depletion of CD11c⁺ cells led to a significant reduction of synovial inflammation and a complete depletion of osteoclasts. Conclusions These data show that in addition to initiating an adaptive immune response, CD11c⁺dendritic cells, are also involved in innate effector mechanisms of inflammatory arthritis. Especially CD11b⁺CD11c⁺ and monocyte derived inflammatory seem to play a role in inflammatory arthritis, suggesting that they could be an important therapeutic target for patients suffering from inflammatory arthritis.

Disclosure of interest None declared

P057

EFFECTS OF ANTI-TNF THERAPY ON VASCULAR BIOMARKER LEVELS IN RHEUMATOID ARTHRITIS

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Introduction Prevoius studies have shown an increased risk of cardiovascular disease in rheumatoid arthritis (RA), due to RA-associated inflammation. Different vascular biomarkers, such as anti-hsp65 antibodies, asymmetric dimethylarginine (ADMA) and B-type natriuretic peptide (BNP) have been associated with atherosclerosis and RA. Anti-hsp65 antibodies were found in RA patients and these antibodies linked also to inflammation and atherosclerosis. ADMA is an endogenous competitive inhibitor of NOS and consequential can lead to increased nitrosative and oxidative stress. ADMA has been implicated with atherosclerosis, and also with rheumatoid arthritis. BNP also associated to cardiovascular diseases.

Objectives The aim of this study was to assess the effects of anti-TNF therapy on different vascular biomarkers, such as anti-hsp65, ADMA and BNP in patients with RA and their correlation with different laboratory and clinical markers.

Methods Altogether 36 RA patients were recruited and treated with either etanercept (ETN) or certolizumab pegol (CZP) in this 12 months follow-up study. Assessments were performed at baseline, at month 6 and 12. Amounts of IgG antibodies reacting with recombinant M. bovis hsp65 (Lionex, Braunschweig, Germany) were measured by ELISA. ADMA was assesed by HPLC with fluorescent detection. BNP fragments were assessed by commercially avaiable ELISA kit (Biomedica, Vienna). In addition, disease activity (DAS28), age, disease duration, CRP, IgM rheumatoid factor (RF), anti-CCP (aCCP) and plasma lipid levels were also measured. Arterial flow-mediated vasodilation (FMD), carotid intima-media thickness (cIMT) and arterial pulse-wave velocity (PWV) were assessed by ultrasound.

Results There were no significant changes in the levels of anti-hsp60, ADMA and BNP due to anti-TNF therapy. However, baseline level of BNP is correlated with the levels of RF (R=0.479, p=0.004) and aCCP (R=0.591, p<0.001). Serum BNP levels at baseline and at month 6 were significantly increased in RF positive compared to RF negative patients (680.60±381.64 versus 292.94±198.27 pmol/L; p=0.007 and 668.95±346.51 versus 312.20±207.01 pmol/L; p=0.001) and also in aCCP postive compared to aCCP negative patients (670.61±323.04 versus 137.98±436.41 pmol/L; p=0.030 and 652.93±283.21 versus 456.48±423.11 pmol/L; p=0.021).

Furthermore we found the following correlations between baseline values: anti-hsp60 level correlated with ADMA (R=0.900, p=0.037), triglyceride (TG) (R=0.462,p=0.040) and PWV (R=0.564, p=0.040). Baseline level of ADMA positively correlated with body mass index (BMI) score (R=0.720, p=0.040) and also with HDL levels (high density lipoprotein) of patients (R=0.473, p=0.047).

Conclusions BNP levels were significantly higher in RF +compared to RF- patients, which imply that BNP may associate with RF positivity. Specific biomarkers, such as ADMA, anti-hsp60 and BNP may play important role cardiovascular disease in RA.

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Disclosure of interest None declared

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S100 PROTEINS EFFECTIVELY DISCRIMINATE SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS FROM HEALTHY CONTROLS, BUT ARE NOT ASSOCIATED WITH MEASURES OF DISEASE ACTIVITY

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Introduction The \$100 proteins are important regulators of diverse calcium-dependent cellular processes including growth regulation, migration and apoptosis. Dysregulated expression of multiple members of \$100 family is a common feature of cancer and several autoimmune diseases.

Objectives The aim of this study was to examine whether circulating levels of \$100A4, \$100A8/9 and \$100A12 proteins could be useful as diagnostic or activity specific markers in systemic lupus erythematosus (SLE).

Methods S100 plasma levels were measured by ELISA in a cohort study of 44 patients with SLE, 8 patients with incomplete SLE (iSLE) and 43 healthy controls (HCs). Disease activity was assessed using SLEDAI-2 K. We examined cross sectional associations between concentrations of S100 proteins and SLE status, SLEDAI-2 K scores, and levels of conventional biomarkers.

Results Plasma levels of all analysed \$100 proteins (\$100A4, S100A8/9 and S100A12) were significantly higher in SLE patients compared to HCs (p<0.001, p<0.01, p<0.001, respectively). In iSLE patients, the levels of S100A4 and S100A12 but not S100A8/9 were significantly higher compared to HCs (p<0.001, p<0.05, p=ns, respectively). ROC curve analysis was performed to establish the optimal threshold to discriminate SLE patients from HCs based on S100 levels. At the optimal cutoff point of 238 ng/ml, the area under curve (AUC) for \$100A4 was 0-990 (95% CI: 0.977 to 1.000, p<0.001) with a sensitivity of 96% and specify of 93.0%. The remaining two proteins also showed significant, but not as strong discriminative value [\$100A8/9: AUC 0.684 (95% CI: 0.572 to 0.795, p<0.05); S100A12: AUC 0.809 (95% CI: 0.719 to 0.898, p<0.001]. We found that only S100A12 levels were significantly associated with the SLEDAI-2K score (r=0.318, p=0.035). Both S100A8/9 and S100A12 levels were significantly higher in SLE patients with arthritis (p=0.043, p=0.015, respectively) and with haematological