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Congenital Cytomegalovirus (CMV)

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Congenital Cytomegalovirus (CMV)

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Introduction

Cytomegalovirus (CMV) is a common herpes virus infection that is usually harmless and belongs to a group of herpes viruses that includes the herpes simplex viruses, varicella-zoster virus (which causes chickenpox and shingles), and the Epstein-Barr virus (which causes infectious mononucleosis). Once CMV is in a person's body, it stays there throughout their life. (Centers for Disease Control and Prevention, 2010). CMV is shed in various bodily secretions, especially urine and saliva (Congenital Cytomegalovirus Foundation, 2014). According to the Centers for Disease Control and Prevention (CDC), the majority of otherwise healthy children and adults infected with CMV are asymptomatic while others may develop a mild illness when they get infected. Among every 100 adults in the United States, 50–80 are infected with CMV by the time they are 40 years old (2010). So why is a viral infection that is likely to cause a mild illness (if it causes symptoms at all) a concern? Because CMV can cause serious disease and have life-long effects on a newborn if a mother passes CMV on to the fetus during pregnancy (congenital CMV Infection). Congenital CMV is a known to be one of the most common fetomaternal viral infections (Kaneko et. al, 2013). This paper aims to bring awareness and an improved understanding of congenital CMV to advanced practice nursing students.

Prevalence

- CMV is the most frequent congenital infections in newborns and is the leading cause of hearing deficit and cognitive disability in the United States with a direct economic cost of \$1 billion to \$2 billion annually (Stowell, Forlin-Passoni, Cannon, 2010).
- Out of 1,000 live births, about 8 (less than 1%) infants will have congenital CMV infection. 1 or 2 of those 8 infants will have permanent problems (such as developmental disabilities or hearing loss) due to the infection (CDC, 2010).

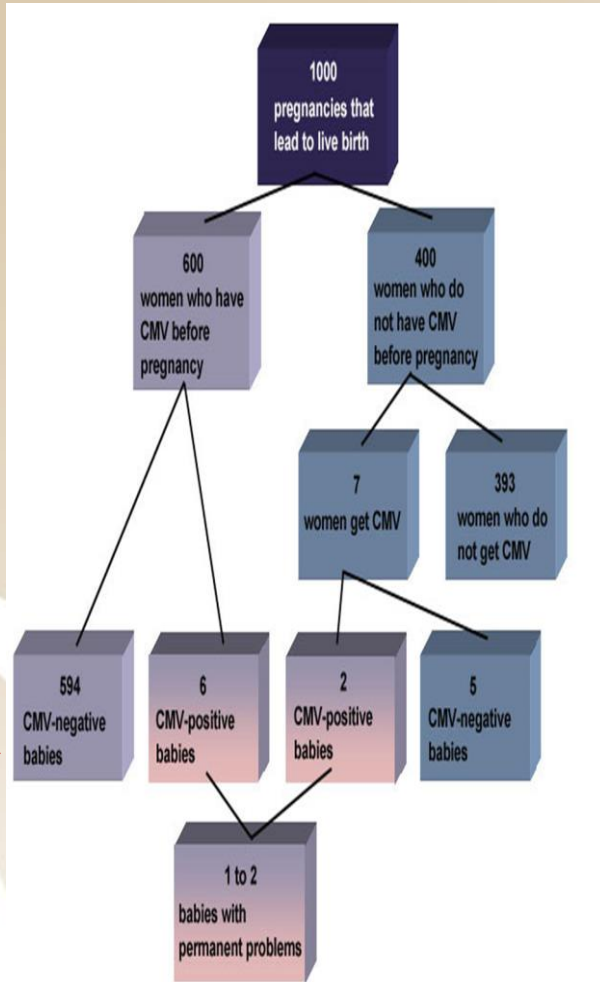


Figure 1 (above) shows that for every 1,000 pregnancies that result in a live birth, about 400 of those women will not have had a CMV infection prior to becoming pregnant. Of those 400 women, about 7 of them will become infected with a primary CMV infection during their pregnancy. Of the 7 infected women, about 2 will have infants with CMV infection. And, of the 600 women who have had CMV infection prior to their pregnancy, about 2 of them will have babies with CMV infection. (Cannon, M.J. Congenital cytomegalovirus (CMV) epidemiology and awareness. J Clin Virol. 2009;46 [Suppl 4]:S6-10; retrieved from www.CDC.gov/cmv/trends-stats.html)

Pathophysiological Processes

Sign & Symptoms

According to Schleiss (2015), approximately 10% of infants with congenital CMV will have symptoms of the disease at birth and may include; intrauterine growth retardation, enlarged liver and spleen, thrombocytopenia, and a variety of cutaneous manifestations including purpura and petechiae. Schleiss (2015) further states, "...the most significant manifestations involve the CNS. Microcephaly, ventriculomegaly, cerebral atrophy, chorioretinitis, and sensorineural hearing loss are the most common neurological consequences".

Intracerebral calcifications frequently exhibit a periventricular distribution and are frequently encountered using CT scanning (see Figure 2 below). The finding of intracranial calcifications is predictive of intellectual and audiological deficits later in life. These findings forecast a poor neurodevelopmental prognosis (Schleiss, 2015).

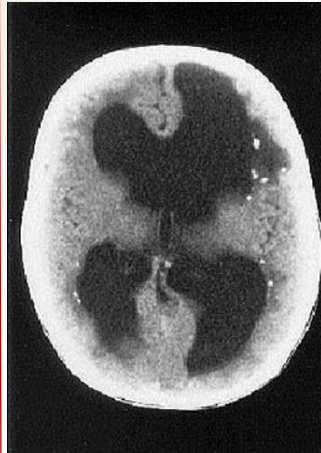


Figure 2. Cranial CT scan of infant born with symptomatic congenital cytomegalovirus infection. Neurological involvement is evident as manifested by ventriculomegaly and periventricular calcifications. (Schleiss, 2015).

Underlying Pathophysiology

According to Schleiss (2015), CMV has a tendency to infect mononuclear cells and lymphocytes. It is the biggest member of the herpes virus family, with a double-stranded DNA genome capable of encoding more than 200 potential protein products. Immediate gene transcription happens within the first 4 hours of infection, when main regulatory proteins that let the virus to take control of cellular machinery are made. Late gene products are made about 24 hours after infection, and these proteins are primarily structural and allow for virion assembly and egress.

One of the classic symbols of CMV infection is the cytomegalic inclusion cell. These extremely enlarged cells contain intranuclear inclusions that have the histopathological appearance of owl's eyes. The existence of these cells indicates productive infection (Schleiss, 2015).

The way in which CMV harms the fetus is complex and probably includes a combination of direct fetal cellular injury (especially in the fetal brain), an incomplete maternal immune response unable to control the infection, and the impact of the infection on placental function (including oxygen and substrate transportation). CMV also encodes gene products that function at both the RNA and the protein level, to interfere with many cellular processes including; modification of the cell cycle, interfering with cell apoptosis, inflammatory response, mediating vascular injury, and proteins that create site-specific breakage of chromosome, dysregulation of cell proliferation, and most importantly, genes that facilitate evasion of host immune responses (Schleiss, 2015).

Significance of Pathophysiology

Immunity to CMV is multifaceted and involves humoral and cell-mediated responses. Recently it has been discovered that CMV utilizes 2 pathways of entry into the cell. The first way is via a fusion-mediated pathway in fibroblasts. The second way is an endocytosis-mediated pathway in epithelial and endothelial cells. Proteins that are important to these pathways (encoded by UL128-131 genes) may emerge as particularly useful vaccine candidates in future studies (Schleiss, 2015).

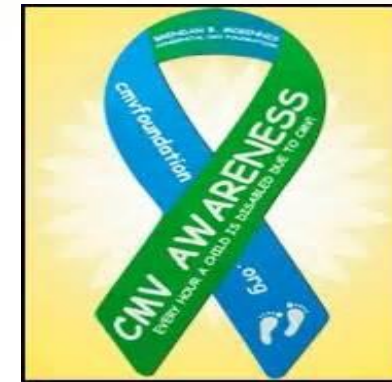
Implications for Nursing Care

Women who have close contact with young children (i.e. daycare workers) are particularly at risk to contracting the CMV virus and passing it along to their unborn infant. However, routine screening for CMV is not recommended and there is not currently a vaccine available. Therefore, prevention of CMV transmission is focused on better hygienic practices including; routine hand washing, not sharing cups, utensils, or food, and not kissing a child on the lips or near saliva. Prevention-based interventions focus on education and counseling (Thackeray, Wright, Chipman, 2014).

Conclusion

Despite recommendations that CMV be part of health promotion counseling women of child-bearing age receive, less than 50% of obstetricians/gynecologists in the United States report counseling their patients about how to prevent CMV infection. Awareness of CMV is relatively low among women with only about 13-22% of women in the United States having heard of CMV (Thackeray, Wright, Chipman, 2014).

As advanced practice nurses, we have an opportunity to heighten awareness about congenital CMV and to educate our patient's on prevention of transmission of this potentially detrimental viral infection while researchers continue to investigate and test potential vaccines for this virus.



CMV Awareness Ribbon @ cmvfoundation.org

Additional Sources


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