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TITLE

TWO GENETIC THEORIES ON PREECLAMPSIA: THROMBOPHILIAS AND ENDOTHELIAL DYSFUNCTION. INFLUENCE ON THE SEVERITY AND PROGNOSIS OF PREECLAMPSIA

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ABSTRACT

Context:Thrombophilias and endothelial dysfunction could represent risk factor of adverse pregnancy outcomes such as preeclampsia.

Objective: to analyse the genetic contribution on preeclampsia, by investigating the most common genetic variations associated with thrombophilias (factor V Leiden, C677T-MTHFR), and endothelial dysfunction (936C/T-VEGF, Glu298Asp-eNOS). Interaction between maternal and fetal genotypes were also taking into account.

Materials and methods:We included in the study 125 preeclamptic women and 131 normal pregnant women. We also evaluated 69 newborns with mothers with preeclampsia and 94 newborns with mothers with normal pregnancies. The genotypes were determined using PCR-RFLP methods.

Results:Increased frequency of all four genetic variations was identified in preeclamptic women as compared with women with normal pregnancy. The risk to develop preeclampsia was 9.47 (p<0.001), 5.38 (p<0.001), 1.49 (p=NS) and 3.44 (p<0.001) in carriers of factor V Leiden, C677T, 936C/T and Glu298Asp. Genetic analysis function the severity of preeclampsia showed an OR of 16.63 (p<0.001), 11.95 (p<0.001), 3.3 (p=0.005) and 4.67 (p<0.001) for severe preeclampsia in association with factor V Leiden, C677T, 936C/T and Glu298Asp. The presence of C677T mutation in both mother and newborn increased the risk for preeclampsia to 6.48 (p<0.001). Also, the presence of factor V Leiden in mother independently of newborn genotypes increased the risk to 6.14 (p<0.001). Carriers of factor V Leiden, C677T and 936C/T delivered at lower gestation age, neonates with lower birth weight as compared with noncarriers.

Conclusions: Our study confirms the influence of thrombophilias and endothelial dysfunction on the severity and prognosis of preeclampsia. The study confirms the interaction between maternal and fetal genotypes as modulating risk factor in preeclampsia.

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