THU0307

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

E. Markelova, D. Novikova, T. Korotaeva, I. Kirillova, E. Loginova. V.A. Nasonova
Research Institute Of Rheumatology, Moscow, Russian Federation

Background: Cardiovascular diseases (CVD) are leading cause of morbidity and mortality in patients (pts) with psoriatic arthritis (PsA). An abnormally prolonged and shorted 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^[26; 47] years, EPsA duration - 6.9^[4; 12] months, DAS - 3.97 [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 (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 (c-IMT) was measured using a high-resolution B-mode ultrasound machine.

Results: QTc 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 QTc interval in EPsA pts and control group.

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

Parameters	EPsA pts	Controls
QTc (ms), day	397[376; 404]	387.5[370.5; 396]*
QTc (ms), night	396[377; 408]	390[367; 396.5]*
QTc (ms), 24 hour	395[378; 406]	387[370; 396]*

Data are present in median values and interquartile range, *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), c-IMT (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

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THU0308

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

¹G. Sakellariou, G. Lombardi², B. Vitolo¹, M. Gomarasca², M. Faraldi², R. Caporali¹, G. Banfi^{2,3}, C. Montecucco¹. ¹Chair And Division Of Rheumatology, IRCCS Policlinico San Matteo Foundation, Pavia; ²Laboratory of Experimental Biochemistry and Molecular Biology, I.R.C.C.S. Istituto Ortopedico Galeazzi; ³Vita-Salute San Raffaele University, Milano, Italy

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 in remission calprotectin 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 age and gender-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 with grey scale (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. Secondary analyses separating polyarticular and oligoarticular (SJC ≤4) PsA and using different definitions of synovitis (GS >1, PD >1) were performed.

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 0.01) 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 polyarticular and oligoarticular PsA. In patients with PsA, calprotectin significantly correlated with GS score (rho 0.340, p 0.007), PD score (rho 0.290, p 0.02) and with the presence of PD (categorical variable) (rho 0.263, p 0.04), while in RA there were no statistically significant correlations. When separating polyarticular and oligoarticular PsA, a significant correlation between calprotectin and GS score (rho 0.369, p 0.01) and PD score (rho 0.363, p 0.02) was confirmed in polyarticular 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 (rho 0.273, p 0.01) and RA (rho 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, calprotectin correlates with US PD-positive synovitis, especially in polyarticular disease. Prospective studies are needed to confirm the use of calprotectin as a biomarker in early inflammatory arthritis.

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THU0309

TEN YEARS FOLLOW-UP STUDY OF CLINICAL DISEASE STATUS AND TREATMENT IN PSORIATIC ARTHRITIS PATIENTS FROM AN OUTPATIENT CLINIC IN SOUTHERN NORWAY

^{1,2}G. Haugeberg, S. Tengedal³, I.J.W. Hansen³, B. Michelsen³, A. Diamantopoulos¹, A. Kavanaugh⁴. ¹Rheumatology, Martina Hansens Hospital, Bærum; ²Research Unit; ³Rheumatology, Hospital of Southern Norway Trust, Kristiansand, Norway; ⁴Center for Innovative Therapy, UCSD, San Diego, USA

Background: In the new millennium remission has become an obtainable treatment goal for chronic inflammatory joint disorders, shown in particular for rheumatoid arthritis (RA). This has been attributed to new treatment strategies (early intervention and treat-to-target) and new drugs e.g. biologic Disease Modifying Anti-Rheumatic Drugs (bDMARDs).¹ For psoriatic arthritis (PsA) there is a lack of longitudinal long term clinical data illuminating potential changes that may have occurred over the years.

Objectives: To explore long term changes in clinical disease status and treatment in PsA patients monitored in an ordinary Norwegian outpatient clinic in the period 2008–2017.

Methods: For each year we collected data from last patient visit recorded in the hospital clinical computer system GoTreatIT Rheuma. Included patients had to fulfil the CASPAR criteria and have peripheral arthritis. Standard clinical data collection included demographic data, clinical measures of disease activity (Disease Activity Score with 28 joint counts [DAS28], Clinical Disease Activity Index [CDAI] and Assessors Global Assessments (AGA) on Visual Analogue Scale 0–100 mm (VAS)) laboratory measures of disease activity (Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP) and Patient-Reported Outcomes Measures (PROMs) of physical function (MHAQ), morning stiffness and VAS scores for joint pain, fatigue and Patient Global Assessment (PGA). Treatment with prednisolone, synthetic DMARDs (sDMARDs) or bDMARDs, was recorded.

Results: Over the 10 years mean annual number of PsA patients monitored was 331, mean age 58.4 years, disease duration 9.6 years, BMI 27.6 kg/m², females 49% and current smokers 17.6%. A statistically significant decrease for measures of disease activity for the period 2008–2017 was seen (all p<0.01): ESR 15.8–10.9 mm/hr, CRP 7.6–4.1 mg/dl, 28 swollen joints 1.5–0.6, 28 tender joints 3.3–1.6, DAS28 3.32–2.46, CDAI 10.1–6.8 and AGA 14.5–8.6 mm. No statistically significant changes in PROMs was seen. Mean values for the period was: MHAQ 0.46, joint pain 36.3 mm, fatigue 44.2 mm, PGA 38.8 mm and morning stiffness 0.95 hour. From 2008 to 2017 the percentage of patients treated with bDMARDs and/or sDMARDs and/or prednisolone increased from 72.6% to 80.9%. For the