Prenatal Prediction of Placenta Accreta Spectrum (PAS) Can Lower Maternal Morbidity and Mortality

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Abstract

High spatial and temporal resolution ultrasound is frequently used as the initial diagnostic imaging method during pregnancy. Placental imaging has seen a significant increase in its use in recent years, despite the fact that MRI in pregnancy has traditionally focused on the fetus. In addition to allowing for the evaluation of function, MRI with a large field of view and high contrast characterize resolution makes it possible to the anatomy of the placenta, particularly in situations where ultrasound cannot be used to pinpoint the exact location, such as when a placenta accreta is suspected. The anatomical evaluation of the placenta using MRI appears to be particularly useful regardless of the maternal body habitus or fetal position. In fact, when fetal concerns are the reason for the examination, the placenta receives surprising little attention during MRI. As a result, we believe that the radiologist should pay attention to certain aspects of the placenta and describe them in the MRI report. The normal placenta, abnormal aspects of the placenta that should be identified on fetal MRI and placental anomalies for which placental MRI may be indicated will all be covered in this overview, along with common features seen in imaging. There is a high risk of obstetrical complications and long-term health issues in children born using assisted reproductive technologies; however, the underlying mechanisms are not completely understood.

Keywords: MRI; Placenta; *In Vitro* Fertilization (IVF); Differentially Methylated Regions (DMRs); Diabetes mellitus

Introduction

The growth and health of the fetus are closely linked to normal placental function. We used a mouse model to investigate how *In Vitro* Fertilization (IVF) affected glycogen storage in placentas because of the significance of glycogen metabolism in placentas. Placenta Accreta Range (PAS) is a condition of unusual connection of the placenta, including placenta accreta, placenta increta and placenta percreta. Because the placenta cannot separate on its own, causing continuous bleeding, this condition can be fatal. One option for treating PAS is a hysterectomy followed by a c-section [1]. During the PAS patient's operation, there was a significant liability for injuries to their urinary tract. Pandemics of infectious diseases brought on by newly emerging viruses have occurred at various points in history [2]. Pregnant women have a higher risk of contracting the diseases and experiencing negative outcomes, as evidenced by previous outbreaks. However, while some viruses appear to have no effect on pregnancy, others may pose significant health risks to the mother and her unborn child. The infection begins when viral surface proteins bind to specific receptors on the cell membrane of the host cell. The various outcomes may be explained by the different molecular characteristics of these proteins during pregnancy, which may identify specific target cells in the placenta. Information on the Variola, Influenza, Zika and Corona viruses, with a focus on their surface proteins, effects on pregnancy and potential target placental cells, is presented in this review. Understanding viral entry during pregnancy and developing strategies to reduce the occurrence of obstetrical issues in both current and future infections will both benefit from this. Due in large part to the difficulties associated with allelic analyses in outbred animals, our understanding of genomic imprinting in primate's lags behind that of mice. We profiled uniparental monkey embryo transcriptomes, DNA methylomes and H3K27me3 in order to comprehend primate imprinting dynamics [3]. To find germline Differentially Methylated Regions (DMRs) in somatic tissues, we further developed the SNP free TARSII and CARSII methods. In uniparental monkey embryos, our comprehensive analyses revealed that Paternal-Biasedly Expressed Genes (PEGs) are correlated with allelic DNA methylation, but not H3K27me3.

Literature Review

It is interesting to note that placenta-enr ched DMRs in primates differ from PEG-associated DMRs in early embryos. Surprisingly, cloned monkeys lack the majority of germline DMRs that are specific to the placenta [4]. Our research shows imprinting defects in cloned monkey placenta, defines primate developmental imprinting dynamics, and establishes SNP-free germline DMR identification methods, all of which can be used to improve primate cloning. Maternal mortality and morbidity are significantly impacted by placenta accreta syndrome. To overcome this disorder, which poses a threat to both the mother and the fetus, a multidisciplinary approach is therefore necessary. During pregnancy, the function and growth of the placenta are influenced by the mechanistic Target of Rapamycin (mTOR) pathway. The mTOR pathway responds to growth factors and the availability of nutrients, which control the and expression of proteins the growth of cells. Numerous obstetric complications are linked to the onset of disrupted mTOR signaling [5]. Various mTOR associated proteins were found to be expressed differently in the placenta during Preeclampsia (PE), Gestational Diabetes Mellitus (GDM), Intrauterine Growth Restriction (IUGR) and normal gestation (Control). Activated proteins (phospho) pinpointed by were immunohistochemistry, mTOR, pp70, 4EBP1, AKT and ERRK [6].

Discussion

Additional mTOR associated genes were found to be expressed differently in the placenta through the use of a realtime PCR array. pAMPK protein was detected by Western blot. We noticed: A rise in pmTOR during GDM and a fall in pmTOR during IUGR and PE; a rise in pp70 during IUGR and a fall in pp70 during GDM and PE; a rise in p4EBP1 during GDM, IUGR, and PE; a rise in pAKT during GDM; a rise in pERK during IUGR and a difference in the expression of We that the development of conclude these obstetric complications is specifically influenced by regulation of the mTOR pathway. These diseases may be alleviated if this pathway is altered with new insights. High rates of morbidity and mortality continue to result from the ongoing SARS-CoV-2 pandemic. Placental pathological changes and severe maternal and neonatal outcomes have been described during pregnancy. Using Precision Cut Slices (PCSs) of the human placenta, we assess SARS-CoV-2 infection at the maternal-fetal interface. Surprisingly, SARS-CoV-2 exposure causes a full replication cycle and the release of the infectious virus in placenta PCSs. Additionally, ACE2 expression levels are related to the susceptibility of placental tissue to SARS-CoV-2 replication [6]. Syncytiotrophoblasts, cytotrophoblasts, the villous stroma and possibly Hofbauer cells all contain viral proteins or RNA. Interferon type III transcripts are upregulated by one order of magnitude when placenta PCSs are infected with SARS-CoV-2, but neither cytotoxicity nor a proinflammatory cytokine response are observed.

Conclusion

In conclusion, our findings provide a foundation for further research into the biology of SARS-CoV-2 at the maternal-fetal interface and demonstrate that the virus can infect and spread through the human placenta. Prenatal prediction of Placenta Accreta Spectrum (PAS) can lower maternal morbidity and mortality because PAS is a life threatening obstetric

complication. The clinical factor associated with PAS were the focus of this prospective cohort study. Placental disorders are the cause of a lot of bad things that happen during pregnancy. Due to the overlap between identifiable placental pathology and pregnancy outcomes, it has been challenging to determine the underlying cause of many of these disorders. The absence of a suitable control placenta may be the cause of this confusion. It is uncommon to find an ideal control placenta that is unrelated to adverse pregnancy outcomes. At the Pregnancy Collaboration (CoLab), we suggest that Global researchers might find a solution in our pooled database. The metabolic pathways and molecular mechanisms that regulate development and functions remain placental poorly understood, despite the fact that the human placenta performs numerous functions that are necessary for a pregnancy to be successful. In normal physiology, the breakdown of the essential amino acid tryptophan plays a number of important roles, one of which is inflammation. In the placenta, Indoleamine 2,3 Dioxygenase 1 (IDO1) acts as a mediator for the kynurenine pathway, which is responsible for roughly 90% of the breakdown of tryptophan. Changes in IDO1 activity or expression cause fetal resorption and a condition similar to preeclampsia in pregnant mice Preeclampsia, in utero growth restriction and recurrent miscarriage have all been linked to decreased IDO1 expression at the maternal fetal interface in humans. The sum of these observations suggests that IDO1 plays an important role in maintaining a healthy pregnancy. The precise temporal, cell-specific, and inflammatory cytokine mediated patterns of IDO1 expression in the human placenta have not been thoroughly characterized throughout gestation, despite these significant functions.

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