Background 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 hematoxilin/eosin (HE). Thereafter, FACS analysis of lymph node cells revealed similar amounts of T-cells, B-cells, dendritic cells and macrophages. In contrast, serum analyses revealed a trend toward increased IL-6 levels suggesting that CA exacerbated IL-6 levels driving disease onset and severity in CIA model.

Conclusions With regard to GDF15, a marker of lung involvement in systemic sclerosis, is involved in altered cytokine secretion by fibroblasts, but does not impair fibrosis development.

Results The numbers of CD4+ and CD8+ T-cells in the gastric wall were comparable in SSc (22.1 ± 7.3 resp. 15.3 ± 2.2) and gastritis (23.8 ± 3.3 resp. 15.3 ± 4.0) patients. SSc patients showed a higher infiltration of CD68+ macrophages than gastritis patients (13.3 ± 2.2 versus 6.8 ± 1.9), reaching statistical significance (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 pathophysiology.

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