International Conference "ADVANCES IN PNEUMOLOGY"
Poznań, 6 – 7 June, 2008

MODULATORY EFFECTS OF SERA FROM PATIENTS WITH VARIOUS TYPES OF PULMONARY FIBROSIS ON MONONUCLEAR CELL-INDUCED ANGIOGENESIS IN RELATION TO RADIOLOGICAL AND FUNCTIONAL PULMONARY CHANGES

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Pulmonary fibrosis occurs in many lung diseases. Angiogenesis plays an important role in the pathogenesis of idiopathic pulmonary fibrosis, but whether that is the case in other fibrotic pulmonary disorders is unclear. The aim of the study was to examine the effect of sera from patients with different type of pulmonary fibrosis on angiogenesis induced by human mononuclear cells (MNC) in relation to radiological, clinical and functional status. The study population consisted of 32 patients with idiopathic pulmonary fibrosis (IPF), 11 patients with drug-induced pulmonary fibrosis (DIPF), 6 with cryptogenic organizing pneumonia (COP), 12 patients with silicosis, 13 with systemic sclerosis (SSc) and 20 healthy volunteers. As an angiogenic test we used animal model of leukocyte induced angiogenesis assay. Spirometry, whole-body plethysmography, static lung compliance (Cst), and diffusing capacity of the lungs for CO (DLco) were performed in all patients. Sera from IPF, SIL, and COP patients significantly stimulated angiogenic activity of MNC compared to sera from healthy donors and from DIPF patients ($p<0.001$). However, sera from healthy donors and DIPF significantly stimulated angiogenic activity of MNC compared with the control with PBS and SSc ($p<0.001$). Angiogenic activity of sera did not correlate with pulmonary function of patients with pulmonary fibrosis. However, proangiogenic effect of sera from systemic diseases patients depended on the radiological changes. Sera obtained from patients with pulmonary fibrosis constitute the source of mediators modulating angiogenesis, but the pattern of reaction is different in various diseases. Sera from IPF, SIL, COP patients stimulate neovascularisation, but sera from SCL patients exert an inhibitory effect on angiogenesis.