response relationship was found for phase 2, phase 3 and PVM, but not in phase 1 (table 1). Few joints showed enhancement in phase 1 and a clear dose-response relationship was found for pain during the last 24 hours only (data not shown).

Table 1: Associations between FOI Enhancement and Pain in the Same DIP and PIP Joints.

<table>
<thead>
<tr>
<th>FOI Phase</th>
<th>Self-reported pain last 24 hours</th>
<th>Self-reported pain last 6 weeks</th>
<th>Tender joint on clinical exam</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
</tr>
<tr>
<td>Grade 1</td>
<td>1.50 (0.94, 2.40)</td>
<td>1.92 (1.20, 3.09)</td>
<td>2.42 (1.37, 4.25)</td>
</tr>
<tr>
<td>Grade 2</td>
<td>1.26 (1.02, 1.55)</td>
<td>1.15 (0.93, 1.44)</td>
<td>2.15 (1.81, 2.55)</td>
</tr>
<tr>
<td>Grade 3</td>
<td>1.94 (1.58, 2.38)</td>
<td>1.92 (1.52, 2.69)</td>
<td>2.21 (2.21, 3.26)</td>
</tr>
<tr>
<td>Grade 4</td>
<td>2.25 (1.61, 3.15)</td>
<td>2.53 (1.73, 3.69)</td>
<td>4.00 (2.85, 5.59)</td>
</tr>
</tbody>
</table>

Conclusions: In this first hand OA study, FOI enhancement was frequently found in the DIP and PIP joints, whereas the method seems insensitive to detect inflammation in the CMC-1 joints. FOI enhancement was related to self-reported pain and to tender joints on clinical examination, supporting the validity of the FOI examination in patients with hand OA.

Disclosure of Interest: None declared


PREVALENCE OF ANTI-ACETYLATED PROTEIN ANTIBODIES IN INFLAMMATORY ARTHRITIS, OSTEOARTHRITIS, CONNECTIVE TISSUE DISEASES AND ITS DISCRIMINATIVE CAPACITY AS DIAGNOSTIC MARKER FOR EARLY RHEUMATOID ARTHRITIS

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Background: Numerous post-translationally modified proteins have been described as auto-antigens in rheumatoid arthritis (RA) patients. Antibodies (abs) against acetylated (ac) peptides (AAPA) have recently been reported in RA patients, but not yet been evaluated in other inflammatory and non-inflammatory rheumatologic conditions; therefore their specificity (spec) and sensitivity (sens) remains unclear.

Objectives: To determine the prevalence of AAPA in RA, healthy subjects and other rheumatic diseases in order to evaluate their diagnostic potential for discriminating RA, healthy and other rheumatic diseases.

Methods: We obtained serum samples of patients with early untreated RA, established RA (>3 years), osteoarthritis (OA), systemic lupus erythematosus, granulomatosis with polyangiitis (GPA), polymyositis, axial spondyloarthritis, primary Sjögren’s syndrome and healthy subjects. AAPA were measured by ELISA using peptides derived from mutated vimentin (acyetilation of lysine or ornithine in position 7 or 2 (inverse peptide), as antigen. Receiver operating characteristics and logistic regression analyses were used to assess the discriminative capacity of AAPA.

Results: Areas under the curves (AUC) were significant in early RA (era; n=120: 76% female, mean disease duration: -0.07±0.51 years, mean symptom duration 1.49±2.01 years) versus healthy subjects for IgG-als against ac lysine, inverse lysine and ornithine (AUC of 0.666, 0.687, 0.800, respectively). We chose a cutoff of 20 U/ml putting an emphasise on high spec, with balanced sens (ac-lysine: spec: 97.0%, sens: 32.5%, LR: 10.7, CI: 3.4–33.7; ac-ornithine: spec: 80.7%, sens: 42%, LR: 2.2, CI: 1.3–3.6; ac-ornithine: spec: 93.9%; sens: 39.2%, LR: 6.5, CI: 2.9–14.5). Analyses of positivity for multiple ab-reactivity revealed increasing LR by number of abs, with 100% specificity when all 3 AAPAs are detected (table 1). Testing this cutoff against OA patients showed similar specificities, but with lower LR (2 AAPA: LR 3.48, CI: 1.9–6.6). Sens is increased when testing established RA versus healthy controls, with ac-ornithine performing best (ac-lysine: 49.2%, CI: 42.0–56.5; ac-inv-lysine: 35.2%, CI: 28.5–42.4; ac-ornithine: 53.9%, CI: 46.6–61.0). We found that practically only RA patients showed three different AAPA reactivities in early RA patients (n=120) with 2 AAPA reactivities with even 97% respectively. Of one AAPA identified RA patients vs. healthy subjects with a spec of 77.7% and sens: 39.2%; LR 6.5, CI: 2.9–14.5.

Graph 1A: Prevalence (in%) of IgG antibodies against the 3 different acetylated peptides using 20 U/ml as cutoff

Graph 1B: Venn diagram, outlining the distribution and overlap of AAPA (blue circle), ACPA (striped circle) and RF (rose circle) in early rheumatoid arthritis patients (n=120).

Conclusions: AAPA are highly prevalent autoantibodies in early RA, closing a further gap of seronegativity, with only 24.6% of early RA patients remaining negative for RF, ACPA or AAPA. In particular, multiple reactivity to AAPA increased the specificity for era, also adding diagnostic value beyond RF and ACPA.

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LOW BACK PAIN DUE TO ENTHESOPATHY OF ERCTOR SPINE MUSCLE: A COMPARATIVE US AND MRI STUDY IN PATIENTS WITH IliAC CREST PAIN SYNDROME

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Background: Iliac crest pain syndrome (ICPS) is a regional pain syndrome, that is particularly common in patients with low back pain (LBP). It is characterised clinically by pain perceived maximally at the most medial part of the posterior iliac crest, and patients’ recognition of the pain provoked by a systematic digital palpation in this area, as ‘their’ typical one. Though ICPS is very frequently encountered in LBP, the exact etiology of this syndrome was not established. Based on anatomical data, it was suggested that ICPS could be caused by a tendinopathy/enthesisopathy of erector spine (ES) muscle attachments to the medial iliac crest (MIC).1 In a previous anatomical and ultrasound (US) study we showed that this in fact might be the case.

Objectives: The purpose of this study was, regarding the existing clinical diagnostic criteria for the ICPS as a “gold standard”, to establish the sensitivity, specificity, positive and negative predictive values (PPV and NPV) and the overall accuracy of the US and the Magnetic Resonance Imaging (MRI) assessment of the entheseal of the ES muscle at the MIC in patients with LBP and features indicative of ICPS.

Methods: 25 patients (9 men, 16 women, mean age 43.12±11.83. mean BMI 25.07±2.36) with anamnesis of chronic “nonspecific” (after lateral X-ray and standard clinical examination) LBP perceived maximally in the region of the MIC uni- or bilaterally were included. First a systematic palpation of the posterior MIC bilaterally was performed to diagnose ICPS clinically. Then in two successive days each patient underwent MRI examination of the lower back (lumbar and sacroiliac regions), with an enlarged field of view in the sagittal plane, and a standardized US examination of the ES terminal tendons and entheses bilaterally. The MRI were assessed by a radiologist with 10 years of experience in MRI. The sonographic parameters were performed by a rheumatologist with 7 years of experience in US and analysed in regards with the OMERACT definition of entheseopathy. As a single lesion at any enthesis is a common finding, the presence of at least two pathological features were required to classify given entheses as abnormal.

Conclusions: This study shows that entheseopathy of the ES muscles could be the unrecognised cause for most of the cases of ICPS—a regional syndrome particularly common in LBP. US performed better than the MRI in diagnosing this pathological condition, that may reflect the fact that radiologists are not used to assess these structures. The good diagnostic properties of US in ICPS could be of value when assessing patients with otherwise “nonspecific” LBP.


Disclosure of Interest: None declared