REFERENCES

None.

Acknowledgements Supported by AZV NV18–01-00161A, MHCR 023728 and GAUK 312218

Disclosure of Interest None declared.

P167 ASSOCIATION OF CHANGES OF BODY COMPOSITION IN SCLERODERMA PATIENTS WITH DISEASE ACTIVITY, PHYSICAL ACTIVITY AND SERUM LEVELS OF INFLAMMATORY CYTOKINES

[1,2]S Oreska*, [3]M Spiritovic, [3]P Cesak, [3]M Cesak, [1,2]H Storkanova, [1]H Smucrova, [3]B Hermankova, [1,2]O Ruzickova, [1,2]H Mann, [1,2]K Pavelka, [1,2]L Senolt, [1,2]J Vencovsky, [1,2]R Becvar, [1,2]M Tomcik. [1]*Department of Rheumatology, Institute of Rheumatology*; [2]*1st Faculty of Medicine, Charles University*; [3]*Department of Physiotherapy, Faculty of Physical Education and Sport, Charles University, Prague, Czech Republic*

10.1136/annrheumdis-2018-EWRR2019.149

Career situation of first and presenting author Student for a master or a PhD.

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 (CRP, 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

none

Acknowledgements Supported byAZV NV18–01-00161A, MHCR 023728 and GAUK 312218.

Disclosure of Interest None declared.

P168 PREVALENCE AND CHARACTERISTICS OF CARDIOVASCULAR RISK FACTORS AND CORONARY DISEASE AMONG PATIENTS WITH PSORIATIC ARTHRITIS

N Plaza Aulestia*, SA Rodriguez Montero, MJ Perez Quintana, ML Velloso Feijóo. *Rheumatology, Valme Hospital, Sevilla, Spain*

10.1136/annrheumdis-2018-EWRR2019.150

Career situation of first and presenting author Resident.

Introduction Patients with psoriatic arthritis have increased cardiovascular disease risk and comorbidity compared with the general population.

Objectives To describe prevalence and characteristics of coronary disease and cardiovascular risk factor in a cohort of patients with psoriatic arthritis over 40 years old, in each group of treatment (DMARDS vs biologic therapy).

Methods Patients older than 40 years old with psoriatic arthritis were identified based on medical records of Rheumatology department database. Primary outcomes included: age, sex, disease duration and age at the coronary event, HLAB27 positivity, hypertension, type II diabetes and hyperlipidemia.

Results Of the 137 patients, 57% were men and mean age was 57.05±10.6 years old. 82% had only peripheral arthritis, while 18% also showed axial involvement. Regarding the latter subgroup, 16% of patients had a positive HLA-B27 test, 56% were HLA-B27 negative and 28% showed lack of the test. 87% of patients were in DMARDs therapy, while 31% received biologic therapy: etanercept 42%, secukinumab 16%, adalimumab 12%, ustekinumab 12%, infliximab 9,5%, golimumab 4,7% and certolizumab 2%. About 7% of patients didn´t receive DMARDS neither biologic therapy, because of intolerance. Prevalence for DMARD group and biologic group for hypertension was 43% and 26%, respectively; for diabetes 19,5% and 7%; hyperlipemia 47,5% and 38%; coronary disease 10,9% and 2,4%. In DMARD subgroup, we found 6 myocardial infarction (all of them revascularized) and 3 angina, versus 1 myocardial infarction in biologic subgroup

Conclusions There is solid epidemiologic evidence linking PsA to cardiovascular risk factors and an increased risk of developing cardiovascular disease. In addition, over the past two decades it has become clear that chronic inflammation is an independent risk factor for cardiovascular events. In our study the ratio of ischemic heart disease for patients with PsA in DMARD therapy is four times higher than biologic treatment group. This may be due to the greater percentage of cardiovascular risk factors in the first group, although, the cardioprotective effect of biologic therapies, must be considered, as there are some studies that show association between antiTNF and significant reduction in carotid IMT. Proper management of cardiovascular risk requires aggressive control of disease activity.

REFERENCES

1. *Int J Mol Sci* 2018;**19**(1):58.
2. *Int J Rheumatol* **2012**;2012:714321.

Disclosure of Interest None declared.

P169 EFFECT OF MACROPHAGE MIGRATION INHIBITORY FACTOR ON HUMAN MACROPHAGES FROM ARTHRITIS PATIENTS

[1]T Rakovich*, [1]C Cunningham, [1]S Ansboro, [2]T McGarry, [2]DJ Veale, [1]U Fearon. *[1]Molecular Rheumatology, Trinity College Dublin; [2]ERC, St. Vincent's University Hospital, UCD, Dublin, Ireland*

10.1136/annrheumdis-2018-EWRR2019.151

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 examining M1 and M2 Mf specific markers by RT-PCR and 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 rhMIF 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, Hey-1, VEGF and Hif1a. 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.

Acknowledgements Author would like to thank Arthritis Ireland for funding this project.

Disclosure of Interest None declared.

P170 POTENTIAL ROLE FOR THE CHEMOKINE CCL22 IN THE DEVELOPMENT AND EARLY PROGRESSION OF RHEUMATOID ARTHRITIS

[1]B Réthi*, [1]A Krishnamurthy, [1]A Circiumaru, [1]K Sakurabas, [1]V Joshua, [1]M Sun, [1]C Szu-Ying, [1]M Engström, [1]H Wähämaa, [1]A Hensvold, [2]JE Smith, [3]AI Catrina. *[1]Department of Medicine, Center for Molecular medicine, Karolinska Institutet and Karolinska University Hospital, Stockholm, Sweden; [2]Immunoinflammation TA, GSK Medicines Research Centre, Stevenage, UK; [3]Division of Rheumatology, Department of Medicine, Karolinska Institutet and Karolinska University Hospital, Stockholm, Sweden*

10.1136/annrheumdis-2018-EWRR2019.152

Career situation of first and presenting author Young investigator.

Introduction CCL17 and CCL22 are chemokines that bind to the receptor CCR4, which is expressed on various immune cells as well as on neurons. CCL17 has been shown to mediate the pro-inflammatory and algesic actions of GM-CSF in murine arthritis models[1] and CCL22 could activate nociceptive neurons in cell culture[2] or induce hyperthermia by acting on the hypothalamus.[3] Our previous studies have indicated that chemokine production by osteoclasts (OCs) might contribute to bone damage and arthralgia in the presence of anti-citrullinated protein antibodies.[4 5]

Objectives We analyzed the expression and potential roles of CCL17 and CCL22 during rheumatoid arthritis (RA).

Methods We compared CCL17 and CCL22 levels in the sera of individuals at risk of developing RA, in patients at early stages of RA and in healthy controls. We also studied the production of these molecules in OC cultures. We analyzed whether CCL22 can induce arthralgia or affect OC development.

Results Serum levels of CCL22 were elevated in individuals at risk of developing RA and in early untreated RA, when compared to healthy controls. On the contrary, CCL17 levels showed no significant difference between the studied cohorts. In the group of RA patients, higher CCL22 concentrations were associated with smoking. In OC cultures CCL22 production was triggered by M-CSF or GM-CSF and CCL22 levels correlated with both of these cytokines in the synovial fluid of RA patients. CCL22 induced arthralgia when injected into the ankle joints of mice and it stimulated OC differentiation in cell culture.

Conclusions The early increase of CCL22 might contribute to the development of RA, potentially by inducing pain and osteoclastogenesis. Inflammatory cytokines, like GM-CSF and M-CSF, and also by environmental triggers such as smoking can contribute to the increase of CCL22.

REFERENCES

1. Achuthan A, *et al*. *J Clin Invest* 2016.
2. Oh SB, *et al*. *J Neurosci* 2001.
3. Osborn O, *et al. Cytokine* 2011.
4. Krishnamurthy A, *et al. Ann Rheum Dis* 2016.
5. Wigerblad G, *et al. Ann Rheum Dis* 2016.

Disclosure of Interest B. Réthi: None declared, A. Krishnamurthy: None declared, A. Circiumaru: None declared, K. Sakurabas: None declared, V. Joshua: None declared, M. Sun: None declared, C. Szu-Ying: None declared, M. Engström: None declared, H. Wähämaa: None declared, A. Hensvold: None declared, J. Smith Employee of: GSK, A. Catrina: None declared.