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Focus on Diagnosis : Congenital Infections (TORCH)

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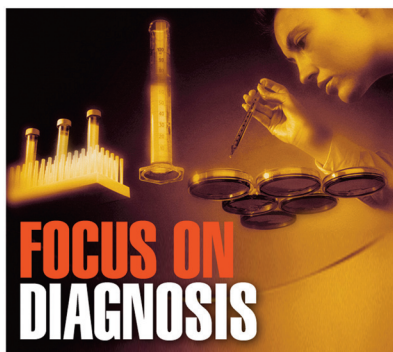
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Abbreviations

CDC:	Centers for Disease Control and Prevention
CMV:	cytomegalovirus
CNS:	central nervous system
CSF:	cerebrospinal fluid
HBsAg:	HBV surface antigen
HBV:	hepatitis B virus
HIV:	human immunodeficiency virus
HSV:	herpes simplex virus
IgG:	immunoglobulin G
IgM:	immunoglobulin M
IUGR:	intrauterine growth restriction
PCR:	polymerase chain reaction
VZV:	varicella-zoster virus

Congenital Infections (TORCH)

Jeannine Del Pizzo, MD*

Introduction

TORCH is an acronym for a group of congenitally acquired infections that may cause significant morbidity and mortality in neonates. TORCH stands for the following:

Toxoplasmosis

Other: syphilis, hepatitis B, varicella-zoster virus (VZV), human immunodeficiency virus (HIV), parvovirus B19, enteroviruses, lymphocytic choriomeningitic virus

Rubella

Cytomegalovirus (CMV)

Herpes simplex virus (HSV)

Some experts consider the acronym TORCH outdated, largely due to the growing number of infections listed in the “other” category. However, use of the acronym may aid in remembering the causative organisms.

While each of the congenital infections possesses distinct clinical manifestations and sequelae, some of these infections share characteristics. It is important to think of one or more of these infections when a neonate presents with microcephaly, intracranial calcifications, rash, intrauterine growth restriction (IUGR), jaundice, hepatosplenomegaly, elevated transaminase concentrations, and thrombocytopenia. However, many congenital infections may be silent at birth, with symptoms manifesting years later.

Also, some agents, such as VZV, are associated with infection in utero as well as infection during or after delivery, with differing effects depending on the time of infection. This article includes discussion of

true congenital infections that are present at the time of delivery as well as some transmitted during or after delivery.

When a congenital infection is suspected, a thorough maternal history should be obtained, including immunization status, past and recent infections, and exposures. A careful physical examination of the neonate is vital because different clinical findings may indicate a specific diagnosis. Diagnostic testing should be directed only toward those infections that fit the clinical and historical picture. The sometimes employed TORCH titers should *never* be used as a single test to diagnose or rule out a congenital infection.

Toxoplasmosis

The causative agent in toxoplasmosis is the protozoan and obligate intracellular parasite *Toxoplasma gondii*.

ROUTE OF INFECTION. *T gondii* is spread via the fecal–oral route. Oocysts of *T gondii* are excreted via cat feces and ingested by humans through inadequately cooked meat, contaminated water and soil, and unpasteurized goat milk. Oocysts remain infectious for variable amounts of time, and after excretion can endure in damp soil for as long as 18 months.

CLINICAL MANIFESTATIONS. Toxoplasmosis is transmitted to the fetus during a mother’s primary infection or if the mother is immunocompromised and has chronic infection. The risk of fetal transmission during a maternal infection increases with gestational age. However, the earlier in pregnancy the fetal infection occurs,

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the more likely it is to be severe. Transmission during the first trimester may result in death, or if the fetus survives, frequently it will demonstrate ophthalmologic and central nervous system (CNS) sequelae. Transmission in the second trimester causes multiple effects, including the “classic” triad of hydrocephalus, intracranial calcifications, and chorioretinitis, as well as jaundice, hepatosplenomegaly, anemia, lymphadenopathy, microcephaly, developmental delay, visual problems, hearing loss, and seizures. Fetuses infected in the third trimester often are asymptomatic at birth.

DIAGNOSIS. Definitive diagnosis of toxoplasmosis is made by organism isolation from the placenta, serum, and cerebrospinal fluid (CSF); however, organism isolation can be challenging and is not generally available. A positive maternal enzyme-linked immunosorbent assay suggests the diagnosis but does not clinch it. Other studies that can build a case for congenital toxoplasmosis are ophthalmologic examination evaluating for chorioretinitis, a computed tomography scan of the head looking for calcifications, and CSF studies demonstrating elevated protein and pleocytosis. An infant’s toxoplasmosis immunoglobulin G (IgG) titer will be positive if the mother was infected; however, this finding does not prove the infant’s infection. If the infant is infected, the IgG concentration will continue to be elevated after maternal-derived antibody concentration decreases, at approximately 6 months to 1 year of age.

TREATMENT. Congenital toxoplasmosis is treated with pyrimethamine, sulfadiazine, and leucovorin for 1 year. Infants who receive treatment have improved hearing loss, al-

though they remain at risk for recurrent chorioretinitis.

Syphilis

Syphilis is caused by infection with the gram-negative spirochete *Treponema pallidum*.

ROUTE OF INFECTION. *T pallidum* is spread through direct contact with a spirochete-containing lesion, sexually, or transplacentally.

CLINICAL MANIFESTATIONS. The majority of infants born with congenital syphilis are asymptomatic at birth. The time of onset of clinical manifestations is used to classify early congenital syphilis and late congenital syphilis. The former presents at 1 to 2 months of age with development of one or more of the following: maculopapular rash, snuffles, generalized lymphadenopathy, hepatomegaly, thrombocytopenia, anemia, meningitis, chorioretinitis, pneumonia alba, and osteochondritis. Late congenital syphilis presents after 2 years of age with signs such as Hutchinson teeth (small teeth with an abnormal central groove), mulberry molars (bulbous protrusions on the molar teeth resembling mulberries), hard palate perforation, eighth nerve deafness, interstitial keratitis, bony lesions, and saber shins (due to chronic periosteitis).

DIAGNOSIS. The definitive diagnosis of congenital syphilis is demonstration of spirochetes under dark-field examination or direct fluorescent antibody in fluid from a lesion, the placenta, or the umbilical cord. However, because this testing is not always available, a presumptive diagnosis is made using nontreponemal and treponemal tests. Nontreponemal tests such as the venereal disease research laboratory test and rapid plasma reagin are used for

screening and monitoring treatment of the disease. Treponemal tests such as the fluorescent treponemal antibody absorption test or *T pallidum* particle agglutination are used to confirm diagnosis. Treponemal tests are not used alone due to false positives that may occur with other infections such as Lyme disease, yaws, pinta, and leptospirosis. A false-negative result also may occur because of an overwhelming quantity of antibodies, which is called the prozone effect.

The recommendations for screening mothers and infants are established in the United States by the Centers for Disease Control and Prevention (CDC). The best way to determine an infant’s risk for congenital syphilis is to know the mother’s status. The CDC recommends that all pregnant women be screened for syphilis with a nontreponemal test and, if positive, receive a confirmatory treponemal test. Infected pregnant women should be treated with penicillin G and followed up with both a nontreponemal test and treponemal test 4 weeks after treatment and then monthly.

An infant should be tested with the same nontreponemal test as the mother if the mother has a nontreponemal titer that increased fourfold; had a positive treponemal test without documented treatment; had a positive treponemal test not treated with penicillin; had a positive treponemal test and was treated less than 1 month before delivery; or if the infant has signs of congenital syphilis. If the infant’s nontreponemal titer is more than fourfold higher than the mother’s or if there is any clinical finding consistent with congenital syphilis, the infant must be treated and undergo a venereal disease research laboratory test of CSF, liver function tests, complete blood count, and long bone radiographs.

TREATMENT. The treatment of choice for *T pallidum* infection at any age is penicillin G.

Hepatitis B

Hepatitis B virus (HBV) is a DNA virus that hails from the hepadnavirus family.

ROUTE OF INFECTION. Transmission of HBV occurs after exposure to contaminated blood or body fluids. Transplacental transmission is a rare occurrence. Most neonates are infected during delivery through exposure to maternal blood during delivery.

CLINICAL MANIFESTATIONS. The majority of neonates who acquire perinatal HBV infection are asymptomatic. Rarely they may demonstrate signs consistent with hepatitis including jaundice, thrombocytopenia, elevated transaminase concentrations, and rash.

COMPLICATIONS. The risk of morbidity of HBV is inversely proportional to the gestational age at the time of initial infection. Children infected at a younger gestational age have a higher risk of progressing to chronic infection and disease. As the gestational age at the time of acute infection increases, the risk of chronic infection decreases. Progression of HBV infection to chronic disease is worrisome because 25% of children chronically infected with HBV will develop hepatocellular carcinoma or cirrhosis.

DIAGNOSIS. It is essential to know the mother's status to determine if an infant has been exposed to HBV. In the United States, pregnant women are screened for HBV surface antigen (HBsAg). The presence of this antigen signifies that the mother has an acute or chronic infection. Infants

born to mothers with a positive HBsAg should receive HBV vaccine and hepatitis B immune globulin within 12 hours of birth. These infants should then complete the HBV vaccine series with two more additional immunizations per the CDC's recommended schedule, as well as undergo HBsAg and anti-HBs testing after 9 months of age. If the mother's HBV status is unknown at the time of delivery, she should be tested for HBsAg immediately. The infant should receive the HBV vaccine while awaiting the mother's results. If the mother's HBsAg is negative, no further treatment is required. However, if it is positive, the infant should receive hepatitis B immunoglobulin within 7 days of birth. Preterm infants exposed to HBV and weighing less than 2 kg should be treated as outlined above with one exception: the dose of HBV vaccine received within 12 hours of birth should not be counted toward completion of the vaccine series. They should begin the usual three-dose vaccine series at 1 month of age.

TREATMENT. There is no treatment for acute HBV. Lamivudine is approved for treating chronic HBV infection in children 2 years of age and older.

Varicella-Zoster Virus

VZV is a member of the herpesvirus family.

ROUTE OF INFECTION. VZV is transmitted through contact with fluid from vesicles or through airborne contact with respiratory secretions. Congenital varicella is acquired transplacentally.

CLINICAL MANIFESTATIONS. A maternal varicella infection transmitted to the fetus under 20 weeks' gestation results in fetal demise or devel-

opment of anomalies, including ophthalmologic malformations, cutaneous scarring, limb hypoplasia, and damage to the CNS. If maternal infection and subsequent fetal transmission occur later in gestation, the infant may develop the typical signs of varicella after birth or may be asymptomatic but have a risk of developing zoster later on. Perinatal infection occurring several days before or after birth may result in neonatal death.

DIAGNOSIS. Varicella should be suspected in a mother who demonstrates the classic signs of varicella: a prodromal illness with dewdrop lesions developing in crops that form crusts. In an infant who presents with typical vesicular lesions, a polymerase chain reaction (PCR) or direct fluorescent antibody of the fluid can be performed. Acute and convalescent immunoglobulin M (IgM) titers can diagnose in hindsight but will not identify acute disease.

TREATMENT. Pregnant women who acquire varicella may be treated with acyclovir. Pregnant women who are exposed to varicella can be given prophylactic varicella-zoster immunoglobulin or immunoglobulin intravenous. Infants born with congenital varicella should be treated with acyclovir and varicella-zoster immunoglobulin.

Human Immunodeficiency Virus

HIV is an RNA virus belonging to the Retroviridae family. There are two types of HIV: HIV-1 and HIV-2, with HIV-1 being the predominant virus found in the United States. Humans are the only known hosts of HIV-1 and HIV-2.

ROUTE OF INFECTION. HIV is spread parenterally through exposure

to infected blood, semen, vaginal and cervical secretions, contaminated needles or sharp objects, contaminated blood transfusions, and vertically. HIV can be transmitted to the infant at any time during pregnancy: transplacentally, during labor and delivery, or after birth through breastfeeding. The highest risk of neonatal infection occurs during delivery with exposure to maternal blood.

CLINICAL MANIFESTATIONS. Neonates suspected of having perinatally acquired HIV will be asymptomatic and have normal-for-age lymphocyte counts. As the infection evolves, T-cell function declines. Depending on T-cell counts, various opportunistic infections can take hold, such as encapsulated bacteria, *Pneumocystis jirovecii*, VZV, CMV, and HSV, among others.

DIAGNOSIS. The American Academy of Pediatrics and CDC recommend routine HIV-1 testing for all pregnant women in the United States. Knowledge of the maternal infection can prompt measures to decrease transmission, including HIV drug prophylaxis, cesarean section before rupture of membranes for women with a viral load of greater than 1,000 copies/mL at full term delivery, avoidance of breastfeeding, and early detection in the infant. HIV serum DNA and RNA assays have low sensitivity shortly after birth. Either HIV-1 DNA or RNA PCR should be analyzed in the infant born to an HIV-infected mother at the following times: 14 to 21 days after birth, 1 to 2 months of age, and 4 to 6 months of age. An infant is considered uninfected if he or she meets either of the following laboratory criteria: 1) two negative HIV-1 DNA or RNA assays, one obtained after 1 month of age and the other at

4 months of age or older, or 2) two negative HIV-1 antibody tests from separate specimens obtained at 6 months of age or older. Some practitioners may follow antibodies until after 18 months of age because maternally derived antibodies rarely persist beyond this age.

TREATMENT. Infants suspected of having HIV infection are started on zidovudine until 6 weeks of age. Infants with confirmed HIV infection are started on further antiretroviral treatment.

Parvovirus B19

Parvovirus is a single stranded DNA virus.

ROUTE OF INFECTION. Parvovirus is spread through respiratory tract secretions, exposure to contaminated blood, and transplacentally.

CLINICAL MANIFESTATIONS. Infants who are infected with parvovirus are at risk for hydrops, pleural and pericardial effusions, IUGR, and death. Infection during the first half of pregnancy confers the greatest risk to the fetus. Infected infants demonstrate the extremes of outcomes with almost no middle ground: either life-threatening infection or no residua.

DIAGNOSIS. If congenital parvovirus is suspected, an IgM titer should be obtained from infant serum.

TREATMENT. Treatment is limited to supportive care. There is evidence that intravenous immunoglobulin may be beneficial.

Rubella

Rubella, also known as German measles, is a member of the Togaviridae family.

ROUTE OF INFECTION. Rubella is spread through contact with respiratory secretions (both direct and droplet) and transplacentally.

CLINICAL MANIFESTATIONS. Signs at birth of congenital rubella include “blueberry muffin” rash (dermal erythrocytosis), lymphadenopathy, hepatosplenomegaly, thrombocytopenia, interstitial pneumonitis, radiolucent bone disease, and IUGR.

COMPLICATIONS. As with toxoplasmosis, the earlier during gestation infection occurs, the more severe the disease will be. Fetal transmission during the first trimester often results in readily apparent sequelae at birth, such as congenital defects. In contrast, infection after 12 weeks may have no clinical manifestations but is more likely to result in future hearing loss and visual problems. Congenital rubella can affect multiple systems. Eye problems associated with congenital rubella include microphthalmos, pigmentary retinopathy, cataracts, and congenital glaucoma. Cardiac manifestations include peripheral pulmonic stenosis and patent ductus arteriosus. Endocrinopathies can occur, the most common being diabetes mellitus. Neurologic sequelae include developmental delay, encephalitis, and sensorineural hearing loss.

DIAGNOSIS. A positive infant rubella IgM titer is indicative of recent infection; however, this test can be complicated by both false positives and false negatives. The virus can be isolated in culture from certain body fluids, including blood, urine, CSF, and oral and nasal secretions. The diagnosis of congenital rubella can be established by persistently elevated or rising IgG titers over time.

TREATMENT. Treatment is limited to supportive care.

Cytomegalovirus

CMV is classified as part of the herpesvirus family and is the most common congenital infection in the United States. The prevalence of congenital CMV infection in live-born infants in industrialized nations is estimated to be 0.5% to 1%.

ROUTE OF INFECTION. CMV can be transmitted to an infant during pregnancy (transplacental transmission), during delivery (via contact with infected genital tract secretions), or postnatally (via ingestion of contaminated human milk or direct contact with other body fluids such as urine and saliva). Mothers who have been exposed to CMV before pregnancy are still at risk for transmitting the infection to the fetus by way of reactivation or infection with a new strain. However, maternal infection before pregnancy and subsequent development of immunity significantly decrease the risk of congenital CMV.

CLINICAL MANIFESTATIONS. The majority of neonates born with congenital CMV are asymptomatic at birth. Symptomatic infants with CMV may have IUGR, microcephaly, periventricular calcifications, hepatosplenomegaly, jaundice, thrombocytopenia, and retinitis. Some infants may demonstrate hypotonia, lethargy, and poor suck. It is important to note that preterm infants may present as if they have sepsis (apnea, bradycardia, intestinal distention, and poor color). The risk of fetal morbidity is increased when the mother has a primary infection during pregnancy, especially during the first trimester. Postnatal infection via ingestion of human milk causes no

clinical sequelae, likely due to protection from maternal antibody.

COMPLICATIONS. Whether an infant is symptomatic at birth can help predict future morbidity. Approximately one half or more of symptomatic neonates will develop CNS sequelae, including retinitis, sensorineural deafness, and developmental delay. This incidence is in contrast to asymptomatic infants, fewer than 20% of whom will develop CNS sequelae. There is an increased likelihood of developmental delay when infants manifest chorioretinitis, microcephaly, and intracranial calcifications.

DIAGNOSIS. Congenital CMV is diagnosed by demonstration of the virus in body fluids such as urine or pharyngeal secretions in the first 3 weeks after birth. After 3 weeks of age, it is difficult to determine whether the infection was congenital or postnatal. Virus can be detected in body fluids by culture, rapid centrifugation-enhanced culture (requires 24 h incubation), or PCR. Antibodies are not useful in diagnosing congenital CMV because neonatal IgG indicates maternal infection but does not specify when it occurred, and assays for IgM have poor sensitivity and specificity.

TREATMENT. There is no approved agent for the treatment of congenital CMV. Treatment with ganciclovir has been shown to improve both hearing loss and neurodevelopmental outcomes.

Herpes Simplex Virus

HSV 1 and 2 are double-stranded DNA viruses from the Herpesviridae family.

ROUTE OF INFECTION. HSV is transmitted primarily through direct

contact with infected lesions or mucosa. Neonates most often acquire the infection while passing through an infected vaginal canal during birth or from the virus ascending after rupture of membranes. Fetal transmission via the placenta occurs only rarely and can result in congenital anomalies and death. Primary maternal infection during pregnancy, especially in the third trimester, imparts the greatest risk to the fetus. Postnatal infection can occur from infected caregivers kissing or touching the infant.

CLINICAL MANIFESTATION. Infants with congenitally acquired HSV infection usually will present in the first 6 weeks after birth. Early signs may be vague and include irritability, poor feeding, lethargy, skin vesicles, fever, and seizures. There may be no signs at all. It is essential to have a high degree of suspicion, because there is a known maternal history of herpes in only 12.5% of infants diagnosed with congenital HSV. The manifestations of neonatal herpes can be classified in three ways: primarily skin, eyes, and mucosal involvement (SEM disease); primarily CNS disease; and disseminated disease with multiple organ involvement. However, these categories are not exclusive of each other and infants can have signs from more than one. Infants given a diagnosis of SEM disease also may have occult CNS infection.

COMPLICATIONS. Untreated, neonatal HSV infection causes high morbidity and mortality. If treated, the infants with SEM disease have the best prognosis with respect to both survival and neurologic development; however, about one half will suffer recurrent skin outbreaks. Treated infants who have CNS disease have a good prognosis for sur-

vival but suffer significant neurologic sequelae.

DIAGNOSIS. An infant is considered infected with herpes if any of the following tests are positive: serum HSV IgM, HSV PCR of the CSF, or HSV culture of a lesion or any other mucosal surface. Because of its high sensitivity (ranging 75% to 100%), HSV PCR is the test of choice for evaluation of the CSF. It is important to note that the CSF PCR may be negative the first 5 days of illness. If HSV remains strongly suspected, despite an initial negative result, the CSF PCR should be repeated. For SEM disease, HSV culture of a cutaneous or mucosal lesion is the test of choice. Neither PCR nor culture of the blood has a particularly high sensitivity. HSV serologies may be helpful; however, not all patients seroconvert initially and false negatives may persist up to 2 weeks into the illness. Maternal HSV IgG antibodies also may be present in the infant.

TREATMENT. The preferred treatment for neonatal herpes infection is intravenous acyclovir. Treatment with acyclovir improves mortality rates for all infants who have neonatal herpes and neurologic development in those who have SEM and disseminated disease.

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