Objectives: Osteopontin (OPN) is required for osteoclast recruitment. We hypothesised that AD exacerbates bone erosion by inducing OPN expression in synovial tissue. This study aimed to evaluate a novel role for AD in RA.

Methods: The serum levels of AD and OPN were determined in 38 RA, 40 osteoarthritis (OA) patients, and 20 healthy controls using enzyme-linked immunosorbent assay (ELISA). AD and OPN production were measured by double immunofluorescence of RA and OA synovial tissue. This study aimed to evaluate a novel role for AD in RA.

Results: Our results indicated that the AD and OPN expression levels increased noticeably and were associated with each other in the RA serum. The AD distribution was coincident with that of OPN in the RA synovial tissue. AD stimulation of RASFs increased OPN production in a dose-dependent manner. AD-treated RASFs promoted RAW264.7 cell migration, and the effect was blocked by a specific antibody against OPN. Silencing of OPN using lentiviral-OPN shRNA reduced the number of TRAP-positive osteoclasts and the extent of bone erosion in the AD-treated CIA mice. When bound to integrin αvβ3, OPN functions as a mediator of AD and osteoclasts.

Conclusions: Our study provides new evidence of AD involvement in bone erosion. AD induces the expression of OPN, which recruits osteoclasts and initiates bone erosion. These data highlight AD as a novel target for RA treatment.

Disclosure of Interest: None declared

THU0081
IDENTIFICATION OF NOVEL AUTOANTIBODIES IN THE SYNOVIAL FLUID FROM PATIENTS WITH RHEUMATOID ARTHRITIS

K.I. Goto1, T. Kawamoto2, A. Nakajima3, M. Tahara4, T. Ebata1. 1Orthopaedic Surgery, Sakura Orthopaedic Hospital, Sakura, 2Orthopaedic Surgery, Matsudo City Hospital, Matsudo; 3Orthopaedic Surgery, Toho Univ. Sakura Hospital, Sakura; 4Orthopaedic Surgery, Chiba-East National Hospital, Chiba, Japan

Background: Rheumatoid arthritis (RA) is a chronic, autoimmune and inflammatory joint disease with a poorly understood etiology. Despite widespread diagnostic use of anti-citrullinated protein antibodies and rheumatoid factor, there is strong demand for novel biomarkers to improve the diagnosis of this disease.

Objectives: The purpose of present study is to investigate novel autoantibodies in the synovial fluid of RA patients.

Methods: 1 By using SEREX (Sero logical identification of antigens by recombinant cDNA expression cloning), we identified ten and several antigens from sera of RA patients. 2) Three epitope sites in the candidate antigens proteins were predicted and 18 mer peptides were synthesised. 3) Synovial fluid of the knees was obtained from 48 RA and 48 osteoarthritis (OA) patients. 4) Furthermore, Alpha-LISA was used to analyse the antibody levels in synovial fluid using synthetic poly-peptide as antigen.

Results: Significantly higher proportion of antibodies against lamin A (LMNA, RA 1987±13924 VS OA 672±3975, p<0.0000001) and cell growth-regulating nucleolar protein (CGRN, RA 1963±1331 VS OA 1616±6391, p<0.000007) were found in synovial fluid of RA as compared with OA.

Conclusions: We identified two novel autoantibodies in the knee synovial fluid of RA patients. These antibodies would have the potential to become diagnostic biomarkers of RA.

REFERENCES:

Disclosure of Interest: None declared

THU0082
IMPAIRED LEFT VENTRICULAR RELAXATION AND ASSOCIATION WITH INFLAMMATORY MARKERS IN COLLAGEN-INDUCED ARTHRITIS

L. Mukeda; 1 F.S. Michel; 1 C. Mogane; 1 P.H. Desein; 2 A.M. Millen; 1 1Physiology, University of the Witwatersrand, Parktown, Johannesburg, South Africa; 2Rheumatology Division, Vrije Universiteit Brussel, Brussels, Belgium

Background: Patients with rheumatoid arthritis (RA) experience an increased risk of developing heart failure with a preserved ejection fraction. Although there is some evidence to support a role of chronic inflammation in the pathogenesis of impaired left ventricular (LV) function in RA, the direct effects of inflammatory cytokines on the LV function in collagen-induced arthritis (CIA) (an experimental model most similar to RA) require further elucidation.

RESULTS:

<table>
<thead>
<tr>
<th>n</th>
<th>Irisin (mean ±SD)</th>
<th>Myostatin (mean ±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA patients treated with biologics</td>
<td>13</td>
<td>31,7±1,7</td>
</tr>
<tr>
<td>RA patients non-treated with biologics</td>
<td>27</td>
<td>25,9±1,6</td>
</tr>
<tr>
<td>Controls</td>
<td>30</td>
<td>30,3±10,95</td>
</tr>
</tbody>
</table>

Results: RA patients had decreased serum levels of irisin (25,61±2,25 vs 30,36±10,95 ng/ml; p<0,05) and myostatin (3011,28±1271,11 vs 4049,08±1610,01 pg/ml; p<0,05), decreased ALMI (6,09±0,88 vs 6,50±1,10; p<0,05) and increased FMI (11,26±3,30 vs 9,44±2,65; p<0,05), compared to controls. No correlations were observed among irisin and myostatin levels and ALMI and FMI. Of the 122 RA patients, 40 were analysed for the use of biologic medication. Serum levels of irisin and myostatin were different between RA patients treated and non-treated with biologics (table 1).

Conclusions: RA patients presented loss of lean mass and gain of fat mass, as well as lower irisin and myostatin serum levels, in comparison with controls. Additionally, the use of biologic medication by patients impacted on myokines serum levels. Further analyses are needed for a better comprehension of irisin and myostatin roles in RA, and to verify their correlation to other RA features.

REFERENCES:

Disclosure of Interest: None declared

THU0082
IMPAIRED LEFT VENTRICULAR RELAXATION AND ASSOCIATION WITH INFLAMMATORY MARKERS IN COLLAGEN-INDUCED ARTHRITIS

L. Mukeda1, F.S. Michel1, C. Mogane1, P.H. Desein2, A.M. Millen1 1Physiology, University of the Witwatersrand, Parktown, Johannesburg, South Africa; 2Rheumatology Division, Vrije Universiteit Brussel, Brussels, Belgium

Background: Patients with rheumatoid arthritis (RA) experience an increased risk of developing heart failure with a preserved ejection fraction. Although there is some evidence to support a role of chronic inflammation in the pathogenesis of impaired left ventricular (LV) function in RA, the direct effects of inflammatory cytokines on the LV function in collagen-induced arthritis (CIA) (an experimental model most similar to RA) require further elucidation.

RESULTS:

<table>
<thead>
<tr>
<th>n</th>
<th>Irisin (mean ±SD)</th>
<th>Myostatin (mean ±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA patients treated with biologics</td>
<td>13</td>
<td>31,7±1,7</td>
</tr>
<tr>
<td>RA patients non-treated with biologics</td>
<td>27</td>
<td>25,9±1,6</td>
</tr>
<tr>
<td>Controls</td>
<td>30</td>
<td>30,3±10,95</td>
</tr>
</tbody>
</table>

Results: RA patients had decreased serum levels of irisin (25,61±2,25 vs 30,36±10,95 ng/ml; p<0,05) and myostatin (3011,28±1271,11 vs 4049,08±1610,01 pg/ml; p<0,05), decreased ALMI (6,09±0,88 vs 6,50±1,10; p<0,05) and increased FMI (11,26±3,30 vs 9,44±2,65; p<0,05), compared to controls. No correlations were observed among irisin and myostatin levels and ALMI and FMI. Of the 122 RA patients, 40 were analysed for the use of biologic medication. Serum levels of irisin and myostatin were different between RA patients treated and non-treated with biologics (table 1).

Conclusions: RA patients presented loss of lean mass and gain of fat mass, as well as lower irisin and myostatin serum levels, in comparison with controls. Additionally, the use of biologic medication by patients impacted on myokines serum levels. Further analyses are needed for a better comprehension of irisin and myostatin roles in RA, and to verify their correlation to other RA features.

REFERENCES:

Disclosure of Interest: None declared