International Journal of Health Systems and Medical Sciences

ISSN: 2833-7433 Volume 2 | No 4 | April -2023



Modern Views on the Ways of Transmission of Torch Infection to the Body and Its Effect on the Human Body

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Summary: The term TORCH complex or TORCHes infection includes toxoplasmosis, others (syphilis, hepatitis B), rubella, cytomegalovirus, herpes simplex. These are caused by toxoplasma gondii, treponema pallidum, hepatitis B virus, rubella virus, cytomegalovirus, and herpes simplex virus (HSV), respectively. Human immunodeficiency virus and Zika virus are sometimes included in this grouping. This activity highlights the role of interprofessional team in evaluation and management of patients with TORCH complex. The term TORCH complex or TORCHes infection refers to the congenital infections of toxoplasmosis, others (Syphilis, Hepatitis B), rubella, Cytomegalovirus (CMV), and herpes simplex. These are caused by Toxoplasma gondii, Treponema pallidum, Hepatitis B virus, Rubella virus, cytomegalovirus, and herpes virus simplex (HSV) viruses respectively. Other pathogens associated with congenital infections include human immunodeficiency virus (HIV), parvovirus, and varicella virus.

Keywords: Toxoplasmosis, congenital rubella syndrome, herpes simplex virus, cytomegalovirus, syphilis.

It is the intrauterine transmission of these infections to the fetus which produces multiple symptoms when the child is born. Maternal risk factors include lapsed immunizations, sexually transmitted infections, and animal exposures during pregnancy. The timing of maternal infection if a key epidemiologic factor because fetal damage usually depends on the gestational age. With the exception of HSV, infections during the first trimester have the worst outcome.

The TORCH infections include causative organisms *Toxoplasma gondii*, rubella virus, cytomegalovirus, HSV 1 and 2, hepatitis B virus, HIV, and others like syphilis, parvovirus, and varicella. Transmission of the pathogens may occur prenatally by the transplacental route, perinatally by blood or vaginal secretions. Postnatal infections tend to be less impactful. Others, such as HIV, hepatitis B, and syphilis, can be transmitted via sexual contact to a susceptible mother. Rubella and varicella can be prevented by properly immunizing mothers.

Approximately 2% to 3% of all congenital anomalies are attributed to perinatal infections. Initial evidence of infection may be seen during the intrauterine period, at birth, in infancy, or not even until years later. Intrauterine manifestations of congenital infections include abnormal growth parameters or developmental abnormalities. The infected newborn infants may show abnormal growth, developmental anomalies, or multiple clinical and laboratory abnormalities.

Many of the clinical syndromes for those viruses that present in the immediate neonatal period overlap with each other. They usually cause a rash, which can be maculopapular, petechial (blueberry muffin rash), or purpuric. Microcephaly, sensorineural hearing loss (particularly with



CMV), and chorioretinitis may be present. Hepatosplenomegaly and cardiac anomalies are also frequent findings.

The incidence of maternal CMV and toxoplasmosis are 2 to 10 per 1000 births. Rubella is common in countries where mothers are unvaccinated but only occurs in the United States in cases of imported disease after universal immunization. Humans are the natural hosts for the herpes virus, and the newborns usually get HSV-2 as it predominantly causes genital infections. Risk factors for toxoplasmosis include exposure to cats and the ingestion of improperly prepared foods such as undercooked meat or unpasteurized dairy products. Raw vegetables served in the restaurant probably caused toxoplasmosis in Brazil.

Toxoplasma gondii oocysts transmission occurs by ingesting the infected tissue or inhaling the fecal particles. Transplacental transmission causes congenital toxoplasmosis. This is most commonly occurs in the third trimester of pregnancy. However, earlier the infection, more severe will be the congenital malformations.

Syphilis is transmitted through the placenta or vertically in the birth canal. The transmission rate is more than 80% in recently infected mothers.

Rubella is transmitted to the mother by aerosols and to the fetus through the placenta.

CMV transmits to the mother by blood transfusion, organ transplants, or most commonly through the mucus membrane exposure. It then passes either through the placenta, birth canal, or breast milk to the fetus or neonate. CMV infection rates in primary infection have long been proposed to be greater than secondary infection, but there has been some recent analysis that this may not be as significant as previously thought.

HSV transmits to the mother by sexual contact and later to the fetus via either ascending infection or exposure during parturition. Maternal primary infection during the third trimester has the highest percentage of neonatal infection.

Secondary reactivation of HSV is 10 to 30 times less likely to result in transmission to the infant.

HIV transmission to infants can occur either in transplacentally in utero, during parturition, or via post-natal maternal exposures like breast milk.

Maternal history is a key area of investigation for patients who have a concern for congenital infections. The history of a febrile illness with or without rashes and poor maternal weight gain could raise concern for clinicians. Furthermore, fetal abnormalities such as intracranial calcifications may be detected in routine, or specific maternal testing may occur. Fetal loss can occur, particularly with infections during the first trimester.

The history and physical findings for each individual TORCH pathogen are listed below: In general, a physical exam may reveal rashes, low birth weight, microcephaly, findings suggestive of cardiac abnormalities (murmurs), chorioretinitis and cataracts, and intracranial calcifications.

Toxoplasmosis: The primary manifestations of congenital toxoplasmosis include intrauterine growth restriction and low birth weight, hepatosplenomegaly, jaundice, chorioretinitis, intraparenchymal calcifications, and anemia. Less commonly, petechiae, hydrocephalus, and microcephaly can be found.

Congenital rubella syndrome: It includes low birth weight, hepatosplenomegaly, cataracts, congenital heart disease (patent ductus arteriosus, and ventricular septal defect), and a petechial rash. Congenital sensorineural hearing loss is very common.

Herpes simplex virus: HSV rarely presents with *in utero* infection but instead presents due to perinatal exposure. Therefore, clinical manifestations normally will present ten to twenty-one days after infection. There are three major manifestations: Skin-eye-mucous membranes (SEM), central nervous system (CNS), and disseminated disease. All will often present with fever in the neonatal period. The disseminated disease will present earliest after approximately one week of age. These children will present with a sepsis-like syndrome with skin lesions to include vesicles, hypotension,



hepatosplenomegaly, and lethargy. These patients often have evidence of meningoencephalitis. SEM disease is thought to be limited to a rash, normally vesicular, and is often noted in areas of trauma such as fetal scalp electrodes or the use of forceps with delivery. CNS disease is more likely to present with lethargies or perhaps seizures. All patients should be evaluated for evidence of disease, including the CNS.

Cytomegalovirus: CMV is the most common congenital infection. It will present with intrauterine growth restriction and low birth weight, hepatosplenomegaly, jaundice, paraventricular calcifications, cataracts, and sensorineural hearing loss and bone marrow suppression that will present with thrombocytopenia and anemia. Patients often have a petechial rash at birth.

HIV: Patients with congenital HIV rarely have any evidence of outward manifestations at birth. They may have a low birth weight and hepatosplenomegaly at birth.

Syphilis: In utero, there may be fetal loss or *hydrops fetalis*. In the neonatal period, children with primary syphilis may present with cutaneous lesions on the palms and soles, hepatosplenomegaly, jaundice, inflammation of the umbilical cord (funisitis) and discharge from the nose (sniffles). Periostitis may be found on x-rays of the bones. Late findings include frontal bossing, high palatal arch, sensorineural hearing loss, a saddle nose, perioral fissures, and Hutchinson teeth.

The TORCH titer is a test that is often run in this setting. Basically, it is usually a panel of IgG tests for the pathogens noted. It may provide some useful insight into whether a mother has been infected if high titers are detected; it is not that useful in making a definitive diagnosis of any pathogen associated with congenital infections. Therefore, the investigation of each pathogen is warranted if the clinical syndrome is suggestive of that disease. The evaluation of each illness is noted in the following passages.

Toxoplasmosis: In newborns with a concern for congenital toxoplasmosis, evaluation should include laboratory testing, consultations, and radiologic studies. Due to the possibility of ocular involvement, an ophthalmologist should be consulted to assess for possible chorioretinitis. Neuroimaging studies should be conducted to assess intracranial calcifications and/or hydrocephalus. Laboratory testing can be a bit complicated. The most sensitive and specific testing includes a mixture of tests to assess for IgA, IgG, and IgM. Most experts recommend the use of a reference laboratory that can assess the newborn with IgG (Dye test), IgM ISAGA, and IgA ELISA. If the child has not been born, maternal testing can also be conducted, which may include either an Avidity panel or a differential agglutination test depending on the week of pregnancy.

Congenital rubella syndrome: If concerns exist for this infectious syndrome, consultations with specialists and laboratory testing should occur. In order to investigate for clinical stigmata, ophthalmology (cataracts, glaucoma) and cardiology (patent ductus arteriosus (PDA), ventriculoseptal defect (VSD), and pulmonary artery stenosis) should be consulted early in the patient's course. As hearing loss is very common, audiology consultation with hearing tests should be conducted in all patients. Laboratory testing could include attempts to culture the virus from the nasopharynx or the assessment for IgM in the newborn.

Congenital cytomegalovirus: Evaluation of the being considered for the diagnosis of congenital CMV, should begin with confirmation of CMV infection. Infection is usually confirmed by the isolation of the virus within the first month of life. While any sterile site can be used, the urine is the most common source of isolation. Traditional viral culture or PCR testing is sufficient. CMV has been found in the blood and CSF as well. After confirmation, subspecialty consultation with ophthalmology (cataracts, chorioretinitis) and audiology (hearing loss) are essential. The workup usually includes neuroimaging to assess for the presence or absence of intracranial, periventricular calcifications. Laboratory evaluation should include assessment for liver transaminitis as well as liver function and bone marrow suppression by obtaining chemistries, liver function tests, PT/PTT, and a complete blood count. Herpes simplex virus infections: Any child who presents with a concern for neonatal HSV should be aggressively evaluated.



They should undergo a complete sepsis evaluation to include a lumbar puncture. It is recommended that swabs from the mouth, nasopharynx, conjunctivae, and anus. Be obtained for HSV culture and PCR. If skin vesicles are present, they should be unroofed and sent for culture and PCR as well. CSF and whole blood should also be sent for HSV culture and PCR. Measurement of liver transaminases should be conducted as they are an early clue that disseminated disease may be present. Further evaluation and consultation may depend on the type of infection the child has to include consultation with ophthalmology, neurology, and audiology. Hearing tests must be conducted as they may help with decision-making with regard to treatment. HIV: Patients with concern for mother-to-child transmission of HIV should be evaluated. Children should have a PCR test sent at birth as infants will receive transplacental antibodies from their mothers. The first PCR can be sent within the first two days of life to determine if in utero infection occurred. Children are considered HIV negative if they have two tests conducted, and negative, after two weeks of age with one after four weeks of age. Alternatively, one negative PCR obtained after eight weeks is sufficient. Children should not breastfeed. Obtaining a complete blood count and baseline chemistries to include renal and hepatic function is also warranted. Complete guidance can be found at the U.S. Department of Health and Human Services.

Syphilis: The evaluation of a child with congenital syphilis depends on whether the mother was diagnosed during pregnancy and properly treated or not. All children should have a rapid plasma reagin (RPR) to be compared to the mother's RPR titer. If a mother is inadequately treated during pregnancy or the child has evidence of an elevated RPR with findings consistent with syphilis, the child needs a robust evaluation. The evaluation includes a complete blood count, CSF examination for cell count, protein, and venereal disease research laboratory (VDRL). Additionally, long bone radiographs, neuroimaging, ophthalmologic examination, liver function testing, and hearing tests can be conducted.

Here are the management considerations for TORCH infections:

- There are no programs on a large scale that offer both maternal or neonatal screening to identify infection in mothers and infants. No vaccines are present to prevent infection, and no efficacious and safe therapies are available for the treatment of maternal or fetal CMV infection. In some setups, gancyclovir is being given.
- ➢ For toxoplasmosis, observational studies have demonstrated an effective reduction in transplacental transmission and/or severity of clinical manifestations in symptomatic infants. The two regimens often used are spiramycin (fetal prophylaxis preventing intrauterine infection) and combined pyrimethamine/sulfadiazine/ folinic acid (treatment of evolving fetal infection).
- Congenital rubella, once developed, cannot be treated. But it is the most common vaccinepreventable neonatal disease. A single dose of rubella vaccine to mother can produce life long immunity. Mothers should have their immunity checked at the beginning of a pregnancy.
- Patients with neonatal HSV should be treated aggressively. Clinical trials have demonstrated that high-dose intravenous acyclovir (60 mg/kg/day intravenously divided three times daily) for acute therapy. The length of this therapy varies from fourteen to twenty-one days depending on the severity of disease (10 for SEM/21 for disseminated and CNS) followed by long-term oral acyclovir suppressive therapy (300 mg/m/dose, given orally three times daily for six months is best for the management of neonatal herpes infection. This work has dramatically reduced morbidity and mortality from neonatal HSV. Complete blood counts and renal function should be monitored. Dosing should be adjusted as the infant grows.
- Patients with symptomatic cytomegalovirus infections should be treated with ganciclovir and valganciclovir. The primary reason for this therapy is to preserve hearing. Neonates with symptomatic congenital CMV disease with or without central nervous system (CNS) involvement show better outcomes at two years when treated with oral valganciclovir (16 mg/kg/dose, administered orally twice daily) for six months. Dosing should be adjusted as the infant grows.



- The treatment of HIV to prevent mother-to-child transmission depends on whether the mother was treated with viral suppression during the pregnancy. Therapy for children born to wellcontrolled mothers should include the treatment of the infant with zidovudine (4 mg/kg, twice daily) for the first 4 to 6 weeks of life for term children. Multiple drug regimens are recommended for children whose mother was not on antiretroviral therapy during pregnancy.
- Syphilis must be diagnosed and treated immediately. Expectant mothers should be tested during pregnancy and, if positive, treated. Treatment of the neonate will depend on whether the mother was treated appropriately during pregnancy. Normal neonates born to mothers adequately treated during pregnancy and greater than four weeks before delivery or have a non-reactive RPR but were born to mothers not treated properly should receive a single intramuscular injection of benzathine penicillin G (50,000 U/kg), although no evaluation is required or recommended. Infants with serologic tests that confirm congenital syphilis should receive aqueous penicillin g (200,000 to 300,000 units/kg/day IV, administered as 50,000 units/kg every 4 to 6 hours for ten days). If the child has a negative evaluation for clinical and laboratory evidence of syphilis, treatment with up to 3 weekly doses of benzathine penicillin G (50,000 U/kg IM) can be considered.

As many of these illnesses have similar manifestations, they are often all considered as a possible diagnosis when a child presents with signs and symptoms suggestive of congenital infection. Therefore, in a child who presents small for gestational age with additional clinical findings such as a rash or heart murmur or ocular findings, all of the TORCH complex pathogens should be considered. In addition to those mentioned, the new pathogen Zika virus can cause significant disease in newborns. In particular, the virus causes CNS disease that is significant and can present with intracranial calcifications. Parvovirus B19 infection can also cause fetal *hydrops fetalis* and can present with profound bone marrow suppression.

Maternal factors such as preeclampsia, hypertension, smoking, drug and medication use, and anemia may contribute to growth problems. Additionally, many metabolic and genetic syndromes can present in a similar manner to a TORCH infection. These may range from common issues like hypothyroidism to much more complex and rare genetic syndromes.

The prognosis for congenital infections will vary depending on the severity of the initial presentation. For toxoplasmosis, there may be findings at birth, such as intracranial calcifications and chorioretinitis, which may suggest a poor prognosis with seizures and developmental delay likely. Unfortunately, there may be some long-term problems detected, such as school dysfunction, hearing and visual issues, and gross motor problems that require close monitoring. Patients with congenital rubella syndrome continue to have a poor prognosis with multiple organ systems impacted to include cardiac malformations, hearing loss, cataracts, and brain anomalies Long-term followup portends a poor prognosis, particularly in children with cardiac disease.

The prognosis of patients with congenital cytomegalovirus is variable. Some patients with a congenital infection have extremely good outcomes and are relatively asymptomatic. On the other hand, children with CNS disease at presentation are at high risk for sensorineural hearing loss and developmental delays. If valganciclovir can be administered to symptomatic newborns in a timely manner, it has shown value.

Patients with neonatal HSV infections will have an outcome that is dependent on the presentation as well. With the advent of the aggressive use of acyclovir in the neonatal period plus the use of suppressive acyclovir for six months, long-term outcomes have improved for neurologic outcomes. The outcome for patients presenting with the disseminated disease remains grim. Patients with syphilis will have good outcomes as long as they are recognized and diagnosed at birth and receive the correct and prompt therapy.



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