9th Yazd International Congress and Student Award on Reproductive Medicine with 4th Congress of Reproductive Genetics

Key Lectures

K-8
Feto-maternal immunological cross talk

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Not only embryo aneuploidies, but also the other factors such as immune-related maternal tolerance to pregnancy might contribute to implantation failure or miscarriage.. Maternal tolerance begins at the uterine level following by successful adaptation to the semiallogeneic fetus is a complicated process. The fetal cells that come into direct contact with the mother's immune cells in the uterus are uterine natural killer (uNK) cells in trophoblast cells, the layer that surrounds the blastocyst. The key function of the materno-fetal tolerance process is the remodeling of the spiral arteries, with the destruction of the media by invading extra villous trophoblasts (EVT) cells. The EVTs that invade the maternal decidua have fetal origin, and express high levels of human leukocyte antigen-C (HLA-C) recognized by uNK killer cell immunoglobulin-like receptors (KIRs). Maternal and paternal HLA-C allotypes are expressed at the same time and at high levels on the EVT cell surface. Placentation is regulated by interactions between maternal KIRs expressed by uNKs, and fetal HLA-C molecules, expressed by EVTs. Insufficient invasion of the uterine lining by trophoblasts and vascular conversion in the decidua are thought to be the primary defect among disorders such as recurrent miscarriage, preeclampsia, and fetal growth restriction. This process is regulated by the interaction between maternal KIRs, expressed by the uNKs, and their ligand HLA-C, expressed by EVTs. Pregnancies are at increased risk of recurrent miscarriage in mothers who are homozygous for KIR haplotype A (KIR AA) when the fetus has more HLA-C2 genes than the mother does and when additional fetal HLA-C2 alleles are of paternal or oocyte donor origin. There is a lower live birth rate and an increased miscarriage rate after double embryo transfer in KIR AA patients. In addition, the live birth rate decreases significantly as the embryo HLA-C2 load increases. The maternal immune system is one of the main actors at the maternal–fetal interface, and its lack of activation but not a rejection seems to influence the placentation and pregnancies outcomes.

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