

Dysregulated levels of novel circulating autoantibodies in preeclampsia

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Introduction: The contributions of the T-cell population and Natural Killer cells have been extensively studied in relation to pregnancy and adverse pregnancy outcomes. Less attention has been given to the B-cell population although maternal levels of circulating autoantibodies against the angiotensinII type 1(AT1)-receptor and antiphospholipid antibodies (aPLs) have been shown to be associated with preeclampsia. Members of the PAR (protease activated receptor) family and VEGF (vascular endothelial growth factor) family have previously been shown to be implicated in preeclampsia.

Objectives: The aim of this exploratory study was to study levels of IgG autoantibodies against these families of PAR and VEGF proteins in pregnant women with and without preeclampsia.

Methods: We developed novel immunoassays detecting levels of IgG autoantibodies against PAR-1, PAR-2, PlGF (Placental Growth Factor), VEGF-A, VEGF-B, VEGF-receptor 1 (VEGFR-1) and VEGF-receptor 2 (VEGFR-2).

Results: We found that levels of autoantibodies against PAR-1, PAR-2, VEGF-A, VEGF-B, PlGF, VEGFR-1 and VEGFR-2 were lower ($p < 0.05$) in preeclamptic pregnancies ($n=42$) compared to normotensive pregnancies ($n=46$). Clinical features associated with augmented risk for the preeclampsia syndrome (such as primigravidity, primiparity, obesity and a small for gestational age fetus) were also associated with lower levels of the autoantibodies we investigated, although not always reaching statistical significance.

Conclusion: We speculate that these autoantibodies may play a protective role in the development of preeclampsia and are involved in the dysregulation of the PAR and VEGF pathways.