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Congenital Cytomegalovirus (CMV)

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Introduction
Cytomegalovirus (CMV) is a common herpesvirus 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, 2013). CMV is shed in various bodily secretions, especially urine and saliva (Conaghty 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 cold when they get infected. Among every 100 adults in the United States, 50-80 are infected with CMV by the time they have reached their 40s. So why is it a viral infection that is likely to cause a mild cold (if it causes symptoms at all)? Because CMV can cause serious disease and have lifelong effects on individuals, particularly mothers who pass CMV on to the fetus during pregnancy (Conaghty CMV Infection). Cytomegalovirus (CMV) is a known causative agent of the most common fetal-maternal infections (Kawecko 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 and visual disability in the United States with a direct economic cost of $1 billion to $2 billion annually (Stowell, Farley-Parsons, Connors, 2000).
- Out of 1,000 live births, about 8 (less than 1%) infants will have congenital CMV infection (Cowan, 2014). Of these 8 infected infants, 2 of them will have permanent disabilities (such as developmental disabilities or hearing loss) due to the infection (CDC, 2010).

Sign & Symptoms
According to Schleiss (2015), approximately 19% of infants with congenital CMV will have symptoms at birth and may include intracranial growth retardation, an enlarged liver and spleen, thrombocytopenia, and a variety of ophthalmic manifestations including ptosis and strabismus. Schleiss (2015) further states, “...the most significant manifestations involve the CNS. Microcephaly, ventriculomegaly, cerebellar atrophy, chorrioretinitis, and sensorineural hearing loss are the most common neurological consequences”. Intracranial calcifications frequently exhibit a periventricular distribution and are frequently encountered using CT scanning (see Figure 2 below). The finding of intracranial calcifications is prone to false-negative and false-positive deficits later in life. These findings forecast a poor-neurodevelopmental prognosis (Schleiss, 2015).

Pathophysiological Processes
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-early gene products are expressed within the first 4 hours of infection, while main regulatory genes are not made available until after cell infection, these proteins are primarily structural and allow for virus assembly and release.

One of the classic symbols of CMV infection is the cytoplasmic inclusion cell. These extremely enlarged cells contain intranuclear inclusions that have the histopathological appearance of eye’s “eyes”. The production of virions is an immediate-early 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 liver), 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 normal cellular processes including: modification of the cell cycle, interfering with cell apoptosis, inflammatory response, mediating vascular injury, and proteins that create site-specific breaks of chromosome, dysregulation of cell proliferation, and most importantly genes that facilitate evasion of host immune responses (Schleiss, 2015).

The immune system is modified 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; the second way is an endocytosis-mediated pathway in epithelial and endothelial cells. Proteins that are important to these pathways (encoded by UL128-115 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 educating our patient’s on protection from transmission of this potentially detrimental viral infection while researchers continue to investigate and test potential vaccines for this virus.

References Cited


