

Placental expression of the angiogenic growth factors in gestational diabetes mellitus

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Objective: To explore the levels of vascular endothelial growth factor (VEGF), endothelial NO synthase (eNOS), insulin-like growth factor -1 receptor (IGF1R) and mast cells (MC) expression in GDM placenta.

Methods: 55 placentas were divided into 4 groups: GDMA1 (n=20, group I-diet); GDMA2 (n=20, group II-insulin); preeclampsia (n=10, group III); normal (n=5, group IV). Immunohistochemistry tests were performed using antibodies to NOS-3, VEGF IGF1R, α -amylase.

Results: The highest area of expression of VEGF was observed in GDMA1 – 5,27 %, which was twice higher than in control group 2,86 % and in preeclampsia 2,07% ($p<0,01$). Expression VEGF in GDMA2 was lower – 0,75%. The biggest area of expression of eNOS was observed in group I – 7,28 % and the smallest in group II - 1,1% ($p<0,01$). The expression of eNOS in control and preeclampsia groups was in average levels - (3,9%-2,05%). The area of α IGF1R expression was the biggest in GDM - (3,44%-5,18%). The smallest area was detected in preeclampsia – 1,02% ($p<0,01$). The area in the control group was average – 2,47%. In GDM the number of MC was significantly higher by 1,5 times compared with groups III and IV.

Conclusion: In GDMA1, the increased synthesis of VEGF and eNOS that lead to hypervascularization terminal villi in the placenta. In GDMA2, the decrease of expression VEGF and eNOS leads to a higher rate of preeclampsia and IUGR. The increased level of IGF1R in GDM placenta may be a possible cause of fetal macrosomia. The minimal area of IGF1R expression in preeclampsia maybe one of the reasons for IUGR. Changes in the number of MC in the placenta can detect placental pathology associated with local inflammation.

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