Perspective

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# Infectious viruses at Maternal-Fetal Interface

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## Perspective

Infections like Zika Virus (ZIKV) have highlighted the importance of understanding diseases that can be transmitted vertically from mother to fetus. Viruses make up a substantial fraction of TORCH pathogens [Toxoplasmosis, Other (syphilis, varicella-zoster, parvovirus B19, Zika), Rubella, Cytomegalovirus (CMV), and Herpes infections], which can cause prenatal infections that have an influence on newborn health and neurodevelopment. Pathogens have developed to avoid both innate and adaptive immune responses that limit infection transmission, therefore maternal and fetal immunity are crucial for infection clearance. A full-term human pregnancy lasts around 40 weeks and is separated into three trimesters that correspond to developmental stages. One of the most important factors of congenital illness outcomes is gestational age at the time of infection exposure. Infection with ZIKV during the first trimester of pregnancy, for example, is linked to an increased risk of birth abnormalities such microcephaly and ocular problems in babies. This was eloquently substantiated by researchers in a study that used intravaginal injection of ZIKV in rhesus macaques during developmental time periods corresponding with the first trimester of pregnancy to imitate sexual transmission of ZIKV infection.

Pregnant women who were exposed to ZIKV during the first trimester had non-viable embryos, and viral RNA was discovered in the demised embryos, indicating a risk of pregnancy loss during the first trimester. In contrast to ZIKV, CMV infections are linked to poor fetal health outcomes when they arise late in the second or third trimester of pregnancy. Researchers employed a Guinea pig model of CMV infection to investigate the increased susceptibility to CMV infection during late pregnancy. The maternal and placental virus load did not differ between the two infection groups when guinea pigs were infected at days 21 and 35 of gestation (similar to mid to late gestational time periods in humans). However, transcriptome analysis in the gestational day 35 infection group revealed substantial alterations in transcripts associated with immune activation, suggesting placental sensitivity to damage during late gestation CMV infection. HIV transmission from mother to child can occur at various stages of pregnancy, at the moment of birth, or after birth, in contrast to ZIKV infection, which is acute and mostly transmitted in utero. Some studies looked at the complexities of HIV transmission from mother to kid. They went into great detail on HIV infection and early life immunological responses, as well as viral persistence. These studies show that the timing of viral infection during pregnancy and early life has a significant impact on the immunological and functional consequences of viral infections.

The placenta is a special organ that develops during pregnancy to provide nutrients and gas exchange for the fetus. Immune functions that protect the mother and growing baby from negative consequences, such as congenital infections, balance this vital role. The placenta's structure is crucial to this function, with the maternal decidua separated from the placenta by a network of tight barriers including syncytiotrophoblast cells and fetal endothelium, where active transport pathways allow certain chemicals and molecules to flow from mother to fetus. The existence of a fully functional maternal immune system, which must interact with freshly developing fetal immunity, adds to the complexity of maternal-fetal interactions at the placental interface. Immunological tolerance characterizes immune responses at the maternal-fetal interface during healthy pregnancies, which must be balanced with the requirement to offer pathogen protection. Using a pregnancy model of rhesus macaques, the complexity of the maternal immune system is highlighted. In the maternal decidua and peripheral blood of pregnant moms, several innate and adaptive cell types were characterized. When comparing healthy and ZIKV-infected pregnancies, markers of immune suppression were seen in ZIKV-infected pregnancies, along with lower recruitment of functional cytotoxic T cells, suggesting a diminished ability for infection clearance.

Viral infection at the maternal-fetal interface can induce placental insufficiency, which can lead to intrauterine growth restriction (IUGR), which limits normal fetal growth. ZIKV infection caused the production of pro-inflammatory mediators and altered the abca1 transporter in the placenta, resulting in placental insufficiency and IUGR in wild type and type-I interferon deficient

 

mice. Researchers studied placenta tissue from pregnant women infected with ZIKV and HIV in humans. Their findings demonstrated pathological similarities such as higher numbers of knots, sprouts, and CD163+ Hofbauer cell hyperplasia in ZIKV-infected pregnancies compared to HIV-infected pregnancies, with more significant Hofbauer cell hyperplasia in ZIKV-infected pregnancies. In the context of maternal SARS-CoV-2 infection, morphologic characteristics of the placenta were also studied. Although vertical transmission of SARS-CoV-2 from mother to fetus is not well established, this work underscores the potential for maternal viral infection to alter fetal outcomes by affecting placental tissue. During COVID-19, the authors noticed both maternal and fetal malperfusion, and discovered that pregnant women with SARS-CoV-2 infection were more likely to develop chronic histolytic intervillositis, which is an inflammatory disorder characterized by the infiltration of histiocytes/macrophages into the placenta. These studies comparing virus-induced changes to placental ultrastructure in the context of chronic versus acute viral infections show that similar pathways are likely triggered to limit viral infection during pregnancy at the maternal-fetal interface, and that these pathways may influence placental tissue health.

Researchers studied the impact of SARS-CoV-2 infection on immunological components in breast milk, recognizing nursing as another key component of maternal-infant contact with the potential to alter immunity and infection. They discovered a profile of immunological activation in the form of greater levels of pro-inflammatory cytokines such Eotaxin, IP-10, MIP-1, and RANTES, as well as numerous growth factors, using a case-control research design. Adaptive immune responses in donors who had been infected with ZIKV during pregnancy was defined by some researchers. The accelerated decline of ZIKV-specific CD8 T cells relative to CD4 T cells in the months following infection resolution was underlined by extensive analysis of antigen-specific CD4 and CD8 T cells. The authors point out that there were no significant variations in T cell responses between moms of asymptomatic children and mothers of children with ZCS. In the context of ZIKV infection, our study demonstrates that moderate and severe congenital illness produce equivalent memory T cell responses.