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V - V
IgE- e - V IgA- IgA- -IV
V- V.
V, IgA IgE- - V
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1, TIMP-1, MMP-9 TGF-1.

INVESTIGATION OF SOME CLINICAL AND IMMUNOLOGICAL PARAMETERS IN PATIENTS WITH SYSTEMIC FORMS OF LUPUS ERYTHEMATOSUS AND SCLERODERMA

SUMMARY: The aim of this clinical and immunological study is investigation of clinical symptoms and some basic parameters of humoral and cellular immune response to elastin and collagens in patients with Lupus Erythematosus (SLE) and Scleroderma Progressiva (SSc), looking for the connection between clinical experience of this connective tissue diseases (CTD) and immune imbalance of ECM' (extracellular matrix) proteins.

To achieve the aim of our research work, we used ACR classification criteria for diagnosis of CTD, evaluated the index of disease activity of Systemic LE (SLEDAI) and the index of skin thickening degree (TSS) in SSc patients. Using "sandwich" variant of ELISA, we proved the quantitative changes in elastin and collagen turnover – increasing of ECM' proteins degradation (high levels of elastin and collagen derived peptides), as well as autoimmune response to them (high levels of antielastin and anticollagen antibodies of all immunoglobulin subsets).

We determined cell-mediated immuno-deficiency (CMID) in all of the testing groups – patients with SLE, patients with SSc and healthy controls. Positive intradermal skin test to -elastin antigen present only suffers with CTD and CMID. This fact is an important evidence of the specific character of Delayed-type hypersensitivity reaction. Correlations between lupus patients with IgE- and IgM- antielastin antibodies, and scleroderma patients with IgE- and IgA- AEAbs, are incontestable argument of connective tissue immune imbalance. Elastolysis results as high levels of elastin derived products. Collagen type IV derived peptides and increasing of anticollagen IV antibodies is normal for CTD. High levels of IgA- and IgE- AColl IV Abs are typical fact also.

IgA- AColl IV Abs., persisting in these disorders, are a sign of stimulating of delayed type immune response to collagen IV degradation products enhancing, because of intensive collagenolysis. Compared to healthy controls, the collagen derived peptides' levels are significantly higher during exacerbation of the disease, as well as in remission. We accept, that these immunological changes are markers of the basement membranes lesions in patients with CTD.

Quantitative immunological characteristics change themselves in all different phases of disease evolution. The elastin and collagen immune parameters, we describe in this research work, are easy of access and reproduction. They ought to be use for evaluation of connective tissue turnover, lesions of skin', blood vessels' or internal organs' basement membranes. In constellations with other clinical signs, as age, duration and clinical form of the disorders, concomitant immune deficiency, organ injury, treatment' complications, they may bring up for discussion, concerning disease activity, progression and prognosis of autoimmune CTD.