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CE 2.5 HOURS Continuing Education

The ABCs of CMV

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ABSTRACT

Most infants exposed to cytomegalovirus (CMV) in utero will not be symptomatic; however, infants born with symptomatic CMV will have more severe consequences and poorer prognosis than will asymptomatic infants. The timing of infection during pregnancy largely affects the expected outcomes and consequences to the fetus. It is possible for a fetus to acquire congenital CMV infection from a nonprimary infection, although this accounts for a small number of cases. The presence of microcephaly, intrauterine growth restriction, petechiae, encephalitis, hepatosplenomegaly, and deafness are some of the physical characteristics of a congenital CMV infection. Treatment options remain limited at this time, so no routine screening has been recommended. The need for a vaccine or preventative treatment has been identified as a priority in the United States.

KEY WORDS: chorioretinitis, congenital cytomegalovirus, hepatosplenomegaly, hyperbilirubinemia, microcephaly, primary infection, TORCH infection

Cytomegalovirus (CMV) infection is one of the most frequently occurring viruses worldwide, with an incidence of 0.2% to 2.3% of live births.[1,2] However, only 1.5% to 2% of women with CMV infections are believed to have acquired the infection during pregnancy.[3,4] All socioeconomic groups and geographic locations are at risk of CMV infections, although a higher incidence is found in areas of dense population and lower socioeconomic conditions.[5] Cytomegalovirus is classified by mode of exposure into congenital; exposure in utero, perinatal; exposure during delivery and postnatal; and exposure after birth through breast milk, saliva, or other bodily fluids. This article specifically focuses on congenital CMV.

Congenital CMV infections remain the leading infectious cause of congenital malformations and mental retardation in developed countries with an increasing seroprevalence of between 50% and 85% of adult women in the United States testing CMV positive by age 40 years.[3,5] Cytomegalovirus is the most common of the toxoplasmosis, rubella, cytomegalovirus, herpes and other infection groups (TORCH infections), yet it is not included in routine prenatal screening or routine newborn screening.[6] The need for a vaccine or for a preventative treatment has been identified as a priority as a result of the extensive colonization.

INCIDENCE AND DEMOGRAPHICS

All socioeconomic groups and geographic locations are at risk of becoming colonized with CMV, although a greater risk is posed to people living in dense populations and lower socioeconomic conditions.[5] Of the herpes virus group, CMV affects the largest number of people, with up to greater than 2% of all live births worldwide.[1,2] Recently it has been suggested that CMV is responsible for persistent infertility and recurrent pregnancy losses in addition to an increase in premature delivery. This adds substantially to infant morbidity and healthcare costs.[6]

The incidence of exposure to CMV is extensive in all populations but greater in women of low socioeconomic groups (Figure 1). Of women acquiring a primary CMV infection during pregnancy, 50% of the fetuses will be infected.[3] Only 3.2% of all infants with congenital CMV infections acquired these from a maternal recurrent infection.[3]

Approximately 40% to 50% of infants are infected with CMV in utero when their mothers have a primary CMV infection during pregnancy; however, only 10%

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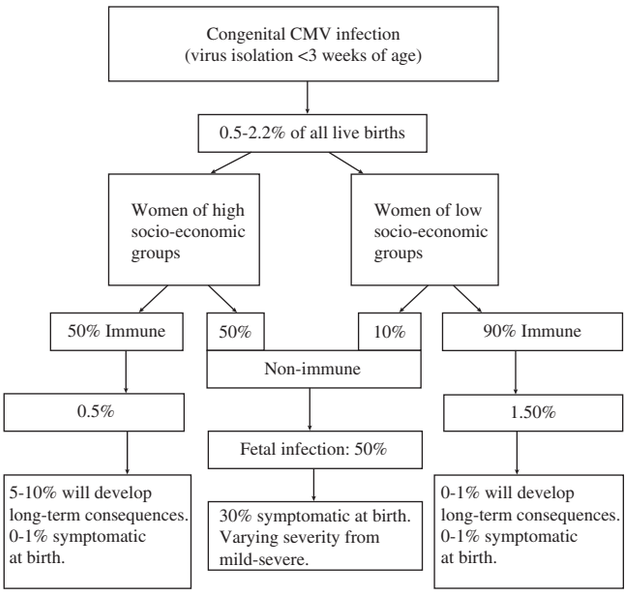
FIGURE 1.



Incidence of congenital CMV. Adapted from Trincado DE, Rawlinson WD. Congenital and perinatal infections with cytomegalovirus. *J Paediatr Child Health.* 2001;37,187-192.

to 15% of these infants are symptomatic at birth.[2,7] Of the symptomatic infants, 20% will die as a consequence of the infection, such as prematurity or encephalitis.[7]

Pathophysiology and Transmission

Congenital CMV infections cause illnesses in the neonate ranging from asymptomatic infection to extreme prematurity, encephalitis, deafness, and even death.[3] As with all herpes viruses, once a person is infected, the virus continues to be present for the remainder of the person's life but may be dormant.[8] Exacerbations of the dormant virus often occur during pregnancy as a result of the physical and emotional stress on the body. The rate and timing of transplacental transmission is unknown, but the pathology suggests that the frequency of transmission is equal in each trimester of pregnancy.[6] Cytomegalovirus is transmitted by infected leukocytes traveling from the mother through the placenta to the fetus. Cytomegalovirus infects the cell and can either destroy it or become part of the cell's DNA. Because the virus can become part of the cell, macrophages and T cells do not easily recognize it, and replication occurs rapidly.[6] The leukocytes can then replicate in placental fibroblast, syncytiotrophoblast, and cytotrophoblast cells and enter the fetal circulation via the umbilical vein.[3] If the transmission begins early within the first trimester, the damage to the fetus will be the most exaggerated, leading to severe injury to the fetus. The replication of the CMV-infected cells in the placental tissue can also be shed into the amniotic cells and fluid that is swallowed by the fetus. These cells will continue to replicate, colonizing the oropharynx, which leads to colonization of the gastrointestinal and genitourinary tracts.[8]

Transmission of the virus has been linked to saliva, breast milk, respiratory secretions, urine, blood, and feces, as well as semen and vaginal secretions.[6] Transmission of CMV to a fetus can occur throughout the entire pregnancy and during or after delivery. Transmission during pregnancy, which results in congenital infection, is thought to occur through the placenta.[9] Perinatal and postnatal CMV infections are most often transmitted via body fluids, such as vaginal secretions at delivery or breast milk.[10]

The pathogenesis of intrauterine infection is not completely understood; unlike the presence of antibodies for rubella or toxoplasmosis, the presence of antibodies for CMV does not provide protection against intrauterine transmission of the infection.[6] This means pregnancies occurring after a primary CMV infection are still at risk for CMV. Although it has not been determined, there is some thought that congenital CMV infections that occur with a recurrent or nonprimary maternal CMV infection may be related to exposure to a different strain of the virus.[6]

The risk of acquiring congenital CMV from a nonprimary infection is less than the risk associated with

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a primary infection. The decreased risk is thought to be related to the ability of the maternal immunoglobulin G (IgG) to cross the placenta and provide some protection against CMV in subsequent pregnancies.[6] Because CMV does not pose any significant health threat to healthy adult women, most women are not aware that they have been infected before or during pregnancy.

Screening

Prenatal screening for CMV is not routinely done in the United States, even though 50% to 85% of adults are infected with CMV.[5] Recommendations for serologic screening for all pregnant women were suggested in the late 1990s, but cost and lack of treatment options have prevented screening from becoming the standard of care.[3,11] Because the transmission of CMV can occur at any time throughout pregnancy, pinpointing a specific screening time has been difficult. A negative screen at 20 weeks' gestation would not indicate that the mother of the baby has not been exposed to CMV when the infant delivered. The negative screen at 20 weeks' gestation would only indicate that she was not infected with CMV before that point, but exposure could happen after that point and still put the infant at risk. Women with a primary infection during pregnancy can be screened by serological testing; if the screen is positive, the healthcare provider may recommend a nucleic acid test (NAT) and culture of the amniotic fluid.[3] Serological testing in the mother proves her exposure but does not indicate whether the virus has been transmitted to the fetus. Although a positive culture from amniotic fluid provides a 94% certainty of fetal CMV infection, it does not predict sequelae to infant.[12] Specimens from chorionic villus sampling can also be used to test for CMV infection. These invasive procedures are not recommended for screening for CMV; however, if these procedures were performed to detect the presence of chromosomal defects, a screen for CMV could also be done with no increased risk to the fetus.[3]

Screening an infant for congenital CMV infection must be completed within the first 3 weeks of life.[6] After 3 weeks of age, a positive result may indicate a perinatal or postnatal infection, and diagnosis of a congenital CMV infection can not be isolated.[1]

Urinary CMV polymerase chain reaction (PCR) is a reliable and rapid method of screening infants for CMV infections and remains the standard of practice.[1] The urinary PCR test detects CMV DNA in urine and therefore can accurately diagnose a congenital CMV infection without an invasive procedure.[8] Polymerase chain reaction can also be done on saliva and amniotic fluid, with the sensitivity and specificity reported at 89.2% and 95.8%, respectively.[8] Dried blood spots on filter paper similar to phenylketonuria (PKU) testing has recently been studied and has sensitivity and specificity comparable to urine PCR.[13] The dried blood spot method is simple and less expensive than the traditional viral isolation currently used.[13] Adding this screening tool to the current newborn screen may be seen in the near future as a way to avoid the long-term consequences CMV can cause. New PCR testing of meconium for CMV DNA is also being investigated for its ability to detect CMV in newborns.[6]

Physical Assessment

Congenital CMV has a variety of symptoms ranging at birth from none to full end organ dysfunction. Cytomegalovirus has a particular attraction to the liver, eyes, and central nervous system (CNS), which accounts for the symptoms found in infants with congenital CMV infections.[7] Table 1 lists commonly found symptoms in the physical exam of infants with congenital CMV infections.

Upon inspection, findings at birth may include intrauterine growth restriction (IUGR), petechiae, hyperbilirubinemia within 24 hours of life, seizures, and microcephaly.[8,14] Intrauterine growth restriction is present in most cases. If IUGR is present without known maternal cause, TORCH infections should be on the list of differential diagnoses.

Petechiae or purpura found throughout the skin surface is known as blueberry muffin rash for its resemblance to a blueberry muffin, as displayed in Figure 2. Blueberry muffin rash is not observed frequently but is characteristic of viral infections. The presence of petechiae or purpura at birth covering more than just the infant's head or upper chest indicates the presence of thrombocytopenia and is a sign that damage to the liver has occurred. The causes of thrombocytopenia are discussed in more detail in the next section. Infants born with the umbilical cord wrapped around their neck or upper chest are often born with petechiae noted above the placement of the cord without indicating any coagulopathy. Continuing with the visual assessment of the infant, yellowing of the skin

TABLE 1. Physical Assessment Findings of Congenital CMV[8,14,16]

Physical Signs and Symptoms of CMV	
• Microcephaly	• Petechiae
• IUGR	• Jaundice
• Hepatomegaly	• Seizures
• Splenomegaly	• Neurologic abnormalities, including hypotonia, jitteriness, reflexes

CMV, cytomegalovirus

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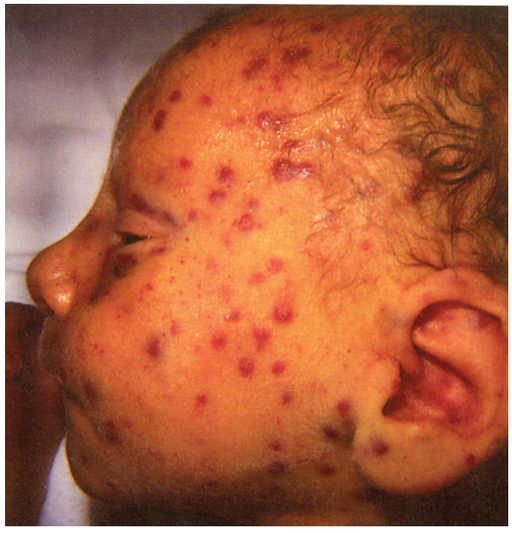
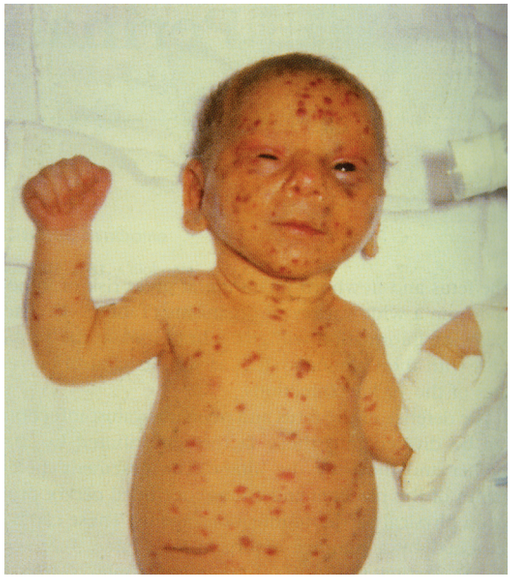
FIGURE 2.



Petechiae and purpura in infant with congenital CMV infection. From Clark DA, Thompson JE, Barkemeyer BM. *Atlas of Neonatology.* Philadelphia, PA: WB Saunders; 2000: 56. Reprinted with permission.

FIGURE 3.



Hepatosplenomegaly with jaundice in infant with congenital CMV infection. From Clark DA, Thompson JE, Barkemeyer BM. *Atlas of Neonatology.* Philadelphia, PA: WB Saunders; 2000: 56. Reprinted with permission.

may be observed. Hyperbilirubinemia or jaundice, in the first 24 hours of life further indicates damage to the liver. Conjugated hyperbilirubinemia and its causes in infants with congenital CMV are discussed in the Laboratory Findings section. Figure 3 depicts an infant with jaundice.

Cytomegalovirus contracted early in gestation has a greater risk of interfering with CNS development; however, all infants with CMV are at risk. Plotting the head circumference is important to assess whether the infant is microcephalic and needs further evaluation. Microcephaly is an occipital-frontal circumference below the 10th percentile with a normal weight and length for gestational age.[15] Infants with microcephaly need further CNS evaluation. These infants should be monitored closely for neurologic abnormalities, such as jitteriness, abnormal reflex response, and seizure activity. Also note the infant's tone and position. Tone can be difficult to assess in premature infants; however, hypotonia may indicate a problem that requires further investigation.

Infants with congenital CMV may have CMV pneumonia, which may be evidenced by an increased work of breathing with use of accessory muscles, nasal flaring, or inter/subcostal retractions found upon inspection of the infant. Further evaluation of the respiratory assessment includes auscultation and radiographs, all of which is described in more detail in this article.

After the visual assessment, begin auscultation of the lung fields and abdomen. Beginning at the apex, continue systematically across the chest and down with bell of the stethoscope on the anterior and posterior of the infant's chest. Normal breath sounds should be clear, without wheezing or crackles; the pitch should be low throughout most of the lung fields. When pneumonia is present, crackles may be heard in various areas throughout the chest. Crackles is used to describe the noise heard as the air passes through a congested or consolidated area, where the infection is present in the lung.[15] To confirm pneumonia, a chest x-ray must be obtained.

When listening to bowel sounds, be sure to assess all 4 quadrants for quality, intensity, and presence.[15] Normal bowel sounds occur at least every 15 to 20 seconds, with a metallic quality to the sound.[15] Infants with congenital CMV infections most often have normal bowel sounds with normal bowel function unless the enlargement of the liver is impeding the intestines.

Once auscultation is completed, begin palpation of the infant. This is a critical part of the physical exam because of the significance of the possible findings. First, superficial palpation should be completed, followed by deep palpation. Superficial palpation will identify the liver and spleen, with deep palpation assessing the kidneys or other abdominal masses. Infants with congenital CMV infections may or may not present with hepatosplenomegaly. Hepatosplenomegaly is defined as enlargement of the

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liver and spleen.[16] A liver edge measuring greater than 2 cm below the right costal margin indicates an enlarged liver and should be investigated.[17] A normal size liver has a lower edge that is not lower than 1 to 2 cm below the right costal margin.[15] The liver should be smooth and firm.[17] To palpate the liver, start just above the right iliac crest, using fingers to gently push down 1 to 2 cm while moving upward toward the chest. Avoid lifting fingers or releasing pressure to prevent missing the liver's edge. To assess for splenomegaly (an enlarged spleen), place 2 to 3 fingers just above the iliac crest on the left side of the abdomen. Gently press down with your fingers, working up the left side of the abdomen toward the chest, to the costal margin. A normal spleen should not be palpable greater than 1 cm below the costal margin; when it is, an ultrasound should be performed to investigate further.[15] A normal spleen is rarely palpable.

Laboratory Findings

When congenital CMV is suspected because of findings on physical exam, further studies will aid in confirming the diagnosis and evaluating the extent of the infection. Table 2 contains common laboratory findings in infants with CMV. A urine sample sent for viral culture for CMV is needed to diagnose the presence of the virus. The urine sample can be collected by clean catch. A sterile catheter-acquired sample is not required because CMV does not live on the skin and thus does not risk contaminating the sample. Blood and cerebrospinal fluid can also be sent for viral culture, but these invasive procedures do not provide a more conclusive diagnosis.[3] Viral replication is highest in the urine, so urine culture is the standard of practice for CMV diagnosis.

A complete blood count (CBC) serologic test will determine the presence of thrombocytopenia, neutropenia, and anemia. Any or all of these findings may be present due to liver impairment as a result of the virus's affinity for the liver.[7] The liver is responsible for hematopoiesis in the fetus; as a result, any damage to the liver affects the development of blood cells, including red blood cells, white blood cells, and platelets, causing the infant to be anemic, neutropenic, and thrombocytopenic.[4]

TABLE 2. Laboratory Findings of Congenital CMV[4,7]

- Jaundice
- Anemia
- Neutropenia
- Thrombocytopenia
- Elevated liver function/enzymes
- Positive urine viral count

CMV, cytomegalovirus

If an infant is noted to have jaundice early in the first 24 hours of life, an abnormal CBC with thrombocytopenia or anemia, hepatomegaly or known CMV exposure, bilirubin levels should be followed. Most infants have a mildly elevated unconjugated bilirubin during the first few days of life.[15] This is a normal finding and occasionally needs phototherapy as treatment. When excessive hemolysis occurs (often in response to infection), the unconjugated bilirubin will be elevated enough to require treatment–phototherapy, hydration, or in extreme cases, an exchange transfusion.[18]

Infants who have had liver damage as a result of infection such as CMV will have an elevated conjugated bilirubin.[4] Conjugated bilirubin should not be present within the first 2 weeks of life, so additional liver function tests are needed if an infant has a measurable level of conjugated bilirubin during this time.[18] Further laboratory studies that measure enzymes that indicate how well the liver is functioning include alkaline phosphatase (AP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transpeptidase (GGT), and blood clotting studies. Viral infections such as CMV can cause elevations in these values once damage has occurred in the liver, so infection should be investigated when elevated liver function tests have been determined.

Radiographic Findings

The physical assessment and laboratory tests provide much of the information in diagnosing an infant with CMV. More information can be gleaned from radiographic and other study findings that are listed in Table 3. Cytomegalovirus pneumonia is found in some infants with congenital CMV exposure. As a result the physical examination of breath sounds is extremely important; however, a chest x-ray is required to diagnose pneumonia. Figure 4 shows an infant with CMV pneumonia. Pneumonia results when the infant breathes in amniotic fluid in which CMV has been shed.

TABLE 3. Radiographic/Study Findings of Congenital CMV[18,19]

- Hearing loss
- Chorioretinitis
- Hepatomegaly
- Splenomegaly
- Chest x-ray (pneumonia)
- Intracranial calcifications
- Seizures
- Abnormal CT scan: ventriculomegaly, white matter lucencies and brain atrophy

CMV, cytomegalovirus; CT, computed tomography

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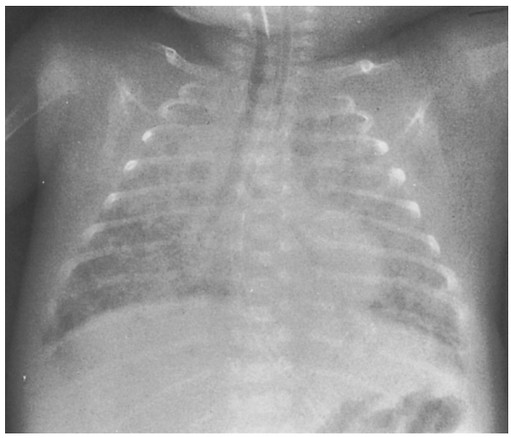
FIGURE 4.



Chest x-ray; diffuse CMV pneumonia. From Clark DA, Thompson JE, Barkemeyer BM. *Atlas of Neonatology.* Philadelphia, PA: WB Saunders; 2000: 57. Reprinted with permission.

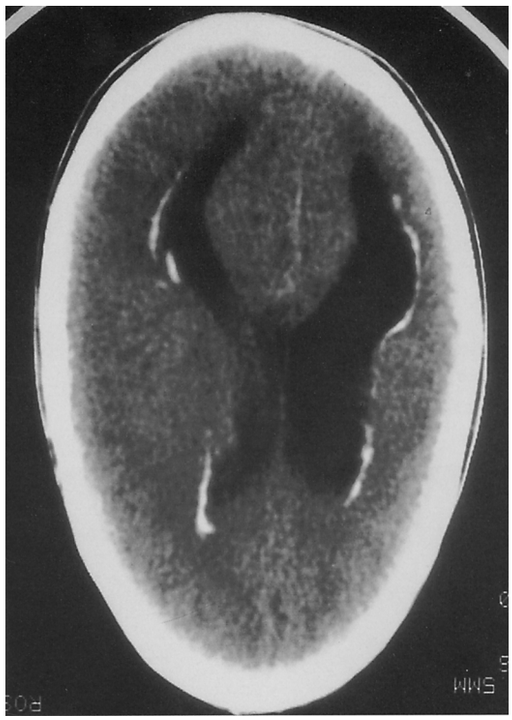
An abdominal x-ray would be a gross measure of hepatosplenomegaly, as shown in Figure 3. In addition an abdominal ultrasound can be done to further investigate the quality, size, and function of the liver and spleen.

A cranial ultrasound can be used to assess for periventricular calcifications often found in infants with congenital CMV. A computed tomography (CT) scan of the brain will also be needed to assess the remainder of the intracranial structures. Figure 5 displays a CT scan positive for calcifications related to a congenital CMV infection. Calcifications have been reported in brains of infants who were thought to have acquired the infection even late in pregnancy, often with minimal symptoms at birth, and then increasing neurodevelopmental problems as the child develops.[19]

Once CMV is diagnosed, an ophthalmologic exam is needed to assess for the presence of chorioretinitis and optic nerve hypoplasia. Chorioretinitis is inflammation of the choroid and retina[18] (Figure 6). Cytomegalovirus is the second leading cause of chorioretinitis, behind toxoplasmosis.[20] Partial or total loss of vision is a potential complication of chorioretinitis.[20] It is present in only 10% to 15% of symptomatic infants.[18] In most cases in which eye complications are present, periventricular calcifications are found and significant mental impairment results.[18]

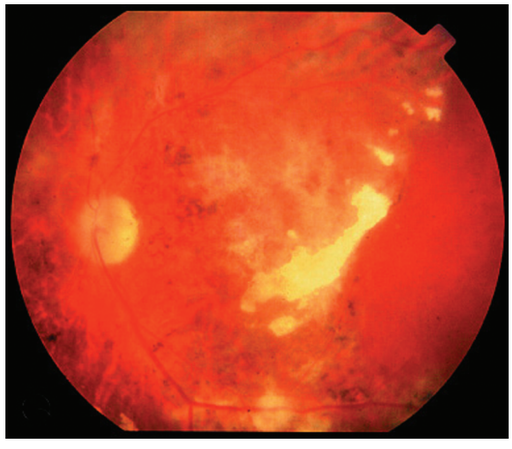
Because hearing loss is present in only one third of infants with CMV at birth, it is critical that infants diagnosed with congenital CMV have their hearing followed closely as they develop.[18] Many infants do not begin to have degeneration of their hearing until after the age of 2 years.[18]

FIGURE 5.



CT scan positive for periventricular calcifications. 1. From Clark DA, Thompson JE, Barkemeyer BM. *Atlas of Neonatology.* Philadelphia, PA: WB Saunders; 2000: 57. Reprinted with permission.

FIGURE 6.



Chorioretinitis. From Taylor D, Hoyt C. (eds). *Pediatric ophthalmology and strabismus.* 3rd ed. Philadelphia, PA: Elsevier; 2005. Reprinted with permission.

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Infants with suspected seizure activity need to have an electroencephalography (EEG) to confirm and diagnose abnormal brain activity. An EEG is needed only after seizure activity is suspected and not as an investigational study.

Treatment

At this time, treatment of congenital CMV infections remains a matter of controversy. Many argue the necessity of treating asymptomatic infants to reduce long-term sequelae, and others argue the adverse reactions of the available medication are not worth the risk even in symptomatic patients. Currently, ganciclovir is the only antiviral available to treat CMV. Ganciclovir is a parenteral antiviral agent that interrupts the replication of CMV in the DNA by competitively inhibiting the binding of deoxyguanosine triphosphate to DNA polymerase.[21] The adverse effects of ganciclovir include bone marrow suppression, gonadal toxicity potentially leading to sterility, neutropenia, thrombocytopenia, anemia, and seizures. Ganciclovir is to be used with extreme caution in neonates because the long-term safety has not been studied. Nevertheless, the current standard treatment regimen involves 6 weeks of intravenous (IV) administration.[8,21] An improvement in status and symptoms has been shown during the course of treatment with administration of the medication in addition to a decreasing viral count present in the urine of the treated infants. However, at the completion of the antiviral therapy, viral shedding returns to pre-treatment levels.[8,22,23] Long-term follow-up indicated a slight decrease in hearing loss in symptomatic infants who were treated.[24] The use of ganciclovir in symptomatic infants for a 6-week intravenous course showed an improvement in hearing loss at 6 months and some improvement at 1 year.[25]

Ganciclovir is not recommended for infants with severe neutropenia, thrombocytopenia, or elevated liver enzymes because these are adverse reactions to the medication. Deciding to use ganciclovir in a symptomatic patient can be difficult because symptoms of CMV often include neutropenia, thrombocytopenia, and elevated liver enzymes.

Because the viral load returns to pre-treatment levels after the completion of the treatment course and the adverse effects of ganciclovir are so great, when to treat and who to treat remains a matter of controversy.[8,23]

There is currently no available vaccine for CMV. Attempts continue to be made to develop a vaccine that would prevent women from obtaining the virus. This development has been complicated by the identification of several strains of CMV.[8] It is now thought that the intrauterine transmission may occur as a result of a primary infection of a new strain of CMV in women who had a CMV infection prior to the pregnancy.[8]

Outcome

Of the infants infected with CMV who survive the infection, 90% will experience long-term neurologic impairments of varying degrees. Figure 1 depicts the incidence of CMV and the probability of having symptomatic versus asymptomatic infection.[12] Infants symptomatic at birth have greater neurologic sequelae than do asymptomatic infants.[12] The more severe the symptoms are at birth, the more significant the neurological impairment.[12,26] The earlier the primary infection occurs in utero, the worse the outcome for the fetus, with the pregnancy often ending in miscarriage or premature delivery.[22,25,26] Infants who have congenital CMV infections who are asymptomatic at birth still have a 5% to 15% risk of developing neurologic abnormalities in early childhood.[2] It is also possible for a symptomatic infant to have acquired a CMV infection in utero from a recurrent maternal infection, although this accounts for a small percentage of all congenital CMV infections.[3] It is not clearly understood whether infection occurring from a nonprimary infection is recurrent or a new strain of the virus, although a new strain is suspected.[8]

The presence of IUGR and microcephaly at birth were the most significant predictors of mental retardation and motor disability.[14,26] In addition, the degree of microcephaly was directly related to the infant's intelligence/developmental quotient (IQ/DQ) at follow-up.[14]

Although microcephaly is a predicting factor used for neurological impairment, it was not predictive of hearing loss related to congenital CMV infections. The presence of petechiae and IUGR were found to be independent predictors of hearing loss.[26] An abnormal CT scan in combination with microcephaly correlates with mental retardation with IQ <70.[14] An abnormal CT scan alone was not found to be a predictor of poor neurological outcome.[14] A diagnosis of chorioretinitis in infants with CMV infection is also associated with poor neurological outcome.[14]

Clinical Implications

Because no vaccine and no effective cure is currently available, prevention of CMV infections needs to be a priority. Women need to be educated regarding ways to protect themselves from this viral infection. Anyone handling or exposed to bodily fluids needs to be aware of the risk of transmission and use standard precautions. The best way to decrease transmission is through frequent hand washing.[27] Women need to be educated about the necessity of hand washing after diaper changes, handling respiratory secretions and after contact with children, especially those who attend daycare. This education is extremely important for pregnant women who are CMV negative, but all women need to be aware of these simple precautions to prevent viral colonization.

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Infants with congenital CMV infections do not require contact isolation precautions; standard precautions are adequate.[5] Because most infants with congenital CMV are asymptomatic, it is important for healthcare providers to use universal precautions while handling bodily fluids of all patients, including diaper changes, to decrease their risk of transmission.[5]

Healthcare providers should be aware that universal hearing screens will detect less than half of the hearing loss caused by congenital CMV infections in neonates.[28] The universal hearing screen does not detect sensorineural hearing loss, which is what is found in infants with congenital CMV.[28] Because many children with congenital CMV are asymptomatic, no further evaluation of hearing is routinely done, and hearing loss may go undiagnosed, leading to developmental delay and impairment. For this reason, it is recommended all infants known to have congenital CMV should be closely monitored for hearing and visual impairments during the first 2 years of life.[26]

The significance of congenital CMV infections lies in the potential to cause long-term neurological impairment, including hearing loss, behavioral dysfunction, cerebral palsy, and cognitive and visual impairments.[22] Early detection of CMV infection would allow for appropriate screening for neurological, hearing and visual impairments and encourage implementation of early intervention programs to avoid long-term consequences of the neurodevelopmental sequelae. Early detection of CMV infections through newborn screening would also allow for appropriate hearing, vision, and developmental follow-up.

As long as reliable vaccines are not available, prevention should be our focus. Education concentrating on how to avoid infection with frequent hand washing and safe sexual practices is needed for all pregnant women to reduce the spread of this life-threatening virus.[8]

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