 35cm (P50).

Both serum IgG and IgM for toxoplasmosis were positive with low avidity. *Toxoplasma gondii* PCR was positive on cerebrospinal fluid. Placenta mice inoculation with Ag *T. gondii* was positive at three weeks. Newborn was started on pyrimethamine (2mg/kg/d), sulfadiazine (100mg/kg/d) and folinic acid (10mg 3/w) on day one of life and maintained during the first year of life with close monitoring. Transfontanelar ultrasound, performed at day 3, revealed intracranial calcifications in the frontotemporal lobe. Ophthalmological observation confirmed microphthalmia and microcornea with anterior uveitis and cataract on the right eye and macular chorioretinitis on the left one. At three years old, despite total loss of vision, he has adequate psychomotor development.

## Conclusion

Although the infection is usually asymptomatic in the pregnant woman, consequences to the newborn can be quite deleterious.

Systematic screening during pregnancy raises questions concerning the cost/benefit ratio, difficulty in interpreting results and misdiagnosis. Furthermore, the precise benefit of prenatal treatment on preventing vertical transmission and long term sequelae in the newborn is not well established. It is important to raise awareness to an adequate prevention, keeping in mind that it is a preventable disease. In confirmed cases, treatment should be promptly started and kept for a minimum of 12 months as it will improve prognosis, reducing the appearance of intracranial calcifications and the progression of chorioretinitis. Follow-up should include Pediatric and Ophthalmology appointments.

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## Conflicts of interest

The authors declare there are no conflicts of interest.

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