Adipokines are bioactive substances secreted by adipose tissue and also by different resident cells. Adipokines are known to have influence on metabolism but there is increasing evidence of the immunomodulatory role of different adipokines including adiponectin, visfatin/PBEF and resistin. For example, these adipokines are strongly expressed in synovial tissue of RA patients compared to controls. Since little is known about the role of adipokines in other rheumatic diseases including SSc, we analysed the expression of the adipokines adiponectin, resistin and visfatin in SSc organ involvement, specifically in gastric and lung tissue of SSc and control patients.

**Methods**

Gastric samples from SSc (n = 9) and gastritis (n = 12) patients were examined for the expression of adiponectin, visfatin and resistin and also for the immune cell markers CD4, CD8 and CD68 by histology and immunohistochemistry. First, sections from each tissue were examined by hematoxylin/eosin (HE). Thereafter, lung samples from SSc (n = 2), IPF (idiopathic pulmonary fibrosis, n = 8), and healthy controls (n = 8) were examined by immunohistochemistry for the expression of adiponectin, visfatin, resistin and surfactant-protein C (SPC). Immune cell and adipokine expression was analysed qualitatively in gastric samples and semi-quantitatively in lung samples.

**Results**

The numbers of CD4\(^+\) and CD8\(^+\) T-cells in the gastric wall were comparable in SSc (22.1 ± 7.3 resp. 15.8 ± 2.2) and gastritis (23.8 ± 3.3 resp. 15.3 ± 4.0) patients. SSc patients showed a higher number of CD68\(^+\) immune cells compared to gastritis patients (15.3 ± 2.2 versus 6.8 ± 1.9), reaching statistical significance (p = 0.036). Visfatin and resistin expression in the gastric wall was present in most patients from both groups without significant difference. Gastric adiponectin expression was decreased significantly in SSc compared to gastritis patients (p = 0.049) being only present in SSc microvasculature if at all. Adiponectin expression was positively correlated to the number of CD4\(^+\) T-cells and inversely correlated to the number of CD68\(^+\) immune cells. Lung samples of healthy and fibrotic (SSc and IPF) tissue showed a intermediate or strong visfatin and resistin expression. The pattern of resistin and visfatin expression was similar in all tissues, being cell-associated and present among bronchial epithel and immune cells, especially within lymphoid aggregates. Adiponectin was expressed in vessels and lung parenchyma, but not by immune cells. Again, adiponectin was decreased significantly within fibrotic lung tissue from SSc and IPF patients compared to healthy controls (p < 0.0001).

**Conclusions**

T-cell involvement in gastritis appears to be linked to inflammation including SSc even prior to onset of fibrosis, potentially enhanced by local macrophages and supported by resistin and visfatin. In contrast, the strong decrease of adiponectin in SSc gastric tissue and SSc and IPF lung tissue supports the idea of a role in fibrosis. Taken together, adipokines appear to be involved in distinct mechanisms of SSc and IPF pathophysiologically.

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