

# **Diagnosis and Management of Foodborne Illnesses**

A Primer for Physicians and  
Other Health Care Professionals

## **Congenital Toxoplasmosis Patient Scenario**

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This learning scenario can be used to reinforce medical management information pertaining to foodborne illnesses, such as that provided from the previous sections of this primer. This case study provides questions that need to be considered when dealing with a potential case of foodborne illness. Answers are provided immediately following the questions to enhance the learning process.

Similar learning scenarios are also available for other foodborne pathogens.

## **Congenital Toxoplasmosis, a Patient Scenario**

Susan, a 6-month-old infant, is brought to your office for evaluation of apparent blindness. Her mother reports that she had been well during the pregnancy and the delivery was uncomplicated. The baby appeared healthy until age 4 months, when the parents became concerned about her vision.

Physical examination was normal except for bilateral macular scars, microphthalmos, and unresponsiveness to visual stimuli. There were no other neurologic abnormalities, and her growth and development were appropriate for her age. A computed tomography (CT) scan of the head was obtained.

**Congenital infection with which of the following should be included in the differential diagnosis?**

**Viruses:**

Cytomegalovirus

Rubella

Herpes simplex

Human immunodeficiency virus

**Bacteria:**

*Treponema pallidum*

*Listeria monocytogenes*

**Parasites:**

*Toxoplasma gondii*

### **What additional information would assist with the diagnosis?**

More history from the mother, including travel to foreign country

Vaccination record, including during pregnancy

History of exposure to cats and raw meat

History of multiple sex partners and sexually transmitted disease (STD)

History of herpes

Evaluation of CT scan

The CT scan of the child's head showed periventricular calcifications and asymmetric dilation of the lateral ventricles. The mother is 35 years old and reiterated that she does not recall being ill during the pregnancy; however, she also indicated that she would not necessarily remember every little symptom. She also denied having a history of STDs. She had received the mumps-measles-rubella (MMR) vaccine as a child but no vaccines during pregnancy. The mother recalled eating insufficiently cooked meat while traveling in France during the first trimester of pregnancy. The family does not own a cat, and she does not recall having been exposed to cats during her pregnancy.

### **What diagnostic tests are needed?**

Serologic evaluation of both mother and child focusing on potential congenital infection (ie, a ToRCH profile) based on the history of the mother ingesting raw meat while traveling in a foreign country during first trimester of pregnancy and the clinical findings (blindness, cerebral calcifications, and hydrocephalus).

Results of serologic testing detected both IgG and IgM antibodies to *Toxoplasma gondii* in both the baby's and mother's serum. The mother's IgM titer was 0 and IgG titer was 1:6400, while those of the baby were IgM titer of 1:160 and IgG titer of 1:6400.

## **How does this information assist with the diagnosis?**

Diagnosis of toxoplasmosis is usually confirmed by serologic tests. Occasionally, organisms are identified in tissue or body fluids or isolated by culture or animal inoculation. Polymerase chain reaction (PCR)-based assays are available from some laboratories for diagnosis of fetal infection and infection in compromised hosts. For immunocompetent persons, seroconversion or a four-fold rise of specific IgG antibodies or demonstration of specific IgM antibodies indicate recent infection. High titers of IgG antibodies in the absence of IgM antibodies are consistent with chronic latent infection acquired in the past. The IgM-capture enzyme-linked immunosorbent assay (ELISA) is more sensitive than the IgM-indirect fluorescent-antibody assay (IFA) test. However, IgM tests may be false-positive, and true-positive IgM tests may persist for a year or more. Therefore, to determine if infection occurred during pregnancy, additional tests, such as an anti-*Toxoplasma* avidity test, may be required at a reference laboratory.

Immunodeficient persons usually do not have measurable IgM antibodies, even in the presence of active disease. The diagnosis of central nervous system (CNS) toxoplasmosis in such persons is therefore based on clinical picture, typical CT scan or magnetic resonance imaging (MRI) showing multiple ring-enhancing hypodense nodules, and a positive IgG test. Brain biopsy is reserved for cases that fail to respond to an empiric trial of anti-*Toxoplasma* drugs.

The baby was diagnosed with congenital toxoplasmosis.

## **How is toxoplasmosis best treated?**

Toxoplasmosis in immunocompetent persons rarely requires treatment, whereas infection in immunodeficient persons or in infants with congenital infections usually requires treatment. The combination of pyrimethamine and sulfadiazine is the treatment of choice. Folic acid (leucovorin) is given to prevent bone marrow suppression. Treatment must be continued for the duration of immunosuppression and for life in AIDS patients

whose immunity is not reconstituted by highly aggressive anti-retroviral therapy (HAART).

For persons unable to tolerate the pyrimethamine and sulfadiazine combination, high doses of pyrimethamine (and leucovorin) and clindamycin are effective.

The management of toxoplasmosis acquired during pregnancy is controversial. Testing of newly pregnant women for *T. gondii* infection is not routinely done, and routine testing is not recommended by CDC or by the American College of Obstetricians and Gynecologists. To prevent fetal infection, one approach is to administer spiramycin (a macrolide antibiotic, which is concentrated in the placenta and is not harmful to the fetus). At the same time, amniotic fluid is submitted for PCR-based testing to determine whether fetal infection has occurred. If so, options may include pyrimethamine and sulfadiazine given after the 16th week of pregnancy (since pyrimethamine is potentially teratogenic) or consideration of terminating the pregnancy. If the fetus is shown to be uninfected, spiramycin is continued throughout pregnancy.

Different protocols exist for treatment of infants born with congenital infection. The most commonly recommended treatment is pyrimethamine and sulfadiazine plus leucovorin during the first year of life. In the present case, the child was treated for 6 months with pyrimethamine and sulfadiazine plus leucovorin.

Human infection with the intracellular protozoan parasite *Toxoplasma gondii* occurs globally. Infection is usually subclinical or produces a mild illness, except in immunodeficient persons and fetuses infected in utero. Most infants with congenital toxoplasmosis appear healthy at birth but have a high incidence of developing serious ophthalmologic and neurologic sequelae during the next 20 years of life. Severe congenital toxoplasmosis may be apparent at birth or become apparent during the first 6 months of life. Chorioretinitis, intracerebral calcifications, and hydrocephalus, as in the present case, are typical features.

The child was treated with pyrimethamine, sulfadiazine, and folinic acid for 6 months. She remains blind, and has developed moderate psychomotor retardation.

**How could *Toxoplasma* infection have been prevented in this child?**

*Toxoplasma gondii* may be transmitted transplacentally to the fetus if the mother acquired toxoplasmosis during pregnancy. There is almost no risk of transplacental transmission if the mother was infected prior to conception; accordingly, women with positive IgG antibody tests for toxoplasmosis at the onset of pregnancy are not at risk for developing acute toxoplasmosis. Women with negative IgG antibody tests during pregnancy should avoid eating insufficiently cooked or uncooked meat and should avoid ingestion of soil and water or food that may be contaminated with cat feces.

Transmission occurs by a) ingestion of tissue cysts in raw or insufficiently cooked meat, especially lamb, pork, and wild game; b) accidental ingestion of food, water, or soil contaminated with cat feces that contain infective oocysts; c) transplacental passage of infective tachyzoites; d) transfusion of infected white blood cells or transplantation of an infected organ; and e) laboratory accidents.

**Prevention of toxoplasmosis is particularly important for uninfected (ie, seronegative) pregnant mothers, HIV-infected persons, and other immunocompromised patients:**

Avoid ingestion of raw or insufficiently cooked meat and poultry; cook meat to 160°F (71°C) or freeze to -4°F (-20°C). For more details on preventing toxoplasmosis, please see the **Suggested Food Safety Resources and Reading List** booklet.

Avoid ingestion of environmental oocysts by avoiding contact with cat litter, soil, water, and vegetables potentially contaminated with cat feces.

**Infection acquired by healthy persons** is usually asymptomatic or may lead to painless lymphadenopathy or a mononucleosis syndrome. Maternal infection is usually unrecognized.

**Disease in persons with depressed cellular immunity** (eg, persons with AIDS, transplant recipients, persons receiving immunosuppressants) usually is due to reactivation of latent infection but can result from acute infection. Toxoplasmosis in these persons leads to lethal meningoencephalitis, focal lesions of the CNS, and less commonly, myocarditis or pneumonitis. The clinical picture may include headache, seizures, mental status changes, focal neurologic signs, and aseptic meningitis. 30-40 percent of AIDS patients with IgG antibodies to *T. gondii* (indicating chronic latent infection) develop active toxoplasmosis unless they take preventive medication.

**Congenital infection** occurs when a previously uninfected mother develops infection during pregnancy. Infection prior to conception, demonstrated by specific IgG antibodies, in nearly all cases guarantees against infection of the fetus. However, transplacental transmission occurs from mothers whose prior infections reactivate when they receive immunosuppressant medications or develop AIDS. Congenital toxoplasmosis may result in abortion, stillbirth, mental retardation, and retinal damage. Recurrent toxoplasmic chorioretinitis in children and young adults is frequently the result of congenital infection that was asymptomatic at birth.