Abstract

THESIS: EFFECT OF ANTICARDIOLIPIN ANTIBODIES ON VASCULAR ENDOTHELIAL GROWTH FACTOR – A SECRETION FROM HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS

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Antiphospholipid syndrome (APS) is an autoimmune disorder defined as the persistent presence of antiphospholipid antibodies (aPL) associated with recurrent thrombotic events and/or fetal loss. The mechanisms for fetal loss in this syndrome have not yet been clearly explained, although several hypotheses based on experimental data have been put forward. It has been shown that proliferation of human umbilical vein endothelial cells (HUVECs) decreased significantly in cultures that contained sera positive for anticardiolipin antibody activity collected from patients with recurrent fetal loss. We explored the effect of ACAs on Vascular Endothelial Growth factor- A (VEGF-A) secretion of cultured HUVECs. VEGF appears to be the most endothelial cell-specific and unequivocal angiogenic factor. In vitro, VEGF causes endothelial cell proliferation and migration; in vivo, it is potently angiogenic and causes vascular permeability. We determined the effect of ACA IgG 40µg/ml and 80µg/ml on VEGF secretion using ELISA. The results were measured in mean ± SD and expressed in pg of VEGF/5 x 10⁴ cells. P ≤ 0.05 considered significant as compared to control. The results showed that ACA increases VEGF-A secretion and/or may bind at VEGF-A binding sites. This finding may be useful for finding therapies for patients with recurrent miscarriages.