AUTOIMMUNE CONNECTIVE TISSUE DISEASES

SERUM ENDOGLIN LEVEL IN PATIENT WITH SYSTEMIC SCLEROSIS FIBROSIS

Zofia Gerlicz-kowalczuk [1] - Bozena Dziankowska-bartkowiak [2] - Elzbieta Dziankowska-zaborszczyk [3] - Joanna Narbutt [1]

Medical University Of Lodz, Departament Of Dermatology, Paediatric Dermatology And Oncology, Lodz, Poland [1] - Medical University Of Lodz, Departament Of Dermatology And Venerology, Lodzi, Poland [2] - Medical University Of Lodz, Departament Of Preventive Medicine, Lodz, Poland [3]

Introduction: Systemic sclerosis (SSc) is an autoimmune connective tissue disease characterized by deviations in immunological investigations, generalized microcirculation disorders and progressive tissue fibrosis. So far, etiopathogenesis of systemic sclerosis has not been fully understood, although fibrosis of the skin and internal organs play a key role in the progression of the disease. On the other hand, endoglin is coreceptor for TGF-β and have ability to enhance fibrotic signaling and is a positive regulator of fibrosis.

Objective: The aim of the study was to determine serum level of endoglin in patients with SSc.

Materials and Methods: Serum level of endoglin was measured in 26 SSc patients and 10 healthy controls by a specific enzyme-linked immunosorbent assay. The correlation between serum level of endoglin and internal organs changes were assessed.

Results: There was statistical difference in serum level of endoglin in SSc patients compared with healthy controls (4.65 ± 1.02 vs. 3.62 ± 0.60 ng/dl, $p < 0.01$). Serum level of endoglin were not different between lSSc and dSSc. Significant correlations among serum endoglin levels and organ changes were also found in SSc patients. Serum level of endoglin was higher in SSc patient with gastrointestinal tract involvement (4.76 ± 1.24 vs. 4.47 ± 0.54 ng/dl, $p = 0.03$), but lower with pulmonary changes in the course of the disease (4.05 ± 0.58 vs. 4.97 ± 1.05 ng/dl, $p = 0.03$).

Conclusion: The increased endoglin concentration in the serum in patients with SSc, may contribute to progression of fibrosis.