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Proimflammatory cytokines and vascular endothelial growth factor levels at the peripheral and coronary atherosclerosis

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The purpose of the research is studying proimflammatory cytokines: interleykin-6 (IL-6), tumor necrosis factor alpha (TNF-a), C-reactive protein (CRP) and marker of endothelial proliferation and migration - vascular endothelial growth factor (VEGF) levels at an atherosclerosis of various localizations. IL-6, TNF-a, CRP, VEGF was analyzed at 28 patients with a peripheral (in iliac-femoral arterial pool) atherosclerosis (PA) and at 80 patients with coronary atherosclerosis (CA). CRP was made in automatic biochemical analyzer "VITROS-350" (Germany). IL-6, TNF-alpha, VEGF were measured using ELISA kits.

Results: IL-6 level and CPR was increased both at PA and CA patient, IL-6 - in 12,2 and 9,2 times and CRP - in 3,1 and 2,8 times concerning to the control respectively. There was significant difference in FNO-a concentration between PA and CA patients ($p<0,05$), it was increased in 1,6 and 2,3 times respectively at PA at CA patients. VEGF level was increased in PA patients at 2, 2 times concerning the CA patients. This data suggest that ischemia of peripheral muscles in PA patient's leads endothelial proliferation and collateral bloodstream, which are more intensive in contrast with CA patients due to VEGF concentration increasing. This data can be used in therapeutic angiogenesis conception development.

Conclusion: Proimflammatory cytokines IL-6, TNF-a and CRP were increased both at PA and CA patients, but more intensively at PA. Probably, chronic ischemia of peripheral tissues leads local inflammatory reaction and VEGF expression.

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Coagulation system: Novel concepts for novel therapeutics

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The initial "waterfall" or "cascade" model for coagulation was proposed in 1964 by MacFarlane, Davie and Ratnoff. Obscure in this model, is the role of the contact (intrinsic) pathway. As the known stimulus for it, has been largely non-physiological such as glass and kaolin. Also people with deficiency of factor XII show no significant bleeding tendencies. While the connection between coagulation, inflammation and thrombosis is well known observation, however its exact mechanism was not clear. In recent years there has been a growing body of literature supporting the role of negatively charged, poly anionic linear polymers such as polyphosphate (poly-P) and extracellular nucleic acids (RNA and DNA) in thrombosis and inflammation. Poly-P is a highly anionic, linear polymer of orthophosphate, which is stored as metachromatic granules in many cells. The recent discovery that the dense granules of the platelets are actually storage of (Poly- P) and the observation that (Poly- P) can strongly stimulate contact pathway has opened a way for a new understanding of coagulation system. Activation of the contact system by long-chain (poly-p) can also be strongly pro-inflammatory, in a manner dependent on factor XII activation and release of bradykinin from high molecular weight kininogen. It was also noted that platelet (poly-p) causes an approximately 3000-fold increase in the rate of back-activation of factor XI by thrombin, enhances the rate of factor V activation to Va by factor XIa. While (poly-p) is released mainly from platelets, extracellular DNA and RNA are released mainly from neutrophils; neutrophil extracellular traps (NETs). Both (poly-p) and (NETs) almost have the same function.

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