THU0307

QT INTERVAL AND ITS CORRELATIONS WITH TRADITIONAL RISK FACTORS OF DEVELOPMENT OF CARDIOVASCULAR DISEASES IN PATIENTS WITH ACTIVE EARLY PSORIATIC ARTHRITIS

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Background: Cardiovascular diseases (CVD) are leading cause of morbidity and mortality in patients (pts) with psoriatic arthritis (PsA). An abnormally prolonged and shortened QT interval are associate with an increased risk of ventricular arrhythmias and sudden cardiac death.

Objectives: to evaluate QT interval during Holter monitoring and cardiovascular (CV) risk assessment using SCORE (Systematic COronary Risk Evaluation) in early PsA (EPsA) pts.

Methods: We included data of 48 (F:23) DMARD-naive EPsA pts (according to the CASPAR criteria) with no history of CVD: mean age - 36 [18-71] years, EPsA duration - 6.9 [1-23] months, DAS – 3.9 [3.27; 4.1], C-reactive protein – 19.4 [8.8; 37.6] mg/l. Controls subjects were matched by age, sex (n=48). All pts were assessed for traditional risk factors of CVD, ESC guidelines, 2016 24 hour ECG monitoring were analysed for QT interval corrected for heart rate (QTC). Prolonged QTC was defined as >460 ms in women and >450 ms in men, short QTc -<330 ms. Ten-year risk of CV death was estimated using SCORE algorithms, ESC guidelines, 2016 categorised as low (<1%), intermediate (1% to <5%), high (≥5% to<10%) or very high (≥10%). Intima-media thickness of the carotid artery (cIMT) was measured using a high-resolution B-mode ultrasound machine.

Results: QT interval during the 24 hours was significantly prolonged in EPsA pts when compared to the control group (table 1). We didn’t find short or prolong QT interval in EPsA pts and control group.

Abstract THU0307 – Table 1. QTc interval in EPsA pts and control group

<table>
<thead>
<tr>
<th>Parameters</th>
<th>EPsA</th>
<th>Controls</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QTc (ms), day</td>
<td>397[376; 404]</td>
<td>387.5[370; 396]</td>
<td></td>
</tr>
<tr>
<td>QTc (ms), night</td>
<td>396[377; 408]</td>
<td>390[367; 396.5]</td>
<td></td>
</tr>
<tr>
<td>QTc (ms), 24 hour</td>
<td>395[376; 406]</td>
<td>387[370; 396]</td>
<td></td>
</tr>
</tbody>
</table>

Data are presented in median values and interquartile range, 1*p<0.05 (nonparametric paired Mann-Whitney U test). 62.5% of patients with EPsA were classified as being at low risk 10 year risk of CV death using the SCORE algorithm, 6.25% pts – intermediate risk, 29.2% pts – high risk, 2.1% pts – very high risk. Increased cIMT was found in 11 (22.9%), atherosclerotic plaques – in 15 (31.3%). We found significant correlations between age and QTc duration during the 24 hours (R=0.48), as well as in both day (R=0.46) and night periods (R=0.45), for all p<0.05. We didn’t find correlations between QTc duration and traditional risk factors of CVD, disease activity of EPsA. Significantly correlations were observed between SCORE level and abdominal obesity (R=0.43, p<0.05), BMI (R=0.41, p<0.0001), cIMT (R=0.41, p<0.05).

Conclusions: QT interval was significantly prolonged in EPsA pts when compared to the control group. The age of pts was associated with increase of the QTc interval. 29.2% of patients were classified as being at high risk 10 year risk of CV death using the SCORE algorithm. The increase level of SCORE associated with a subclinical atherosclerosis. Combination of prolonged QT interval and carotid atherosclerosis confirms presence of high cardiovascular risk in EPsA pts.

Disclosure of Interest: None declared


THU0308

CALPROTECTIN AS A MARKER OF DISEASE ACTIVITY IN PATIENTS WITH NEW ONSET PSORIATIC AND RHEUMATOID ARTHRITIS: CORRELATION WITH ULTRASONOGRAPHIC SYNOVITIS

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Background: Serum Calprotectin has been tested as a marker of disease activity in psoriatic (PsA) and rheumatoid (RA) arthritis. In RA and in PsA on TNF inhibitors remission in comprobtin correlates with power-Doppler (PD) positive ultrasonographic (US) synovitis, while there is no data on untreated patients with new-onset PsA.

Objectives: to investigate the correlation and association between calprotectin and US synovitis in patients with new-onset PsA and in a control group of RA.

Methods: Consecutive patients with PsA and a group of gender- and age-matched patients with RA, referred to an early arthritis clinic (2005–2014) were included. Demographic and clinical features, including a 44 joint count for tenderness and swelling (TJC, SJC) and C-reactive protein (CRP) were recorded. US of wrists (radiocarpal, intracarpal and ulnocarpal) and MCP joints (proximal interphalangeal (PIP) and distal interphalangeal (DIP)) (GS) and PD synovitis scored 0–3 at each site, with a total score from the sum of each site, was available at the same time, as well as serum samples to measure calprotectin concentration. Serum levels of calprotectin were compared by Mann Whitney test in PsA and RA. The correlation between calprotectin, TJC, SJC, CRP and US PD and GS was evaluated by Spearman’s correlation coefficient, while the association of calprotectin concentrations and PD synovitis by regression analysis.

Results: 156 patients (78 PsA and 78 RA) were included (RA: male 28.2%, mean (sd) age 51.9 (13.3); PsA male 32%; mean age 51.7 (13.5)). Patients with RA had significantly higher CRP (median, IQR) (0.6, 0.3–2.1 vs 0.36, 0.3–1, p<0.04), SJC (7; 5–12 vs 6, 3–9, p<0.008), GS (6, 4–11 vs 5, 2–7, p<0.001) and PD (2, 0–9 vs 1, 0–3, p<0.003) scores. Calprotectin (ng/ml, median, IQR) did not significantly differ in PsA (3123, 2063–4669) and RA (2556, 1615–4441), also when separating poliarticular and oligoarticular PsA. In patients with PsA, calprotectin significantly correlated with GS score (r=0.340, p<0.007), PD score (r=0.290, p<0.02) and with the presence of PD (categorical variable) (r=0.263, p<0.04) while in RA there were no statistically significant correlations. When separating poliarticular and oligoarticular PsA, a significant correlation between calprotectin and GS score (r=0.369, p<0.01) and PD score (r=0.363, p<0.02) was confirmed in poliar-ticular but not oligoarticular disease. In both RA and PsA SJC and TJC did not significantly correlate with calprotectin. Calprotectin showed a statistically significant correlation with CRP in both PsA (r=0.273, p<0.01) and RA (r=0.27, p<0.01), showing concurrent validity. In regression analysis, calprotectin levels did not associate with the presence of PD in PsA also when using a more stringent cut-off. Similar results were achieved in RA.

Conclusions: In untreated patients with early onset PsA, but not in RA, calprotec-tin correlates with US PD-positive synovitis, especially in poliar-ticular disease. Prospective studies are needed to confirm the use of calprotectin as a biomarker in early inflammatory arthritides.

Disclosure of Interest: None declared

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