

TEACHING FILES (GRAND ROUNDS)

IS IT CONGENITAL TOXOPLASMOSIS?

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Clinical Problem

A 15 day old boy was referred in April 2016 to rule out congenital toxoplasmosis. His mother's obstetric history was complicated with loss of the first pregnancy at 8 months due to still birth, loss of the second pregnancy at 5 months due to spontaneous abortion, loss of the third and the fourth pregnancy at 5 and 6 weeks respectively. He was born premature at 35 weeks of gestation due to preeclampsia by lower segment cesarean section and spent 2 days in the neonatal intensive care unit (NICU). His birth weight was 2 kg and he was exclusively breast fed thereafter. On examination he was jaundiced without any organomegaly. His mother's serological TORCH titres are shown in the table 1. The child's toxoplasmosis IgG was 40.4 IU/ml and IgM was 0.018 IU/ml (negative). As maternal titres were positive in the first trimester and the newborn developed jaundice on day 15, his PCR for *T. gondii*, ultrasound of brain and eye examination to check for any manifestations of infection along with mother's toxoplasma PCR and eye examination was advised.

How to diagnose congenital toxoplasmosis?

Discussion

Toxoplasmosis is a disease caused by the intracellular protozoan parasite *toxoplasma gondii*.¹ It is acquired primarily through ingestion of cysts in infected undercooked meat (pork and lamb) or through contaminated soil, water and food.² Most immunocompetent individuals who contract the parasite do not develop symptoms, some may develop a non-specific flu like illness. However if *toxoplasma gondii* is acquired during pregnancy it can be transmitted to the fetus and results in congenital toxoplasmosis with other severe complications.¹ Transmission to the fetus most commonly occurs in women who acquire the primary infection during gestation however in rare cases, transmission can also occur in chronically infected women whose infection was reactivated because of their immunocompromised state. Infection in the mother is usually asymptomatic but few mothers may experience low grade fever, malaise,

lymphadenopathy. Rarely, they may complain of visual changes due to chronic chorioretinitis.² For untreated women, the transmission rate is approximately 25% in the first, 54% in the second and 65% in the third trimester.³ It may lead to spontaneous abortion, prematurity, stillbirth like in our patient's mother's obstetrics history. Congenital toxoplasmosis has a wide spectrum of clinic manifestations, but is subclinical in 75% of the infected newborns. Involvement of central nervous system (CNS) is a hallmark and the classic triad includes chorioretinitis, hydrocephalus and intracranial calcifications. Other manifestations include fever, jaundice, convulsions, microcephaly.³ Positive *Toxoplasma* IgM test is considered a marker of an acute infection. Usually IgM antibodies appear as early as two weeks after infection and may persist for years, while IgG antibodies peak six to eight weeks after the infection and decline over the next two years but remain positive. In some cases, such as in our patient's mother, IgM may persist for several months to years after acute infection thus making it challenging to differentiate between acute and chronic infection. To determine if positive IgM and IgG antibodies are due to a recent infection, chronic infection or a false positive result, IgG avidity test can be done in patients with positive IgM test. High titers suggest the patient has been infected for at least 3 to 5 months, however low titres are not necessarily diagnostic of an acute infection.^{4,5} Polymerase chain reaction (PCR) for *T. gondii* DNA in the amniotic fluid can be performed in women >18 weeks gestation with confirmed or strongly suspected recent infection or fetal infection. If PCR is positive, maternal treatment is started, if PCR is negative then the fetus is followed up with serial ultrasounds to detect fatal abnormalities.⁵ The diagnosis of congenital toxoplasmosis infection in the newborn is made based on serological testing, PCR, and other tests to evaluate extent of infection. Serological testing in the newborn includes *Toxoplasma* IgG, *T. gondii* specific IgM, *T. gondii* specific IgA and should be performed immediately after birth. *Toxoplasma* IgG in the newborn may reflect past or current infection in the mother, IgM may or may not be positive depending on the timing of maternal infection and IgA is not necessary for the diagnosis of congenital toxoplasmosis.⁶ Repeat testing in ten days can help make the diagnosis, IgM and IgA titers in a non-infected infant with rapidly decrease whereas will remain elevated in an infant who was

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Table 1: Mother's serological titres

	Sept 2012	Feb 2013	May 2013	Feb 2014	Aug 2015	Oct 2015	Nov 2015	Jan 2016	Feb 2016	April 2016
HSV IgG (IU/ml)	22.7			28.3						
HSV IgM (IU/ml)	0.22 (negative)			0.31 (negative)						
Rubella IgG (IU/ml)	193.1			224.1						
Rubella IgG (IU/ml)	0.42 (negative)			0.35 (negative)						
Toxo IgG (IU/ml)	47.4	196	120	26.3	18.92	72	13.74	13.4	13.04	11.96
Toxo IgM (IU/ml)	1.64 (positive)	1.43 (positive)	1.29 (positive)	1.15 (positive)	0.93 (positive)	0.59 (negative)	0.95 (positive)	0.86 (negative)	0.79 (negative)	0.89 (negative)
CMV IgG (IU/ml)	1292.6			1180.7						
CMV IgM (IU/ml)	0.25 (negative)			0.17 (negative)						

Note: HSV - Herpes simplex, Toxo - toxoplasmosis, CMV - cytomegalovirus

infected in utero. Maternal IgG falls by 50 % every month to undetectable values by 6-12 months, in contrast infected infants will have elevated toxoplasma gondii IgG levels beyond a year.⁶

In our patient, toxoplasma PCR was negative in both child and mother and repeat toxoplasma IgG in the child after 4 weeks showed a 50% fall in the titres. Ultrasound of the skull and eye examination were normal.

Compliance with Ethical Standards

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Conflict of Interest: None

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