REDUCED EXPRESSION OF CALCIUM SENSING RECEPTOR IN THE HUMAN PLACENTA OF MOTHERS WITH GESTATIONAL DIABETES MELLITUS ASSOCIATED WITH EARLY POSTNATAL HYPOCALCEMIA IN THEIR NEWBORNS.

E. Gole, C. Sfikas, P. Nicolaidou, A. Papadopoulou

Background: Gestational diabetes mellitus (GDM) is associated with disturbances in the anatomy and physiology of the placenta as well as with alterations in the expression levels of many proteins involved in placental endocrine pathways. GDM correlates with an increased risk of adverse neonatal outcomes, including a higher rate of early postnatal hypocalcemia.

Aim: We examined whether the expression level of Calcium Sensing Receptor (CaSR), a protein with a particular role in calcium (Ca) homeostasis, is altered in placentas from GDM mothers and whether CaSR expression affects cord blood and postnatal Ca levels in their neonates.

Methods: Our study consisted of 37 pregnant women with GDM and 36 healthy women and their neonates. CaSR expression was evaluated with immunohistochemical analysis of paraffin embedded sections of term placentas, obtained after delivery from both samples and controls. Ca levels were measured in cord blood serum and in the serum of their infants at 2nd day of life.

Results: CaSR was predominantly localized in syncytiotrophoblasts but weaker immunostaining was also detected in villous and extravillous cytotrophoblasts in both samples and controls. Compared with the normal group, CaSR expression was significantly lower in GDM placentas in syncytiotrophoblasts (P<0.001), as well as in villous cytотrophoblasts (P<0.001) and extravillous cytотrophoblasts (P<0.001). In the GDM group, reduced CaSR immunostaining was significantly correlated with lower Ca cord blood levels both in the synctiotrophoblasts (P=0.042) and the extravillous cytотrophoblasts (P=0.002) but not in the villous cytотrophoblasts.

Conclusion: Our results suggest that CaSR expression is downregulated in placentas of GDM mothers. This alteration may give an explanation for the increased rate of early postnatal hypocalcemia observed in GDM infants.