

Analysis of tight junctions in placentas affected by chorioamnionitis: in vivo and in vitro analysis

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The human placenta and fetal membranes provide a barrier regulating the transfer of materials between the mother and the developing fetus throughout gestation. Chorioamnionitis is an important risk factor for preterm delivery that is associated with high perinatal morbidity and mortality. Chorioamnionitis is the term applied to infections of the placenta and membranes resulting in high concentrations of IL-1beta, IL-6, IL-8 and TGF-beta in the amniotic fluid (D'Alquen et al., 2005). With progression of inflammation, immune cells penetrate blood vessels and infiltrate the umbilical cord, resulting in funisitis (Romero and Mazor, 1988). In normal conditions the two important physical entities in endothelial/epithelial paracellular clefts are adherens junctions and tight junctions.

Tight junction governs the paracellular movement of water, solutes and immune cells, through the intercellular space creating a boundary between the apical and basolateral sides of cellular barriers (Gruenheid and Finlay, 2003). We have evaluated the localization of tight junctions studying the Zonula Occludens-1 (ZO-1) and Occludin expressions as well as the localization of adherent junctions, testing the expression of VE-cadherin and beta-catenin in placentas from normal gestations, from preterm idiopathic deliveries and from chorioamnionitis by immunohistochemistry. In addition, we have evaluated the mRNAs by real time PCR, the protein levels of these molecules by Western blot analysis in placental tissues, and to better clarify the action of some cytokines on occludin we performed in vitro analysis of HUVEC cultures.

Our more striking result is the decrease of occludin expression in placentas from chorioamnionitis and an evident action of the cytokines on this molecule.

References

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