Background: Resistin is a soluble factor produced by adipose tissue, implicated in the regulation of inflammatory processes and in microvascular damage. When incubated with resistin, endothelial cells respond by a greater production of endothelial dysfunction (ED) in many cardiovascular and autoimmune diseases, such as diabetic-endothelial interaction. Circulation 2003;108:736–40.

Objectives: To evaluate the association between baseline serum resistin levels and the development of new digital ulcers in patients affected by systemic sclerosis.

Methods: We conducted a one-year prospective cohort study. Patients with SSc and healthy controls (HC) were consecutively enrolled. Baseline serum resistin was assessed by commercial ELISA kit. The development of new DUs was prospectively evaluated during the follow-up after the cross-sectional point in which the resistin levels were measured.

Results: We enrolled 70 SSc patients and 26 HC matched by gender and vital parameters. Mean basal resistin levels were increased in SSc patients compared to HC (6.5±5.48 vs 2.56±0.95, p=0.0004). In SSc group, resistin was higher among patients with active DUs (p=0.0007), infected DUs (p=0.0009) and active pattern at nailfold videocapillaroscopy (p=0.01). During one-year follow-up, 27 (38%) SSc patients presented new skin ulcers. Baseline resistin was increased in patients who developed new DUs (8.4±6.4 vs 5.4±4.5, p=0.026). In multiple logistic regression, the development of new DUs was associated to basal serum resistin concentration (OR 2.1, 95% CI 1.1–3.9), to the presence of active DUs at baseline (OR 3.4, 95% CI 1.0–11.9), and to basal Disease Activity Score (DAI) according to European Scleroderma Study Groups (OR 1.3, 95% CI 1.0–1.6). In a proportional Cox regression, the time to new DUs was associated to basal resistin concentration (HR 1.7, 95% CI 1.1–2.8) and DAI (HR 1.2, 95% CI 1.0–1.4).

Conclusions: Serum resistin seems to be associated to the presence and to the development of DUs, suggesting a possible involvement in micro-vascular dysfunction in patients affected by SSc.

Disclosure of Interest: None declared

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