Methods: We included eighty-five SSc patients, fulfilling the 2013 ACR/ EULAR classification criteria, attending the Regional Rare Disease Center of Policlinico Umberto I of Rome. Fifty presented significant CRD at non-invasive diagnostic techniques (12 Lead ECG, 24-hour Holter ECG). Demographic, clinical, conventional cardiovascular risk factors were examined; instrumental and laboratory assessments were obtained, together with ECG recordings. Thirty-five SSc patients without pathologic finding at ECG traces, matched for demographic and clinical features, were recruited as the control group. In all cases, after obtaining written informed consent, blood samples were taken to measure serum levels of leptin using an ELISA assay (Life Technologies-Italy). We detected significantly higher median values of serum leptin in SSc patients with CRD compared to the control group (12027 pg/ml IQR 12314 versus 6392 pg/ml IQR 7103; p < 0.0009). Additionally, SSc patients with a BMI >25 kg/m² (31 cases) as well as those with PF (47 cases) showed a significantly higher median serum leptin levels compared to those with BMI <25 kg/m² (13161 pg/ml IQR 13610 versus 8187 pg/ml IQR 8255; p = 0.0008) and those without PF (11740 pg/ml IQR 11940 versus 7616 pg/ml IQR 7855; p = 0.0079).

Conclusion: To our knowledge this is the first report on high serum levels of leptin in SSc patients with CRD that also confirms its increase in those cases with a BMI >25 kg/m² and with PF according to scientific literature data. The role of leptin in the pathogenesis of SSc remains unclear although it is already known its involvement in the development of cardiac fibrosis during other chronic diseases. On the basis of these results we speculate on leptin involvement in the pathogenesis of CRD during SSc, although further studies are needed with larger cohort of patients.

References:

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