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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 some may develop a mild illness when they get infected. Among every 100 adults in the United States, 56-80 are infected with CMV by the time they are 40 years old (2010). So why is a viral infection that is likely harmless and belongs to a group of viruses we are frequently encountered using CT scan or ultrasound? (Centers for Disease Control and Prevention, 2010).

Prevalence

• CMV is the most frequent congenital infections in newborns, and is the leading cause of hearing and vision disability in the United States with a direct economic cost of $1 billion to $2 billion annually (Stowell, Forlin-Passoni, Cannon, 2013).

• Out of 1,000 live births, about 8 (less than 1%) infants will have congenital CMV (Cannon, M.). About 2 of these infants will have permanent disabilities such as developmental (disabilities or hearing loss) due to the infection (CDC, 2010).

Pathophysiological Processes

Sign & Symptoms

According to Schleiss (2015), approximately 19% of infants with congenital CMV will have symptoms of the disease at birth and may include: intracranial growth retardation, enlarged liver and spleen, thrombocytopenia, and a variety of outward 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.”

Intrauterine calculations frequently exhibit a perturbenticular distribution and are frequently encountered using CT scanning (see Figure 2 below). The finding of intrauterine calculations is part of the differential of intrauterine developmental deficits later in life. These findings forecast a poor neurodevelopmental prognosis (Schleiss, 2015).

Underlying Pathophysiology

According to Schleiss (2015), CMV has a tendency to infect monocytic 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 begins within the first 4 hours of infection, when major regulatory sequences that control the virus to take control of cellular machinery are made. Late gene products are made about 24 hours after infection; these proteins are primarily structural and allow for virus assembly and egress.

One of the classic symptoms of CMV infection is the cytomegalic inclusion cell. These extremely enlarged cells contain intranuclear inclusions that have the histopathological appearance of eve’s eyes. “The inclusions likely result in 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 to the fetal liver), in an attempt 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 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 wave is a homologous pathway in fibroblasts. The second wave is an endothelial-mediated pathway in epithelial and endothelial cells. Proteins that are important to these pathways (encoded by UL135-117 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 uninfected 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 educating women about their patient’s prevention of CMV infection (McLarty, 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 13-25% of women in the United States have heard of CMV (Thackeray, Wright, Chipman, 2014).

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

References Cited


CMV Awareness Ribbon @ cmvfoundation.org

Additional Sources

