Screening, Prevention, and Treatment of Congenital Cytomegalovirus

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KEYWORDS

• Cytomegalovirus • Congenital infection • CMV diagnosis • CMV prevention
• CMV hyperimmune globulin

KEY POINTS

- Congenital CMV affects 0.64% of births each year and is one of the most common causes of childhood disability.
- Routine screening for primary infection in pregnant women is not recommended by the Centers for Disease Control and Prevention (CDC) or the American College of Obstetricians and Gynecologists.
- There is insufficient evidence to support the use of passive immunization to prevent congenital infection.
- There is no effective vaccine for primary prevention.
- The best means of prevention is through reducing exposure to the virus.
- Pregnant women at risk of exposure should be counseled regarding congenital CMV and hygiene measures.

INTRODUCTION

Congenital cytomegalovirus (CMV) affects approximately 0.64% of newborns each year.[1,2] There are approximately 27,000 cases annually in the United States.[3] Of those children, almost 20% develop permanent disabilities and 1% do not survive. In addition to a significant emotional burden, the annual costs associated with CMV are estimated to be at least $1 to $2 billion.[4] Currently, the only known means of reducing the risk of congenital CMV is by reducing exposure to the virus. However, it has not been shown that educating pregnant women regarding risk-reduction behavior is effective.

The authors have nothing to disclose.
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CLINICAL OUTCOMES

Clinical outcomes of neonates affected by congenital CMV vary greatly. Fowler and colleagues[5] compared outcomes of CMV-infected neonates born to mothers with primary infection (N = 125) with mothers with recurrent infection (N = 64). The only infants with symptoms at birth (18%) were in the primary maternal infection group. Symptoms found in those affected at birth are jaundice, petechiae, hepatosplenomegaly, small for gestational age, preterm birth, microcephaly, and death. Children were followed for up to 6 years after birth, and 25% of primary infections versus 8% of recurrent infections (P = .003) developed sequelae. Sensorineural hearing loss was the most common finding (15% vs 5%; P = .05).[4] Outcomes associated with congenital CMV in the infected newborn are as follows:

- Hearing loss (15%)
- Vision loss (2%)
- Mental disability (13%)
- Microcephaly (5%)
- Seizures (5%)
- Death (2%)

EPIDEMIOLOGY

CMV is a member of the Herpesvirus family. Like other members of this family, after the initial infection, reactivation of a latent infection can occur as can reinfection with a different strain of virus. Seroprevalence rates range from 40% to 83% in women of childbearing age in the United States, whereas seroconversion occurs in approximately 1% to 4% of seronegative pregnant women.[3,6–8] Vertical transmission may occur after either a primary or secondary infection, but the rates are much higher after a primary infection (30%–40% vs 1%).[9,10] However, nonprimary maternal infection may be responsible for up to 75% of all congenital infections.[11] With increasing gestational age, the transmission rates increase, but severity of congenital disease decreases.[12]

The virus is shed in bodily fluids, such as saliva, urine, or semen. Transmission occurs with direct contact. Women at highest risk of infection are those that are exposed to the saliva or urine of young children.[13] Up to 45% of seronegative parents with young children shedding the CMV virus become infected.[14]

BIOLOGY OF CYTOMEGALOVIRUS INFECTION

Once infected with CMV, the incubation period is approximately 1 month. After 1 month, symptoms and/or viremia are present and the virus is shed in multiple bodily fluids. CMV-specific IgM antibodies peak 1 to 3 months after a primary infection and may be detected for up to a year.[15] CMV IgG antibodies are produced during the first few months of infection. They are initially of low avidity and after a maturation process, they have a high avidity. High IgM levels in combination with a low IgG avidity assay is representative of a recent infection (within the past 3 months).[16] The true mechanism of transplacental infection is unknown. One plausible hypothesis is the transport of nonneutralizing IgG-virion particles by transcytosis.[17]

DIAGNOSIS

Routine screening for primary infection in pregnant women is not recommended by the Centers for Disease Control and Prevention (CDC) or the American College of

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