High sensitivity cardiac troponin T in Psoriatic Arthritis patients: a cross-sectional controlled study

V. Furer1, S. Shenhar-Tsartfat2, S. Berliner2, U. Arad3, D. Paran1, O. Rogowski1, I. Shapira1,2, O. Elkayam1.

1Dermatology, Tel Aviv Medical Center, Tel Aviv, Israel
2Department of Medicine, Tel Aviv Medical Center, Tel Aviv, Israel
3Dermatology, Tel Aviv Medical Center, Tel Aviv, Israel

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 controls without inflammation, 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, Tamc. (n=88), manually selected from the Tamecis cohort, based on gender, age, BMI, hypertension, and hyperlipidemia prevalence. Hs-cTnT >5ng/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=0.0114) 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.

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-therapeutic therapies on the cardiovascular profile. Further prospective studies addressing the predictive role of hs-cTnT for CVD events in PsA is needed.

References:

Disclosure of Interest: None declared


Tetherin, a novel type I interferon biomarker on blood leukocytes can capture interferon status and correlates with ustekinumab (Stelara) therapy response in Psoriatic Disease

Y. M. El-Shenbini1, L. Savage1, A. Alase1, E. Vitali1, M. Wittmann1, P. Emery1, D. McGonagle1, 2Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, United Kingdom

Background: Recently Ustekinumab, an IL12/23 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, 24 weeks, 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 B cells (p=0.003, <0.005, <0.035, <0.002) compared to the baseline. No significant changes observed between baseline and 2nd visit 24 weeks. Interestingly, a substantial reduction in tetherin expression at 52 weeks in psoriasis was observed in all subsets in Monocytes, T cells, NK cells and B cells (all p=0.0001) correlating with patient response to therapy. IFN signature by TaqMan revealed higher expression in skin biopsies distinctive ISGs compared to HC, e.g. IFI27, STAT1, IFI16 and IRF7 all corrected post-Ustekinumab therapy. Paired correlation with patient response to therapy, IFN signature by TaqMan revealed higher expression in skin biopsies distinctive ISGs compared to HC, e.g. IFI27, STAT1, IFI16 and IRF7 all corrected post-Ustekinumab therapy.
Conclusions: Psoriasis which is genetically and mechanically linked to IFN-I signature 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

FR0681
FOUR COMORBIDITY INDEXES AMONG PATIENTS WITH RHEUMATOID ARTHRITIS
Y. J. Huang1, C. F. Kuo1, J. S. Chen1, S. F. Luo1. 1Division of Rheumatology, Allergy and Immunology, Chang Gung Memorial Hospital and Chang Gung University, Taoyuan, Taiwan, Province of China

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 any increase in comorbidity score during versus before diagnosis period was compared. Poisson regression was used to calculate incidence rate ratio.

Table 1 Incidence rates (IR) per 1000 patient months and incidence rate ratios (IRR)

<table>
<thead>
<tr>
<th>Comorbidity Index</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>1.351 1.081</td>
<td>2.353 1.013</td>
<td>3.064 1.011</td>
<td>1.31 [1.26 to 1.37]</td>
</tr>
<tr>
<td>ECI</td>
<td>0.499 0.263</td>
<td>1.153 0.246</td>
<td>1.017 0.111</td>
<td>1.33 [1.26 to 1.40]</td>
</tr>
<tr>
<td>MMI</td>
<td>2.349 0.039</td>
<td>2.985 0.057</td>
<td>3.028 0.056</td>
<td>1.26 [1.19 to 1.33]</td>
</tr>
<tr>
<td>RDCI</td>
<td>0.468 0.063</td>
<td>1.196 0.099</td>
<td>4.728 0.073</td>
<td>2.77 [2.67 to 2.87]</td>
</tr>
</tbody>
</table>

Figure 1 Mean scores of comorbidity indexes from diagnosis year by Rheumatoid arthritis

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.


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EXERCISE MAY DECREASE SYMPOTOM 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 RAE 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: Exercise may decrease syncope secondary to posture change in females with rheumatoid arthritis.