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Newborn genotypes associated with endothelial dysfunction as a risk factor for mild and severe preeclampsia

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Context: Fetal genes associated with endothelial dysfunction might contribute to the pathogenesis of preeclampsia.
Objective: To investigate the effects of genetic variations in vascular endothelial growth factor (VEGF), superoxide dismutase (SOD), deiodinase (D1 and D2) on the risk of preeclampsia.
Patients and methods: We determined 936C/T-VEGF, Ala-9Val (MnSOD), Arg213Gly (EC-SOD), C785T-D1, Thr22Ala (D2) genotypes in 69 newborns of mothers with mild and severe preeclampsia and 94 newborns of mothers with normal pregnancies using PCR-RFLP analysis.
Results: A higher frequency of homozygous and heterozygous 936C/T-VEGF, Ala-9Val-MnSOD, Arg213Gly-EC-SOD, C785T-D1, Thr22Ala-D2 genotypes was found in newborns of mothers with mild and severe preeclampsia compared to newborns of mothers with normal pregnancies. The risk to develop mild preeclampsia was 9.16 ($p<0.001$) in carriers of the C785T-D1 genetic variation. The risk to develop severe preeclampsia was 4.5 ($p=0.089$), 2.96 ($p=0.05$), 6.53 ($p<0.001$) in carriers of the 936C/T-VEGF, Thr22Ala-D2, Arg213Gly-EC-SOD variants. Women whose newborns were carriers of homozygous or heterozygous 936C/T-VEGF, C785T-D1, Thr22Ala-D2, Ala-9Val-MnSOD, Arg213Gly-EC-SOD genotypes had a 3.46-fold ($p<0.001$), 14.2-fold ($p<0.001$), 2.64-fold ($p=0.037$), 2.32-fold ($p=0.069$), 4.8-fold ($p=0.001$) increased risk for mild preeclampsia. Women whose newborns were carriers of homozygous or heterozygous 936C/T-VEGF, C785T-D1, Thr22Ala-D2, Arg213Gly-EC-SOD genotypes had a 3.16-fold ($p=0.014$), 2.4-fold ($p=0.059$), 4-fold ($p=0.003$), 5.4-fold ($p<0.001$) increased risk for severe preeclampsia. Significantly higher HDL (mg/dl, 53.93±20.95 vs. 37.65±4.94, $p=0.015$), TG (mg/dl, 271.22±55.67 vs. 223.47±56.82, $p=0.021$) and sFlt-1 (pg/ml, 989.335±549.04 vs. 721.85±430.38, $p=0.031$) levels were found in preeclamptic women whose newborns were carriers of the homozygous 936C/T-VEGF genotype compared to preeclamptic women whose newborns were negative for this genotype. Also, significantly higher FT4 (ng/dl, 1.26±1.07 vs. 1.06±0.34, $p=0.034$) and lower FT3 (pg/ml, 2.48±0.76 vs. 2.73±0.71, $p=0.015$) levels were detected in preeclamptic women whose newborns were carriers of the homozygous Thr22Ala-D2 genotype compared to preeclamptic women whose newborns were negative for this genotype.
Conclusion: The study confirms the influence of newborn genotypes associated with endothelial dysfunction on mild and severe preeclampsia risk.

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