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Diagnosis and Management of Toxoplasmosis

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Vertical transmission of the parasite *Toxoplasma gondii* can produce significant morbidity and mortality to the fetus and newborn and long-term sequelae to children and adults [1]. Congenital toxoplasmosis primarily occurs in the offspring of women who acquired their primary *T gondii* infection during gestation. Congenital disease is almost never seen in women who acquired their infection in the distant past and before conception. The two major exceptions to the latter dictum include chronically infected women who reactivate their latent *T gondii* infection during gestation because of immunosuppression (eg, AIDS) and women who acquired their primary infection shortly (within 3 months) before conception.

Greater than 90% of pregnant women who acquire their primary *T gondii* infection during gestation are asymptomatic, and approximately 85% of children born having congenital toxoplasmosis do not initially exhibit any signs of disease; however, the parasite has the potential to cause significant long-term damage to infected progeny, and may bring major emotional and economic burdens to parents, relatives, and society. Laboratory diagnosis performed during

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pregnancy and at birth can reveal those mother-baby pairs at risk for congenital toxoplasmosis and those who have infected offspring.

Congenital toxoplasmosis is a preventable and treatable disease [1]. From the time a pregnant woman is at risk of ingesting *T gondii* to the moment her progeny is infected with the parasite, there are many opportunities for health care workers to intervene and stop this potentially tragic cascade of events. Primary prevention (aimed at preventing primary *T gondii* maternal infection during gestation) can be achieved through education targeted at women who have never been exposed to *T gondii* (*T gondii* IgG negative). These women need to be reminded throughout gestation of the behavioral risks that could expose them to acquiring the parasite during gestation. Secondary prevention (aimed at preventing fetal infection during gestation) can be attained by putting in place programs for universal serological screening, as practiced in France and Austria, aimed at detecting women who acquire their primary infection during gestation and who do not experience any symptoms. For these women, an attempt can be made to decrease fetal infection with spiramycin, a prenatal diagnosis of congenital disease can be performed by polymerase chain reaction (PCR) examination of amniotic fluid, and in-utero treatment can be initiated for the fetus suspect or proven to be infected.

This article is intended to serve as a summary of the authors' current approach to diagnosis and management of toxoplasmosis during pregnancy. The roles of commercial nonreference laboratories and reference laboratories are differentiated and emphasized.

The organism and its life cycle

T gondii is an obligate intracellular protozoan that exists in nature in three forms: (1) the oocyst (which releases sporozoites), (2) the tissue cyst (which contains and may release bradyzoites), and (3) the tachyzoite. Oocysts are formed in the small bowel of members of the cat family only, and are excreted in their feces for periods varying from 7 to 20 days. As many as 10 million oocysts may be shed in their feces in a single day, and these become infectious (by sporulation) in 1 to 21 days, depending on temperature and availability of oxygen. Tachyzoites are crescent- or oval-shaped and require an intracellular habitat to survive and multiply. Tachyzoites reside and multiply within vacuoles in their host's cells, and can infect most phagocytic and nonphagocytic cell types, including placental cells. The presence of tachyzoites in human fluids or tissues is the hallmark of acute infection or reactivation of a latent infection.

Following cell entry and replication of the tachyzoite form, encystation and formation of tissue cysts may occur. The conditions that result in cyst formation are not known. The tissue cyst is formed within a host cell and may vary in size from those that contain only a few organisms (bradyzoites) to those 200 μm or greater in size that contain several thousand bradyzoites. The central nervous