Table 1 Mean VAS Scores and % of clinical management decision attributed to inflammation, damage, and distress in patients with rheumatic diseases

<table>
<thead>
<tr>
<th>Mean VAS Scores</th>
<th>ALL N=570</th>
<th>RA N=98</th>
<th>OA N=1311</th>
<th>FM N=89</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAS DOCGL</td>
<td>4.4 (1.6)</td>
<td>4.6 (1.8)</td>
<td>4.4 (1.5)</td>
<td>5.2</td>
<td>0.04</td>
</tr>
<tr>
<td>VAS DOCINF</td>
<td>1.8 (2.0)</td>
<td>2.8 (2.4)</td>
<td>0.7 (1.1)</td>
<td>0.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VAS DOCDAM</td>
<td>3.1 (2.2)</td>
<td>3.8 (2.3)</td>
<td>4.4 (1.8)</td>
<td>1.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VAS DOCSTR</td>
<td>2.1 (2.9)</td>
<td>1.2 (2.2)</td>
<td>1.5 (2.5)</td>
<td>6.0</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

% of clinical management decision attributed to:

- % inflammation: 29 (31) vs 39 (29) vs 12 (19) vs 6 (11) <0.001
- % damage: 48 (35) vs 52 (30) vs 73 (31) vs 18 (23) <0.001
- % distress: 22 (34) vs 9 (20) vs 15 (27) vs 76 (27) <0.001

Table 1 Characteristics of the PsA cohort (n=96)

<table>
<thead>
<tr>
<th>Gender, % males</th>
<th>47%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>51.5±13</td>
</tr>
<tr>
<td>Psoriasis duration</td>
<td>17±13</td>
</tr>
<tr>
<td>Psoriasis activity: remission/mild/moderate to severe</td>
<td>34%/56%/10%</td>
</tr>
<tr>
<td>PsA duration</td>
<td>9.4±1.1</td>
</tr>
<tr>
<td>PsA activity: remission/low/moderate/severe</td>
<td>37%/33%/22%/6%</td>
</tr>
<tr>
<td>Concomitant sDMARDs treatment (%)</td>
<td>49%</td>
</tr>
<tr>
<td>Concomitant biologic treatment (%)</td>
<td>65%</td>
</tr>
<tr>
<td>Biologic treatment exposure (past or present) (%)</td>
<td>71.8%</td>
</tr>
</tbody>
</table>

Conclusions: This is the first study to report a detectable hs-cTnT level in up to 30% in patients with well controlled PsA, asymptomatic for CVD, warranting a special attention to monitoring CVD risk factors and manifestations in this group. Traditional CVD risk factors but not measures of disease activity were associated with detectable hs-cTnT. The latter may be explained by a potential positive impact of anti-rheumatic therapies on the cardiovascular profile. Further prospective studies addressing the predictive role of hs-cTnT for CVD events in PsA are needed.

REFERENCES:

Disclosure of Interest: None declared

FR0680 TETHERIN, A NOVEL TYPE I INTERFERON BIOMARKER ON BLOOD LEUCOCYTES CAN CAPTURE INTERFERON STATUS AND CORRELATES WITH USTEKINUMAB (STEELARA) THERAPY RESPONSE IN PSORIATIC DISEASE.

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Background: Recently Ustekinumab, an IL-12/23 p40 monoclonal antibody that is licensed for Psoriatic Arthritis and Psoriasis, showed promising results in phase II trials in SLE- a prototype IFN mediated disease. We previously confirmed a novel IFN gene scoring system associated with the different SLE symptoms (manuscript in review). Additionally, we also recently validated in two independent cohorts the value of BST2/tetherin as IFN-I biomarker assay correlates with clinical activity and predicts clinical flare in SLE. Given that psoriasis has several SNPs in the IFN pathway; Thus mechanistic studies of the effect of Ustekinumab on the IFN pathway can be explored in this disease setting.

Objectives: This work tested the hypothesis that a novel interferon type I (IFN-I) status markers in the blood and skin of Ustekinumab treated psoriasis patients might correlate with therapy responses and provide insights into how p40 blockers may affect IFN pathways in a relevant human disease model.

Methods: Skin biopsies and peripheral blood at baseline (before therapy, 24weeks, 54 weeks) from 23 Ustekinumab patients with psoriasis who had ultrasound imaging confirmed subclinical enthesopathy were recruited. Cellular immunophenotyping was performed using multi-parameter flow cytometry to detect tetherin on (Monocyte, B cells, T cells and neutrophils). All data was compared to age-matched samples from healthy controls (HC). Skin biopsies were digested and RNA extracted, qPCR of common ISGs genes were analysed in lesional and peri-lesional at corresponding time points.

Results: Tetherin showed a higher level of expression on blood subsets of psoriasis compared to HC at baseline on Monocytes, T cells, NK cells and Neutrophils. All data was compared to age-matched samples from healthy controls (HC). Skin biopsies were digested and RNA extracted, qPCR of common ISGs genes were analysed in lesional and peri-lesional at corresponding time points.

Discussion: Tetherin showed a higher level of expression on blood subsets of psoriasis compared to HC at baseline on Monocytes, T cells, NK cells and Neutrophils. All data was compared to age-matched samples from healthy controls (HC). Skin biopsies were digested and RNA extracted, qPCR of common ISGs genes were analysed in lesional and peri-lesional at corresponding time points.

References: None declared

FR0679 HIGH SENSITIVITY CARDIAC TROTONIN T IN PSORIATIC ARTHRITIS PATIENTS: A CROSS-SECTIONAL CONTROLLED STUDY

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Background: High sensitivity cardiac troponin (hs-cTnT) is a biomarker for cardiovascular disease (CVD) in the general population. (1) Psoriatic arthritis (PsA) confers an increased risk for CVD (2) but there are no biological markers to stratify CVD risk in PsA patients.

Objectives: To determine the level of serum hs-cTnT in a PsA cohort and control without inflammatory disease, and further characterize the PsA cohort with detectable hs-cTnT.

Methods: Serum hs-cTnT level was measured with a sandwich immunoassay method in a consecutive PsA cohort (n=96). The control cohort was based on apparently healthy individuals recruited during routine annual health examination, Tel-Aviv Medical Center Inflammatory Survey (TAMCIS) (n=6,052). hs-cTnT was measured in carefully matched controls (n=88), manually selected from the TAMCIS, based on gender, age, BMI, hypertension, and hyperlipidemia prevalence. hs-cTnT > 5 ng/L was used as a cutoff for the detectable level. Multiple regression analysis was used to determine the factors associated with hs-cTnT.

Results: The characteristics of the PsA cohort are presented in table 1. Remarkably, in the majority of patients, both skin and joint disease were well controlled. PsA and TAMCIS cohorts had a similar range of age (51.5 vs 48 yr) but different gender representation: 47% vs 72.5% of males (p<0.001). PsA exhibited a higher prevalence of traditional CVD risk factors compared to the TAMCIS cohort: BMI 28 vs 26.5 (p=0.002), current smokers 20.8% vs 10.1% (p=0.002), hypertension 25% vs 15% (p=0.007), dyslipidemia prevalence 34% vs 27% (p=0.101), diabetes 19.8% vs 4.6% (p=0.001). Due to these differences, a matched control group was used for comparison of troponin. Detectable hs-cTnT was present in 29.5% of the PsA patients compared to 19.3% in the controls. (p=0.114) Factors associated with detectable hs-cTnT in PsA were consistent with traditional factors and included male gender (p=0.007), age (p=0.005), hypertension (p<0.001), and DM (p=0.001). No correlation between detectable hs-cTnT level and psoriasis/PsA duration, disease severity, treatment with DMARDs or biologics was found.

References:

Disclosure of Interest: None declared

References:

Disclosure of Interest: None declared
FOUR COMORBIDITY INDEXES AMONG PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Previous studies have reported an increased risk of multiple comorbidities in people with RA therefore it is necessary to systematically quantify the comorbidity burden of these patients.[1] The comorbidity index is a tool developed under this concept and has multiple clinical and research uses.

Objectives: We compared four comorbidity indexes in patients with rheumatoid arthritis in Taiwan (Charlson Comorbidity Index (CCI), Elixhauser Comorbidity Index (ECI), Mutimorbidity index (MMI), Rheumatic Disease Comorbidity Index (RDCI)).

Methods: All patients with rheumatoid arthritis diagnosed during 1998–2008 in Taiwan were identified using the Taiwan National Health Insurance Database and followed up to 31 Dec 2013. Score accumulation between periods during diagnosis (4 months before and after initial diagnosis) and before/after the diagnostic period was compared. Poisson regression was used to calculate incidence rate ratio.

Results: Among 24767 patients with rheumatoid arthritis, median age at diagnosis is 51 years old and female is 79.2%. The mean score at diagnosis is 0.8 in CCI, 2.8 in ECI, 0.7 in MMI and 1.3 in RDCI. The annual percentage changes are 11.0%, 11.3%, 9.7% and 6.8%, respectively. The score of four comorbidity indexes increased with time after rheumatoid arthritis was diagnosed. The incidence rate ratios (IRR) for occurrence of any disease in comorbidity index before, during, and after diagnosis* in patients with incident rheumatoid arthritis, 2001–2008, Taiwan.

<table>
<thead>
<tr>
<th>Indexes</th>
<th>Before diagnosis</th>
<th>During diagnosis</th>
<th>After diagnosis</th>
<th>IRR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCI</td>
<td>2.301 (1.457)</td>
<td>3.203 (1.673)</td>
<td>2.974 (1.781)</td>
<td>1.37 (1.29 to 1.46)</td>
</tr>
<tr>
<td>ECI</td>
<td>4.999 (1.023)</td>
<td>11.153 (1.664)</td>
<td>10.911 (1.647)</td>
<td>2.44 (2.36 to 2.53)</td>
</tr>
<tr>
<td>MMI</td>
<td>2.249 (1.033)</td>
<td>3.785 (1.065)</td>
<td>2.828 (1.064)</td>
<td>1.26 (1.19 to 1.34)</td>
</tr>
<tr>
<td>RDCI</td>
<td>4.995 (1.020)</td>
<td>13.049 (1.068)</td>
<td>12.738 (1.067)</td>
<td>1.33 (1.29 to 1.44)</td>
</tr>
</tbody>
</table>

Conclusions: Psoriasis which is genetically and mechanistically linked to IFN-I signatures shows responses to Ustekinumab therapy that correlate with improvement in IFN-I signatures in blood and tissues. Given that whole blood ISG signatures were complex and weakly correlating with disease activity. We provide a convenient, validated method to analyse IFN pathway in routine clinical practice using tetherin which could be a future research tool for the cell-specific IFN-I response. These studies support the idea that p40 efficacy in psoriasis is associated with IFN-I pathway modulation and relevant for exploring how p40 blockers may exert potential benefit in SLE.

REFERENCES:

Disclosure of Interest: None declared

FRIDAY, 15 JUNE 2018
Rehabilitation

EXERCISE MAY DECREASE SYCONE SECONDARY TO POSTURAL CHANGE IN FEMALES WITH RHEUMATOID ARTHRITIS: PILOT STUDY

D. C. Janse Van Rensburg1, J. A. Ker2, C. C. Grant1, L. Fletcher3. 1Section Sports Medicine, 2Dept of Internal Medicine, 3Dept of Statistics, University of Pretoria, Pretoria, South Africa

Background: The autonomic nervous system (ANS) regulates the heart rate via sympathetic and parasympathetic influences. Literature has shown that rheumatoid arthritis (RA) patients suffer from autonomic dysfunction. This may consequently lead to syncope with possible falls after posture change i.e. rising from supine to the standing position. Previous research has shown general improvement of the ANS after exercise, but not in specific relation with posture change.

Objectives: To determine the effect of exercise on posture change (supine to standing position) in females with RA as measured by short-term heart rate variability (ANS function).

Methods: Patients with confirmed RA were randomly selected to a control group (RAC) or an exercise group (RAE). The RAC group (n=19) trained two to three times per week under supervision. The RAC group (n=18) continued with their current sedentary lifestyle. The medium intensity exercise programme lasted for

Conclusions: Our study showed that comorbidities continue to accumulate after the initial diagnosis. The comorbidity tends to occur during diagnostic period in patients with rheumatoid arthritis. Different comorbidity indexes are all good at assessing comorbidity burden. Clinicians should screen different comorbidities, determine primary prevention and control disease activity to improve the functional status, quality of life and mortality of rheumatoid arthritis.

REFERENCE:

Disclosure of Interest: None declared
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FRID0682

EXERCISE MAY DECREASE SYCONE SECONDARY TO POSTURAL CHANGE IN FEMALES WITH RHEUMATOID ARTHRITIS: PILOT STUDY

D. C. Janse Van Rensburg1, J. A. Ker2, C. C. Grant1, L. Fletcher3. 1Section Sports Medicine, 2Dept of Internal Medicine, 3Dept of Statistics, University of Pretoria, Pretoria, South Africa

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