— Sat-155 —

ELEVATED SERUM INTERLEUKIN-18 IN PATIENTS WITH SYSTEMIC SCLEROSIS

The Catholic University of Korea, Center for Rheumatic Diseases; Hyo-Jong Kang, Myung-Soo Lee, Seung-Jae Hong, Kyung-Su Park, Chong-Hyeon Yoon, Do-June Min, Jun-Ki Min, Wan-Uk Kim, Sung-Hwan Park, Chul-Soo Cho, and Ho-Youn Kim.

OBJECTIVE : Although the pathogenic mechanism of systemic sclerosis has not been established, T cell hyperactivity is thought to be important in the pathogenesis of SSc. IL-18, formerly called IFN--inducing factor, is a novel cytokine which is thought to play an important role in the TH1 response. In this study, we investigated the association of circulating IL-18 in SSc and its relationship with clinical manifestations and other T-cell stimulatory cytokines in SSc patients.

PATIENT and METHOD : We measured serum levels of IL-18 in 54 patients with SSc and 27 healthy controls. At the time of sampling clinical and laboratory findings were assessed including the presence of interstitial lung disease, reflux esophagitis, digital ulceration, anti-topoisomerase antibody and serum IL-15 and IFN- level in SSc patients.

RESULT : The serum level of IL-18 was significantly higher in SSc patients than in healthy controls (290.2 130.0 pg/ml vs. 122.6 20.6 pg/ml, $p < 0.01$). Patients with diffuse type SSc had higer levels of serum IL-18 (n = 32) than those with limited type (n = 22) (325.4 137.3 pg/ml vs. 239.3 102.1 pg/ml , $p < 0.05$). However, serum IL-18 level was not associated with the presence of interstitial lung disease, reflux esophagitis, digital ulceration, anti-topoisomerase antibody. Serum level of IL-18 showed positive correlation with that of IL-15 and IFN- in SSc patients (IL-15 : r = 0.50, p <0.01, IFN- ; r=0.53, $p < 0.01$).

CONCLUSION : These data suggest that IL-18 may be involved in the pathogenesis of systemic sclerosis.

— Sat-156 —

Elevated soluble fms-like tyrosine kinase and vascular endothelial growth factor in patients with systemic sclerosis.

The Catholic University of Korea, Center for Rheumatic Diseases; Seung-Jae Hong, Kyung-Su Park, Myung-Soo Lee, Chong-Hyeon Yoon, Do-June Min, Jun-Ki Min, Wan-Uk Kim, Sung-Hwan Park, Chul-Soo Cho, and Ho-Youn Kim.

OBJECTIVE : Systemic sclerosis (SSc) is characterized by microvascualr injuries followed by narrowing and eventual obliteration. Soluble fms-like tyrosine kinase (sFLT-1), a soluble receptor of vascular endothelial growth factor (VEGF), is reported to have antagonistic activity against VEGF which is a highly specific mitogen for vascular endothelial cells. We tried to investigate the association of sFLT-1 and VEGF in SSc patients.

PATIENT and METHOD : We measured serum levels of sFLT-1, VEGF, and TGF- in 54 patients with SSc and 27 healthy controls. SSc patients was divided into 2 groups according to skin involvement (diffuse type 32, limited type 22). At the time of sampling, we assessed clinical and laboratory findings including the presence of digital ulceration, reflux esophagitis, interstitial lung disease, and anti-topoisomerase antibody in SSc patients.

RESULT : The serum level of sFLT-1 and VEGF were significantly higher in SSc patients than in healthy controls (154.3 92.3 pg/ml vs. 77.2 27.9 pg/ml, $p < 0.01$, 406.6 233.5 pg/ml vs. 178.9 39.6 pg/ml, $p < 0.01$, respectively). Serum level of sFLT-1 tended to be lower in patients with diffuse type disease than in those with limited type disease (141.9 103.6 pg/ml vs. 172.2 72.2 pg/ml , $p > 0.05$). However, serum level of VEGF was higher in those with diffuse type disease than in those with limited type disease (484.3 256.0 pg/ml vs. 292.1 133.7 pg/ml, $p < 0.01$). The level of sFLT-1 showed negative correlation with that of VEGF and TGF- (VEGF ; r = -0.55, p < 0.01, TGF- ; r = -0.46, $p < 0.01$). VEGF level showed positive correlation with TGF- level (r = 0.48 , $p < 0.01$). There was no significant difference in sFLT-1 level according to the presence of above clinical and laboratory findings. **CONCLUSION** : Our data suggests that elevated serum sFLT-1 and sVEGF is associated with SSc and that sFLT-1 may play a certain role in the regulation of VEGF activity in patients with SSc.