22 (55%) were male and median age was 49.5 ± 11.5 years. Family history of psoriasis was present in 19 (47.5%) and 7 (17.5%) had spondyloarthritis family history. Obesity and overweight were the comorbidities most found, 42.9% and 37.1%, respectively, followed by hypertension 25%, dyslipidaemia and treatment. Eleven patients (28.9%) presenting moderate to severe PASI. Thirty-one patients presenting prolonged disease duration (> 10 years) with cutaneous (74.2%) and articular involvement. Peripheral disease without axial involvement was present in 28 patients (90%) while the others (10%) had both axial and peripheral involvement. Severe or moderate articular disease activity (DAS28) was present in 52.6% of the patients. The most frequent extra-articular manifestation was dactylitis (23.3%) and enthesitis (19.4%). The average number of multidisciplinary clinical consultations for these patients was 2. There was a statistically significant difference at the mean age, gender and disease duration > 10 years between patients with psoriatic disease with and without arthritis (p = 0.047; p<0.01 and p=0.03, respectively); there was no statistically significant differences in family history of psoriasis (p=0.711) and spondyloarthritis (p=0.174), nutritonal status (p=0.732) and comorbidities such as diabetes mellitus (p=0.545), hypertension (p=0.404), dyslipidaemia (p=0.39) and depression (p=0.089).

In the remaining 10 patients screened, the osteoarthritis and/or tendonitis were responsible for the articular complaints in 6 patients and scalp seborrhea and eczema were responsible for the cutaneous complaints in the rest.

Conclusion: Despite the small number of patients observed in our multidisciplinary clinical, we found that this kind of clinical care may facilitate the diagnosis of joint disease and offers a more comprehensive treatment approach for patients with both psoriasis and PsA.

REFERENCES:


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Table 1. Spearman’s correlations of the 3 and 4 VAS scores with TJC, SJC, PsAID and HAQ

<table>
<thead>
<tr>
<th>Patient Reported</th>
<th>TJC</th>
<th>SJC</th>
<th>PsAID</th>
<th>HAQ without aids</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 VAS vas</td>
<td>0.51</td>
<td>0.44</td>
<td>0.88</td>
<td>0.62</td>
</tr>
<tr>
<td>4 VAS vas</td>
<td>0.54</td>
<td>0.47</td>
<td>0.89</td>
<td>0.65</td>
</tr>
<tr>
<td>3 VAS nrs</td>
<td>0.49</td>
<td>0.43</td>
<td>0.89</td>
<td>0.63</td>
</tr>
<tr>
<td>4 VAS nrs</td>
<td>0.53</td>
<td>0.46</td>
<td>0.92</td>
<td>0.67</td>
</tr>
</tbody>
</table>


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Figure 1: Bland-Altman plots comparing VAS and NRS for patient reported components of 3VAS (left) and 4VAS (right).

Figure 2: Visual analog scale (scored 0-10) and numerical rating scale (scored 0-10).

Table 1. Comparison of Numerical Rating Scale (NRS) and Visual Analogue Scale (VAS) in the Patient Reported Outcome Measures of 3VAS and 4VAS in Psoriatic Arthritis

| Key | 3VAS; Patient global and skin VAS, 4 VAS: Patient pain, joint and skin VAS. 3 and 4 NRS; Numeric Rating Scale | TJC/ SJC: Tender/Swollen Joint count. PsAID: Psoriatic Arthritis Impact of Disease. HAQD: Stanford Health Assessment Questionnaire. |
Methods: 140 PsA patients were at baseline examined with dual energy x-ray absorptiometry (DXA) with measurements at femoral neck and spine (L1-4). Trained nurses performed the DXA scans. The DXA machines used at baseline (Lunar Prodigy) and at 5 year follow-up (Lunar iDXA) were stable over the measurement periods. Cross calibration using a spine phantom was performed between the two DXA machines (BMD of the old phantom in the new machine and the old phantom in the old machine, with 55 measurement over a time period of 2 weeks) had a difference of less than 1%. Demographic, disease measures and treatment data was collected at the same day or within 14 days to the date of DXA assessment. For group comparison, we used paired student t-test.

Results: After 5 years, 114 PsA patients (50.4% women) were re-examined. Baseline mean (SD) was for age 51.4 (9.4) years, BMI 28.1 (3.4) kg/m2, disease duration 9.1 (6.7) years. Disease characteristics at baseline and follow-up presented as mean (SD) were: DAS28 5.98 (1.2) and 3.73 (0.9); ASDAS-CRP 3.7 (1.5) and 2.0 (0.6); DAPSA 38.5 (12.9) and 23.8 (10.4); CRP 14.9 (11.1) and 10.9 (2.9) mm/hr; mHAQ 0.40 (0.35) and 0.38 (0.37). The proportions of patients as baseline and follow up giving conventional synthetic disease modifying anti-rheumatic drugs (DMARD) were 64.1% vs. 46.5%; for biological DMARDs 37.7% vs. 48.2%; for prednisolone 5.3% vs. 10.5%, for targeted osteoporosis medication 0.9% vs. 3.3%, for calcium and vitamin-D 11.4% vs. 26.3%. As shown in the table, no significant change in femoral neck and spine BMD and T-scores was found for the 114 patients. However for Z-scores (age and weight adjusted T-score) a significant increase was found both at femoral neck and lumbar spine. When gender was examined separately no significant reduction in bone density was found in men whereas in women a significant reduction during follow up was only found for right femoral neck BMD.

Conclusion: No reduction in BMD at femoral neck and spine was found in our PsA outpatient clinic cohort at baseline or over 5 years follow up. Rather, we found a statistically significant increase in age adjusted bone density (Z-score) both at femoral neck and spine over time. Our data thus adds evidence that PsA patients treated in ordinary clinical practice do not seem to be at an increased risk of developing osteoporosis.

REFERENCES:

A group of 65 patients (RA15/ AS25/ PsA25) with high disease activity at -Division of Rheumatology, Kristiansand.

Disclosure of Interests: None declared.

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Osteoarthritis

POS1087 USING LIPIDOMICS TO PREDICT PREDNISOLONE TREATMENT RESPONSE IN PATIENTS WITH INFAMMATORY HAND OSTEOARTHRITIS: THE HOPE STUDY

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Background: Lipidomics analysis has become a valuable technology for understanding pathophysiological mechanisms and may aid the identification of biomarkers of therapeutic responsiveness.

Objectives: To explore the use of lipidomics for prediction of prednisolone treatment response in patients with inflammatory hand osteoarthritis.

Methods: The Hand Osteoarthritis Prednisolone Efficacy (HOPE) study is a blinded, randomised placebo-controlled trial, that investigated the effect of prednisolone treatment in patients with painful, inflammatory hand OA, fulfilling the American College of Rheumatology criteria. The present analyses comprised only patients randomized to daily 10 mg prednisolone treatment for six weeks. Response to prednisolone treatment was defined according to the OARSI-OMERACT responder criteria at six weeks. Baseline blood samples were obtained non-fasted. Lipid species were quantified in erythrocytes with the Lipidyzer® platform (Lipodyzer, Sweden). After pre-processing of the data, 258 lipid species were available for further analyses (nmol/mL). In addition, we used an in-house LC-MS/MS platform to analyse oxylipins in plasma, identifying 25 oxylipins (area%)

Disclosure of Interests: None declared.

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POS1086 DIFFERENCES IN PROINFLAMMATORY AND LIPID PROFILE BETWEEN PSORIATIC ARTHRITIS, ANKYLOSING SPONDYLITIS AND RHEUMATOID ARTHRITIS

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Background: Patients with rheumatic diseases such as rheumatoid arthritis (RA), psoriatic arthritis (PsA), and ankylosing spondylitis (AS) are at increased risk of developing dyslipidaemia and premature cardiovascular disease (CVD)[1].

Objectives: To investigate the relationship between proinflammatory cytokines, microRNA, and lipid profile in patients with RA, AS, and PsA.

Methods: A group of 65 patients (RA/AS/ Ps A25) with high disease activity (mean DAS28 5.78 / ASDAS-CRP 3.77 / DAPSA 38.5) and 25 healthy controls (HC) were compared. Lipid profile comprised triglycerides (TG), total cholesterol(TC), low (LDL), and high-density lipoprotein (HDL). Serum concentrations of IL-6, IL-21, IL-17, TNF and osteoprotegerine (OPG) were measured by commercially available enzyme-linked immunosorbent assays. Expression of miR-233-5p, miR-92-3p,miR-485-3p,miR-10b-5p,let-7d-5p,miR-26a -2.3-2.5p levels in sera was normalized to miR-16a internal control. The Mann-Whitney test was applied for intergroup comparison, the correlation was assessed using Spearman’s Rank test.

Results: Patients with RA revealed mixed dyslipidemia (mean values:TC175; LDL 117; HDL; 48 TG; 124 mg/dL; PsA revealed hyperglycemia (TC 175; LDL 100; HDL: TG130 mg/dL) and in AS no specific profile was found (TC 179; LDL98; HDL55; TG103 mg/dL). Higher expression of miR-485was observed in patients with PsA (4.8-fold) and AS (5.9-fold) compared to RA and HC groups (3.1, and 1.5-fold p=0.02). Similarly, miR-26a revealed higher expression in patients with PsA (28.4-fold) and AS (21.5-fold) than in RA and HC (3.5, and 2.9-fold p=0.00). PsA patients had higher expression of miR-146b than patients with RA, AS and HC (40.9-fold vs 12.6 vs 15.3 vs 3.4-fold p=0.003) and higher miR-10b (11.7 vs 1.4 vs 4.9 vs 1.7-fold p=0.004). Patients with RA showed higher expression of let-7d than patients with PsA, AS and HC (22-fold vs 1.8 vs 2.3 vs 19.1,9-fold p=0.002). In PsA miR-92b expression correlated negatively with HDL levels (r=-0.62 p=0.02) and positively with fasting glucose (r=0.71 p=0.00). TG levels negatively correlated with TNF (r=-0.47 p=0.01), IL-17 (r=0.49 p=0.01) and OPG (r=-0.51 p=0.00) serum levels. Let-7d correlated negatively with TC (r=-0.56 p=0.03). In RA IL-21 positively correlated with LDL (r=0.71 p=0.00) and TC (r=0.75 p=0.001) concentrations. TG levels correlated positively with expressions of miR-92b (r=0.69 p=0.02) and miR-26a (r=0.69 p=0.03). In AS expression of let-7d was correlated positively with LDL (r=0.41 p=0.00) and TC (r=0.45 p=0.00) levels and negatively with ASDAS-CRP (r=-0.675 p=0.02) and CRP levels (r=-0.53 p=0.01). There were no significant differences in OPG, IL-21 concentrations or miR-146b, miR-92b,miR-233 expressions.

Conclusion: Differences in proinflammatory profile in RA, PsA, and AS seem to be associated with different phenotypes of dyslipidemia. In PsA expression of miR-92b and higher levels of TNF, IL-17 and OPG are associated with altered lipid profile and hypertriglyceridemia. High activity of AS is associated with lowered expression of let-7d, possibly influencing TC and HDL levels. Therefore, measuring lipid profile in active disease might be misleading. In active RA increase of TC and LDL levels was associated with high IL-21 concentration, while hypertriglyceridemia with miR-92b and miR-26a expressions.

REFERENCES:

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Osteoarthritis