ASSOCIATION OF CHANGES OF BODY COMPOSITION IN SCLERODERMA PATIENTS WITH DISEASE ACTIVITY, PHYSICAL ACTIVITY AND SERUM LEVELS OF INFLAMMATORY CYTOKINES

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Introduction Fibrosis of the skin and visceral organs, especially digestive tract, and musculoskeletal involvement in systemic sclerosis (SSc) can have a negative impact on body composition and physical activity.

Objectives The aim was to assess body composition and physical activity of SSc patients and healthy controls (HC) and the association with selected inflammatory cytokines in SSc.

Methods 59 patients with SSc (50 females; mean age 52.5; disease duration 6.7 years; lcSSc:34/dcSSc:25) and 59 age-/sex-matched HC (50 females, mean age 52.5) without rheumatic or tumour diseases were included. SSc patients fulfilled ACR/EULAR 2013 criteria. We assessed body composition (densitometry: iDXA Lunar, bioelectric impedance: BIA-2000-M), physical activity (Human Activity Profile, HAP questionnaire), disease activity (ESSG activity index) and serum levels of 27 cytokines (commercial multiplex ELISA kit, Bio-Rad Laboratories). Data are presented as mean ± SD.

Results Compared to HC, patients with SSc had significantly lower body mass index (BMI), body fat% (BF%) and visceral fat weight (VF), and also significantly decreased lean body mass (LBM), and bone mineral density (BMD). Compared to HC, patients with SSc had increased extracellular mass/body cell mass (ECM/BCM) ratio, reflecting deteriorated nutritional status and worse muscle predispositions for physical activity. Increased ECM/BCM in SSc positively correlated with disease activity (ESSG), skin score (mRSS) and inflammation (CRE, ESR), and was associated with worse quality of life (HAQ, SHAQ), fatigue (FSS), and decreased physical activity (HAP). ESSG negatively correlated with BF%, HAP positively correlated with BMD. Increased serum levels of several inflammatory cytokines were associated with alterations of body composition.

Conclusions Compared to healthy age-/sex-matched individuals we found significant negative changes in body composition of our SSc patients, which are associated with the disease activity and physical activity, and could reflect their nutritional status, and gastrointestinal and musculoskeletal involvement. Serum levels of certain inflammatory cytokines were associated with alterations of body composition in SSc patients.
REFERENCES

Disclosure of Interest None declared.

EFFECT OF MACROPHAGE MIGRATION INHIBITORY FACTOR ON HUMAN MACROPHAGES FROM ARTHRITIS PATIENTS

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Career situation of first and presenting author Post-doctoral fellow.

Introduction Macrophage migration inhibitory factor (MIF) is a key regulator of pro-inflammatory cytokines and has been implicated in angiogenesis and pathogenesis of several diseases such as rheumatoid arthritis (RA). Macrophages are considered to be one of the key players in the hyperplastic synovial tissue that invades and degrades adjacent cartilage and bone in patients with inflammatory arthritis.

Objectives In this study a comparative analysis was performed to examine the expression of MIF, and the effect it has on the macrophage polarisation and on the angiogenic and inflammatory mechanisms of macrophages in patients with RA, Psoriatic Arthritis (PsA), Osteoarthritis (OA) and in Arthralgia patients.

Methods PBMCs (Peripheral Blood Mononuclear Cells) were isolated from healthy donors, and patients with OA, RA, PsA and Arthralgia. Primary macrophages (Mfs) were subsequently differentiated and polarised from circulating CD14+ monocytes into M1 and M2 phenotypes. The levels of MIF expression in PBMC, Mf and Synovial tissue was evaluated by real-time-PCR (RT-PCR) and Immunohistochemistry (IHC). The effect of MIF on polarisation of Mfs was investigated by Flow Cytometry. Polarised Mf supernatants were harvested and assayed for soluble MIF by ELISA. The effect of MIF on angiogenic and inflammatory markers (MCP-1, IL-6, IL-8, Ang-2, VEGF, Hif1a, PDGF-B, bFGF, RANTES and ICAM-1) of polarised Mfs was investigated by Real-PCR, Western blot and ELISA.

Results MIF expression was significantly increased in RA tissue compared to OA and PsA. In contrast MIF expression in RA PBMCs was significantly decreased when compared to HC, Arthralgia, and PsA. RA tissue biopsies demonstrated significantly higher MIF expression when compared to PsA, OA, and Arthralgia. In polarised macrophages MIF expression was found to be increased in RA and PsA compared to healthy controls. Addition of rMIF activated pro-inflammatory and angiogenic responses in unpolarised and polarised HC Mfs with increases in gene expression levels of IL-1β, IL-6, MCP-1, ICAM-1, Hif1α, VEGF and Hif1α. Soluble IL-6 expression was also elevated in M0 macrophages.

Conclusions MIF may have a key role in promoting pathogenesis of RA and has a good potential as a therapeutic for RA.

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