Toxoplasmosis: Diagnosis, Treatment, and Prevention in Congenitally Exposed Infants

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ABSTRACT
Toxoplasmosis is a rare disease caused by the obligate intracellular protozoan parasite, *Toxoplasma gondii*. Most persons with toxoplasmosis in the United States are asymptomatic, but if a woman is infected during pregnancy, the parasite can cross the placenta and cause congenital toxoplasmosis in the fetus. The severity of congenital toxoplasmosis depends on when in the pregnancy the mother is exposed, but it can cause ocular and central nervous system disease as well as lead to growth failure and hearing and vision abnormalities. Congenital toxoplasmosis is treated with a combination of pyrimethamine, sulfadiazine, and leucovorin. It is important for pediatric nurse practitioners to be aware of the clinical presentation and treatment of congenital toxoplasmosis. J Pediatr Health Care. (2011) 25, 355-364.

KEY WORDS
Congenital toxoplasmosis, toxoplasmosis, ocular toxoplasmosis, retinochoroiditis, hydrocephalus, pPediatrics, nurse practitioner, congenital infections, TORCH

The TORCH complex refers to five major congenital infections that, when contracted by a fetus in utero, lead to serious and often life-threatening clinical sequelae. The “T” in TORCH stands for toxoplasmosis, an infection by the intracellular parasite *Toxoplasma gondii* (Gerber & Hohlfeld, 2003). *T. gondii* is a member of the phylum Apicomplexa and parasitic subclass coccidian. The primary host of this parasite is the cat (feline family), and it is passed through the feces of felines (Pradhan, Yadav, & Mishra, 2007). Humans can act as an intermediate host in the parasite’s life cycle. If a woman is infected while pregnant, this parasite can cross the placenta from mother to fetus and cause damaging effects to the fetal eye, brain, and other tissues leading to congenital toxoplasmosis (Gerber & Hohlfeld, 2003). It is important that pediatric nurse practitioners (PNPs) be aware of this disease, recognize when it should be considered as a differential diagnosis, and understand how it is diagnosed and treated. This article will review the epidemiology, pathophysiology, transmission, risk factors, clinical presentation, diagnostic methods, and treatment of toxoplasmosis and will emphasize the role of the nurse practitioner in clinical education and research.

EPIDEMIOLOGY
The prevalence of toxoplasmosis varies greatly around the world (Jones, Kruzon-Moran, Sanders-Lewis, & Wilson, 2007). Prevalence rates are thought to depend on food production and harvesting practices, water treatment, environment, climate, and exposure to soil or sand (Jones et al., 2007). To gather data regarding the prevalence of this parasite in the United States, serum samples were taken from more than 15,000 volunteers as part of the National Health and Nutrition Examination Survey between 1999 and 2004 (Jones et al., 2007). Results of this study demonstrated that among women of childbearing age (15-44 years), the...
prevalence of women with IgG antibodies to *T. gondii* born within the United States is 11%. For women born outside of the United States, the prevalence was higher, at 28.1% (Jones et al., 2007). The prevalence rates of IgG antibodies to *T. gondii* in women of childbearing age are important to monitor because they provide insight into the prevalence of congenital toxoplasmosis. No large-scale studies have examined the prevalence rates of IgG antibodies to *T. gondii* in pregnant women, and toxoplasmosis is not a nationally reported disease (Lopez, Dietz, Wilson, Navin, & Jones, 2000). According to the Division of Parasitic Diseases of the National Center for Infectious Diseases, it was estimated in the United States in the year 2000, 1 in 10,000 live births results in congenital toxoplasmosis (Brown, Chau, Atashband, Westerberg, & Kozak, 2009; Lopez et al., 2000). The incidence of congenital toxoplasmosis is then estimated to be around 400 to 4000 new cases every year (Lopez et al., 2000; Pinard, Leslie, & Irvine, 2005).

**PATHOPHYSIOLOGY**

*T. gondii* is an obligate intra-cellular protozoan parasite that is responsible for the disease toxoplasmosis (Tamma & Serwint, 2007). This parasite has a complex life cycle that is relatively host specific and is divided into three infectious stages (Dubey, 2004; Kravetz & Federman, 2005). The preferred primary host for *T. gondii* is felines (cats), but humans can become infected when they act as an intermediate host. When an intermediate host ingests *T. gondii*, the first stage, tachyzoites, enter a cell and create a vacuole to protect themselves from the host’s immune system (Dubey, 2004). Tachyzoites are resilient and are capable of entering and reproducing in almost any mammalian or avian cell (Rorman, Zamir, Rilkis, & Ben-David, 2006). Tachyzoites contained within certain immune cells can be disseminated throughout the body until an adequate immune response is mounted between 7 and 10 days after infection (Kravetz & Federman, 2005). In response to the host’s immune system, tachyzoites multiply asexually and produce cysts, each of which contains the next stage, bradyzoites (Dubey, 2004). Each individual cyst can contain hundreds of bradyzoites and can be found within many different types of tissue (Dubey, 2004).

When a cat ingests a tissue cyst in meat it consumed, enzymes in the stomach and intestine degrade the cyst and bradyzoites are released. Through asexual reproduction these bradyzoites will become tachyzoites again, the first aforementioned stage (Dubey, 2004). Some bradyzoites will invade the epithelial tissue of the feline intestine and will begin to multiply through sexual reproduction to form a fertilized oocyst (Kravetz & Federman, 2005). These oocysts can only be formed in the intestine of a wild or domestic member of the feline family and cannot be formed in an intermediate host such as humans (Dubey, 2004).

Oocysts pass out of the feline host through feces and become sporulated in the environment, forming the third stage, sporozoites (Jones, Lopez, & Wilson, 2003). Any host that ingests sporozoites from the environment or acquires tissue cysts from eating infected meat will become infected with *T. gondii* (Jones, Lopez et al., 2003). In humans, contaminated fruit, vegetables, or water that has been in contact with cat feces is the source of ingestion of sporozoites from the environment. Tissue cysts are usually acquired through the ingestion of undercooked infected meat (Jones, Lopez et al., 2005; Pradhan et al., 2007). Bradyzoites released from an ingested tissue cyst or sporozoites released from an ingested oocyst penetrate the human intestine and become tachyzoites again. These tachyzoites then follow the cycle described previously (Dubey, 2004).

*T. gondii* has been shown to be a highly mobile parasite and actively travels through blood and lymph fluid and across biological barriers such as the intestinal wall, blood-brain barrier, and the placenta (Rorman et al., 2006). In humans, the transplacental passage of tachyzoites from mother to fetus leads to congenital toxoplasmosis (Dubey, 2004). In healthy adults, an infection of *T. gondii* is asymptomatic in most cases. The immune system will prevent replication of the parasite and destroy any bradyzoites that are released from dormant tissue cysts (Dubey, 2004). However, if a woman is infected during pregnancy, tachyzoites can cross the placenta and infect the fetus (Dubey, 2004). The symptoms and course of infection depend on many factors including inoculation factors, virulence of the particular organism, gestational age at time of infection, sex, genetic factors, and immune status of the mother and fetus (Pradhan et al., 2007). The cycle of exposure that leads to congenital toxoplasmosis is illustrated in Figure 1.

**VERTICAL TRANSMISSION**

Transplacental transmission of *T. gondii* occurs in approximately 40% of pregnancies in which the mother is exposed for the first time during the course of the pregnancy (Bonfioli & Orefice, 2005). In 90% of cases, the mother will be asymptomatic at the time of infection (Kravetz & Federman, 2005). It is estimated that 50% of expectant mothers who give birth to infants congenitally infected with *T. gondii* have no recollection of symptoms or any obvious exposure to the parasite
In symptomatic cases, the mother may experience a range of flu-like symptoms including fever, malaise, and cervical lymphadenopathy (Kravetz & Federman, 2005). Mothers infected prior to conception rarely transmit the parasite to the fetus except in cases where the parasite becomes reactivated because of the immune suppression of the mother (Jones, Lopez et al., 2003).

In the majority of cases of congenital toxoplasmosis, the fetus is exposed during the last trimester and symptoms in the infant range from mild to asymptomatic (Bonfioli & Orefice, 2005). If the fetus is infected during the first trimester, clinical manifestations are significantly more severe and may result in spontaneous abortion of the fetus. Infection during the second trimester also may result in a symptomatic infection, but the clinical manifestations vary from mild to severe and depend on individual factors (Bonfioli & Orefice, 2005; Jones, Lopez et al., 2003).

**RISK FACTORS**

The risk factors for *T. gondii* exposure are directly related to exposure to cats and more specifically to cat feces (Box 1). Because cats are the primary host for *T. gondii*, cats in the house or stray cats in and around the house or property are considered a primary risk factor for acquiring this parasite during pregnancy. Any job or activity that puts a pregnant woman in direct contact with soil, sand, or other material that could contain cat feces puts her at risk for being infected (Rabinowitz, Gordon, & Odofin, 2007). Drinking water that has been contaminated by cat feces also can expose a pregnant woman to *T. gondii* (Holland, 2003). In the United States, the majority of cases of congenital toxoplasmosis can be traced back to an exposure to material containing cat feces or the ingestion of raw food grown in soil containing cat feces (Safadi, Berezin, Farhat, & Carvalho, 2003). The ingestion of undercooked or raw meat during pregnancy is also a risk factor because the tissue may contain *T. gondii* cysts that, unless destroyed by cooking heat or food preparation practices, could infect a pregnant woman (Safadi et al., 2003).

**CLINICAL PRESENTATION**

Congenitally acquired toxoplasmosis causes a wide variety of signs and symptoms and typically presents in one of three ways. In the majority of cases, an infant is infected during the last trimester and shows mild to asymptomatic clinical symptoms in the neonatal period. If the mother is infected during the first trimester and the fetus is infected, the clinical manifestations are significantly more severe and may result in spontaneous abortion. Infection during the second trimester may result in a symptomatic infection, but the clinical manifestations vary from mild to severe and depend on individual factors (Bonfioli & Orefice, 2005; Jones, Lopez et al., 2003).

**BOX 1. Risk factors for contracting toxoplasmosis**

- Cats in the home or stray cats in or around the home
- Any job or activity that requires contact with dirt, soil, or other material that could contain cat feces
- Ingestion of raw meat, raw eggs, or unpasteurized milk
- Drinking untreated water
- Touching the eyes or face during or immediately after food preparation
- Ingestion of unwashed fruit or vegetables
will be asymptomatic or have subclinical symptoms at birth, making the condition difficult to diagnose (Brown et al., 2009; Safadi et al., 2003). A smaller minority of infants will present with overt symptoms in the neonatal period, while the third class of infants will present with symptoms within the first few weeks to months of life (Brown et al., 2009).

A PNP should be aware of any red flags in the history that would allude to the possibility of congenital toxoplasmosis. A history of hydrocephalus, retinochoroiditis, and calcifications in the central nervous system in the newborn period should immediately alert a care provider to the possibility of toxoplasmosis. The signs and symptoms may be less specific, however, and may not be present until later in infancy and childhood (Jones, Lopez et al., 2003). These symptoms include convulsions, palsies, growth or mental retardation, visual or hearing impairment, learning disabilities, organomegaly, lymphadenopathy, fever, and rash (Jones, Lopez et al., 2003). It is important to remember to place congenital toxoplasmosis on a list of differential diagnoses for any of the aforementioned constellation of symptoms. Differential diagnoses for congenital toxoplasmosis also include other congenital infections such as cytomegalovirus (CMV), rubella, or herpes viral infections (Jones, Lopez et al., 2003).

*T. gondii* can enter cells in the eye of the fetus through the blood supply of the eye and cause congenital ocular toxoplasmosis. Ocular toxoplasmosis is responsible for up to 17% of cases of uveitis and 25% of cases of posterior uveitis in the United States (Soheilian et al., 2005). Ocular effects of toxoplasmosis can be categorized depending on whether the signs and symptoms are present in the neonatal period or if they do not occur until later in life (Bonfioli & Orefice, 2005). In up to 80% of infants infected with *T. gondii* who do not receive treatment, ocular lesions will develop by the time they reach childhood or early adolescence (Wallon et al., 2004). The risk of ocular lesions decreases over time if no lesions are noted in the infant period (Freeman et al., 2008). Symptoms of ocular toxoplasmosis can vary depending on the age of the patient. Reduced visual acuity, strabismus, leukocoria, photophobia, pain, and nystagmus are common signs that should alert a medical provider to the possibility of toxoplasmosis (Bonfioli & Orefice, 2005; Jedari, Maliky, & Daneshjou, 2008).

*T. gondii* also can invade tissues in the central nervous system of the developing fetus and can cause areas of focal and diffuse necrosis in the cerebellum, cerebrum, spinal cord, and brain stem (Lago, Baldisserotto, Hoefel Filho, Santiago, & Jungblut, 2007). These areas of necrosis eventually become the central nervous system calcifications that are characteristic of this disease. It is believed that these areas of calcified tissue are formed from an inadequate amount of dendritic cells removing necrotic tissue at the affected sites (Lago et al., 2007). The site of these calcified lesions varies to some degree by the gestational age at which a developing fetus is exposed to *T. gondii*. A fetus exposed before the 20th week of gestation typically will have large dense lesions seen in the basal ganglia (Lago et al., 2007). A fetus exposed between the 20th and 30th weeks of gestation typically will present with small lesions seen in the lateral ventricles. A fetus exposed after 30 weeks’ gestation may have diffuse lesions in the cerebral parenchyma (Lago et al., 2007).

It is important that any infant with suspected or confirmed congenital toxoplasmosis receive imaging of the central nervous system. A computed tomography (CT) scan and ultrasound are the preferred diagnostic methods. It is important to use a diagnostic method that can adequately pick up areas of calcification in the infant brain (Lago et al., 2007). CT scanning is the first-line diagnostic method used in North America to detect central nervous system abnormalities caused by toxoplasmosis. If concerns exist about the effects of radiation in the neonatal period, an ultrasound can be used as an alternative diagnostic method. However, a negative ultrasound in a patient with confirmed congenital toxoplasmosis may need to be followed by a CT scan for confirmation because ultrasound results can vary depending on the examiner and technology used (Lago et al., 2007). Therefore, in an infant with confirmed congenital toxoplasmosis or in an infant who has symptoms, it may be advisable to order a CT scan to limit the amount of diagnostic imaging needed.

**PHYSICAL ASSESSMENT**

**Ocular Toxoplasmosis**

Toxoplasmosis affects the retina and the underlying choroid, causing retinochoroiditis, the most common manifestation of ocular toxoplasmosis (Smith & Cunningham, 2002). Retinochoroiditis is described as macular-pigmented lesions with a central necrotic area primarily found on the retina and can be observed by funduscopic examination. Retinochoroiditis is illustrated in **Figure 2**. In more than 50% of cases of ocular congenital toxoplasmosis, the lesions are found on the posterior pole of the retina and are unilateral (Bonfioli & Orefice, 2005). Upon examination of the eye, a medical provider will see a gray-white area of retinal necrosis with or without exudates with adjacent swelling of the optic disc, vitreitis, vasculitis, and
hemorrhage (Bonfioli & Orefice, 2005; Smith & Cunningham, 2002). A “headlight in the fog” is a common description of ocular toxoplasmosis, and it refers to the retinal inflammation seen through an infected and opaque vitreous (Bonfioli & Orefice, 2005). Active inflammation and infection in the eye typically lasts about 6 weeks, at which time the lesion will begin to regress, leaving behind a characteristic pigmented scar on the retina (Smith & Cunningham, 2002). Ocular toxoplasmosis that is allowed to proceed unchecked without treatment can lead to devastating long-term effects. It has been associated with glaucoma, cataracts, vitreous opacification, retinal hemorrhage or detachment, and optic atrophy. All of these conditions can lead to permanent blindness (Bonfioli & Orefice, 2005). Ocular lesions can recur in adolescence and adulthood, even after treatment in infancy. Follow-up of these patients is extremely important to prevent further damage to the eyes (Phan et al., 2008).

Central Nervous System Toxoplasmosis

A congenitally exposed infant with central nervous system calcifications may or may not have overt neurologic symptoms. Symptoms that have been documented in infants with congenital toxoplasmosis include convulsions, abnormal tearing of the eye, nystagmus, strabismus, hearing and visual impairments, and growth and developmental delays (Jones, Lopez et al., 2003). Many of these symptoms overlap with symptoms of ocular toxoplasmosis and could be attributed to either manifestation of this parasitic infection (Jedari et al., 2008). Toxoplasmosis can also cause hydrocephalus and microcephaly in the developing fetus (Dimario et al., 2009).

Sensorineural Hearing Loss and Toxoplasmosis

Toxoplasmosis also has been associated with sensorineural hearing loss. A literature review looking at the association between this parasitic infection and hearing loss found a scarcity of reliable data (Brown et al., 2009). Only five studies met the inclusion criteria for the literature review, and they reported such different results that it is impossible to discern the association between hearing loss and toxoplasmosis (Brown et al., 2005). Although the association and cause are not fully understood at this time, a nurse practitioner should be aware that the hearing of any child with a history of toxoplasmosis should be evaluated on a regular basis and that the child should be referred to an audiologist and ear, nose and throat specialist for follow-up (Brown et al., 2005).

DIAGNOSTIC TESTS

The prevention and treatment of congenital toxoplasmosis begins with identifying infection in pregnant women. Antibody testing that measures the amount of IgG and IgM is used to confirm exposure to *T. gondii*. IgG and IgM levels rise within 2 weeks of being exposed to the parasite (Jones, Lopez et al., 2003). Elevated IgG levels confirm a patient has been exposed to the parasite but do not differentiate between a recent exposure and an exposure that occurred in the past because IgG will persist at a low level throughout the life of the patient (Jones, Lopez et al., 2005). IgM antibody levels can be used to confirm an acute exposure, and the degree of elevation can be used to discern when the exposure occurred (Lopez et al., 2000). Although IgM antibodies are almost always present following an acute exposure, they can persist in some patients at high levels for up to 18 months, leading to an inaccurate assessment of when the exposure occurred. This situation can be problematic because congenital toxoplasmosis occurs when the mother is infected during her pregnancy, and the severity of the disease is determined by ascertaining when in the pregnancy the infection occurred (Nascimento, Suzuki, & Rossi, 2008). A significant increase in specific antibody titers or seroconversion during pregnancy is usually considered diagnostic of a recent exposure (Nascimento et al., 2008).

Despite serologic evidence demonstrating the likelihood of recent exposure, a *T. gondii* reference laboratory must confirm the diagnosis, in part because of questions about the sensitivity and specificity of IgG and IgM antibody testing (Tamma & Serwint, 2007). The Sabin Feldman Dye test is performed by a reference laboratory and is considered the gold-standard diagnostic test for toxoplasmosis. This test detects a change in *T. gondii*–specific antibody titers (IgG) over a 3-week period or detects a single elevated (IgG) antibody titer (Rorman et al., 2006). A four-fold increase in titer levels over a three-week period or a single titer above 250 IU/ml is considered highly suggestive of infection (Rorman et al., 2006). Polymerase chain reaction testing of amniotic fluid is the preferred method for providing confirmation of fetal exposure (Jones, Lopez et al., 2003).
This test should be performed at or after 18 weeks’ gestation and only in women with preliminary positive serologic results indicative of acute exposure (Montoya & Remington, 2008). Polymerase chain reaction testing of cerebrospinal fluid also can be used to confirm the presence of infection in the central nervous system after birth (Tamma & Serwint, 2007).

TREATMENT

Standard of Care

The goal of initiating treatment is to arrest the replication of the parasite and prevent further damage to the organs involved. It is especially important to stop replication in the eye to prevent irreversible damage to the retina and optic nerve that can lead to permanent blindness (Soheilian et al., 2005). Currently, The World Health Organization and the Centers for Disease Control and Prevention recommend pyrimethamine, sulfadiazine, and leucovorin as the standard of care for persons with congenital toxoplasmosis (Rorman et al., 2006). These medications were proven to be effective in a randomized prospective study called the National Collaborative Chicago Based Congenital Toxoplasmosis Study (NCCBTS). This study found that treatment with the three aforementioned medications significantly decreased adverse signs and symptoms associated with congenital toxoplasmosis, including ocular and central nervous system symptoms and sensorineural hearing loss (McLeod et al., 2006). This combination of medications also is recommended by the American Academy of Pediatrics (AAP). For patients with sensitivity to sulfadiazine, clindamycin can be used in combination with pyrimethamine as an alternative (AAP, 2009). Information regarding these medications and other alternative medications that could be used to treat toxoplasmosis can be found in the Table (Soheilian et al., 2005; Taketomo, Hodding, & Kraus, 2008). A provider also may add a corticosteroid to decrease the inflammation caused by the replication of the parasite and to manage the associated ocular complications (AAP, 2009; Soheilian et al., 2005).

Both pyrimethamine and sulfadiazine act by inhibiting folic acid synthesis in *T. gondii*. By using different mechanisms of action, they complement one another to create a combined effect (Schmidt et al., 2006). Although they have been proven effective, they do not come without serious adverse effects and should never be prescribed without diagnostic confirmation of toxoplasmosis (Schmidt et al., 2006). As previously stated,

<table>
<thead>
<tr>
<th>Medication</th>
<th>Mechanism of action</th>
<th>Dose</th>
<th>Length of therapy</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfadiazine*</td>
<td>Inhibits folic acid synthesis</td>
<td>100 mg/kg/day divided every 12 hours</td>
<td>1 year</td>
<td>Bone marrow suppression, fever, nephropathy</td>
</tr>
<tr>
<td>Pyrimethamine*</td>
<td>Inhibits thymidylate acid synthesis</td>
<td>1 mg/kg/day</td>
<td>First 6 months</td>
<td>Bone marrow suppression, rash, seizures, fever, vomiting, diarrhea, hematuria</td>
</tr>
<tr>
<td>Folinic acid (leucovorin)*</td>
<td>Reduced form of folic acid</td>
<td>5-10 mg every 3 days</td>
<td>First 6 months</td>
<td>Rash, erythema, urticaria, wheezing, thrombocytopenia, hypersensitivity</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
<td>Inhibits dihydrofolic acid synthesis</td>
<td>Consult infectious disease specialist</td>
<td>Consult infectious disease specialist</td>
<td>Pseudomembranous colitis, hypotension, cardiac arrhythmia, rash, bone marrow suppression</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>Inhibits protein synthesis</td>
<td>No standard dose; dose is based on weight and postnatal age; consult infectious disease specialist</td>
<td>Consult infectious disease specialist</td>
<td>Pseudomembranous colitis, hypotension, cardiac arrhythmia, rash, bone marrow suppression</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>Inhibits protein synthesis</td>
<td>Consult infectious disease specialist</td>
<td>Consult infectious disease specialist</td>
<td>Palpitations, chest pain, QT prolongation, diarrhea, nausea, rash, oesophagitis</td>
</tr>
</tbody>
</table>

*Standard of care.
Treatment for Expectant Mothers

Treatment of a woman during pregnancy also has been studied to prevent congenital toxoplasmosis. During the first trimester of pregnancy, pyrimethamine is contraindicated because of the teratogenic effects of this medication. Sulfadiazine may be used alone during the first trimester and pyrimethamine may be added to the regimen after this crucial period of fetal development if the benefit of this drug outweighs the risk to the fetus (Freeman et al., 2008). Although not yet approved by the Food and Drug Administration in the United States, another medication called spiramycin is used to prevent transplacental infection in many other countries. Spiramycin is available in the United States under special circumstances as an investigational medication (Rorman et al., 2006).

Overall, the research pertaining to anti-toxoplasmic treatment is lacking. The majority of studies are retrospective, and few randomized control trials exist that look at medication efficacy. More research must be done in this area to develop the best treatment for pregnant women and neonates congenitally exposed to T. gondii.

NEONATAL SCREENING

Screening for toxoplasmosis is a controversial topic. In populations with a low prevalence, screening of pregnant mothers is not believed to be cost-effective, nor is treatment during pregnancy guaranteed to prevent congenital toxoplasmosis (Dimario et al., 2009). Screening in the neonatal period may be a more feasible option for primary care providers (Jara, Hsu, Eaton, & Demaria, 2001). In 1986, Massachusetts added toxoplasmosis to its newborn screening and created follow-up recommendations for infants with positive serologic findings (Jara et al., 2001). Currently in the United States, Massachusetts and New Hampshire are the only two states that routinely screen for toxoplasmosis at birth. IgM and IgG antibody testing is used to screen all infants in these two states at the same time that all other newborn screening is conducted (Jara et al., 2001). It is important to note that positive serologic results demonstrate that the mother has been exposed and do not definitively indicate congenital toxoplasmosis in the infant (Jara et al., 2001). The results simply allow the primary care provider to be aware of the possibility and provide further follow-up as indicated.

Preliminary data from these two states suggest that the prevalence of congenital toxoplasmosis is 1 in 12,000 live births and that providing treatment to these infants early in life significantly decreases the neurologic and ophthalmologic effects of this disease (Jara et al., 2001). Between 1986 and 1992, 52 infants in Massachusetts and New Hampshire were identified as having been congenitally infected. Fifty of these infants were identified through neonatal screening alone, and after 1 year of treatment, only one infant demonstrated a neurologic deficit and four infants demonstrated lesions in the eye (Lopez et al., 2000). It is important to note that positive serologic results demonstrate that the mother has been exposed and do not definitively indicate congenital toxoplasmosis in the infant (Jara et al., 2001). The results simply allow the primary care provider to be aware of the possibility and provide further follow-up as indicated.

ROLE OF THE NURSE PRACTITIONER

Implications for Clinical Practice

PNPs play an important role in recognizing and treating congenital toxoplasmosis. Because screening for toxoplasmosis does not always occur during the prenatal period, many prenatal infections go unnoticed and undocumented. A mother may be unaware that she has been exposed to the parasite and unaware of the risks that T. gondii can pose to her infant. The first step for
all PNP s in primary care is to be aware of this infection and to ask each new mother about her possible exposure to the organism. Important screening questions presented in Box 2 should be asked at newborn visits and any prenatal visits. These questions can provide an idea of the level of risk and whether congenital toxoplasmosis is a possibility, especially if any abnormalities are noted in the newborn.

An infant that has been congenitally exposed to T. gondii will require medical care, monitoring, and follow-up throughout infancy, childhood, and adolescence. A PNP can provide a medical home and can coordinate primary care with specialty care including ophthalmology, neurology, audiology, and infectious disease specialists, depending on the clinical needs of the patient. Although treatment during infancy can decrease the long-term effects of congenital toxoplasmosis, children and adolescents who were treated in infancy are still at risk for ocular complications later in life. Because of this risk, it is important to ensure that patients receive routine ophthalmologic monitoring to identify ocular complications before they lead to permanent damage of the eye (Phan et al., 2008).

Patient Education

In the United States it is estimated that 85% of pregnant women have never been exposed to T. gondii and thus are at risk for contracting the parasite during pregnancy (Jones, Ogunmodede et al., 2003). Prevention of congenital toxoplasmosis begins with preventing primary infection. Despite the fact that T. gondii can be avoided by implementing relatively simple strategies in daily life, the majority of pregnant women are unaware of how to prevent exposure (Jones, Ogunmodede et al., 2003). A survey of 400 pregnant women in the United States demonstrated that only half were aware of toxoplasmosis. Most of these women knew toxoplasmosis was associated with cat litter but were unsure as to why and did not know about exposure in the environment through food, water, dirt, sand, or soil (Jones, Ogunmodede et al., 2003).

Sporulated oocysts can be found in dirt, sand, or soil and on the skins of raw fruits and vegetables grown in these substrates (Lopez et al., 2000). Limiting contact with dirt, sand, or soil can help prevent the ingestion of oocysts from the environment, and if contact occurs, an expectant mother should be taught to thoroughly wash her hands to avoid ingesting the parasite (Dimario et al., 2009; Lopez et al., 2000). Wearing gloves while gardening, for example, also can limit the contact a pregnant woman may have with these environmental hazards (Pinard et al., 2003). The skins of all raw fruit and vegetables should be washed and then peeled away because oocysts may be attached to these parts of the food and could be ingested. Again, hand washing should be strongly emphasized after handling any raw food including fruits, vegetables, and meat products (Lopez et al., 2000). T. gondii cysts can reside in the meat of many different types of mammals or birds. In the United States, it is estimated that 8% of beef and 20% of lamb and pork meat contains T. gondii tissue cysts (Kravetz & Federman, 2005). All pregnant women should be taught to never ingest raw meat and to cook all meat to an internal temperature of at least 152°F to destroy the tissue cysts (Kravetz & Federman, 2005).

Because cats are the primary host for T. gondii, it is important that pregnant women be aware of the risks they may pose. Contact with cat litter should be avoided if possible, and if contact is unavoidable, gloves should be worn while changing the litter box and hands should be washed thoroughly afterward (Lopez et al., 2000; Pinard et al., 2003). Frequent litter changes should be done because it takes several days for oocysts to become infectious, and the box should be thoroughly cleaned with disinfecting agents (Lopez et al., 2000; Pinard et al., 2003). Preventing a cat from hunting outdoors or eating raw meat also can prevent the feline from being infected with T. gondii. Practitioners should encourage pregnant women to keep indoor-only cats and to feed them only canned or dry food that has been bought in a store (Lopez et al., 2000).

Providing education to expectant mothers is an important part of the provision of primary care for PNP s. A practitioner should provide materials and information

**BOX 2. Important screening questions for Toxoplasma gondii seronegative expectant mothers to assess the risk of T. gondii exposure**

**Questions**

- Do you own a cat?
- If you own a cat, does your cat go outdoors or hunt and eat raw meat?
- Do you garden?
- Do you work or participate in any activity where you are directly exposed to sand, dirt, or soil?
- Do you eat meat? If yes, how is it prepared?
- Do you eat raw fruit and vegetables? If yes, how are they prepared?
- Have you traveled to any foreign countries? If yes, where and what did you eat, and did you drink the water?

Based on the answers to these questions, a practitioner can provide the necessary education to prevent exposure.
in a variety of languages and use common language instead of medical jargon to teach important points to patients. Handouts that are culturally sensitive and appropriate for mothers with low literacy skills or who cannot read should be used (Montoya & Remington, 2008). Using pictures and color demonstrations of hand washing, cooking, and wearing gloves may be helpful when teaching about toxoplasmosis if translation into another language is difficult (Montoya & Remington, 2008). In addition, creating handouts that a patient can simply hang in the home as a quick reminder may be useful. Figure 3 is an example of a handout for expectant mothers. Although research that looks at the role of prenatal education in preventing congenital toxoplasmosis is limited, current recommendations suggest that all pregnant women be given information through written materials and discussions with medical providers (Dimario et al., 2009). PNPs play an important role in providing this information to their patients and to expectant mothers.

**CONCLUSION**

PNPs play an active role in the primary care of infants. A PNP may be the first medical provider who sees a newborn after he or she is released from the hospital and can provide primary care throughout infancy and childhood. It is important that PNPs be able to recognize and diagnose congenital toxoplasmosis as well as provide and coordinate treatment and long-term follow-up care for these patients.

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