

Table 1. The distribution of US-GLOESS according to different categories by indices

	GLOESS 38 joints mean±SD	GLOESS 28 joints mean±SD	Joint counts ≥ score 2 mean±SD	Joint counts with score 3 mean±SD
DAS-28				
Remission (n=77)	10.9±7.5	7.8±7.2	3.5±3.1	0.7±1.3
LDA (n=11)	14.5±6.6	10.0±5.4	4.9±3.0	0.9±1.4
VMDA (n=5)	9.6±6.2	5.4±3.5	3.0±2.3	0.80±1.7
CDAI				
Remission (n=48)	10.0±6.9	6.9±6.2	3.02±2.7	0.60±1.0
LDA (n=48)	12.7±7.7	9.1±7.2	4.3±3.3	0.94±1.5
SDAI				
Remission (n=42)	9.9±6.6	6.6±5.9	2.9±2.6	0.5±0.9
LDA (n=50)	12.4±7.8	9.1±7.5	4.2±3.3	0.9±1.5
MDA (n=1)	13.0	4.0	5.0	0
RAPID				
Remission (n=36)	10.3±6.2	6.7±4.6	3.1±2.6	0.5±0.8
Low activity (n=28)	9.6±5.8	6.6±4.6	2.7±2.2	0.3±0.7
MDA (n=30)	14.0±9.4	10.7±9.7	5.1±3.9	1.4±1.9
HDA (n=2)	15±1.4	8.5±2.1	5±1.4	0

LDA: Low disease activity MDA: Moderate disease activity HDA: High Disease activity.

Conclusions: Our results show that CDAI is superior then other clinical indices to assess remission in RA. US has the superiority over clinical indices to predict flares and 28 joint GLOESS is superior to 38 joints.

Disclosure of Interest: None declared

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SAT0064 HIGH MMP3 SERUM LEVELS ARE ASSOCIATED WITH EXTENSIVE STRUCTURAL DAMAGE IN PATIENTS WITH EARLY, TREATMENT NAIVE RHEUMATOID ARTHRITIS (RA): TWO YEARS PROSPECTIVE CLINICAL AND ULTRASONOGRAPHIC STUDY

S.Z. Prodanovic¹, G. Radunovic², M. Sefik-Bukilica³, M. Zlatanovic⁴, K. Simic-Pasalic⁵, S. Seric⁶, N. Damjanov⁷. ¹Clinical IVb; ²Deputy of head; ³Clinical IVa; ⁴Clinical III; ⁵Clinical VI; ⁶Radiology Department; ⁷Head of Institute, Institute of Rheumatology, Belgrade, Serbia

Objectives: To investigate the association of high baseline MMP-3 serum levels with hands and feet joints structural damage progression, estimated by ultrasonography (US), in patients with early, treatment "naïve" RA without X-ray visible erosions.

Methods: Sixty-three pts. (9 males and 54 females; mean age 53.4 yrs 21–81±14.1) with early RA (EULAR/ACR 2010 criteria) and symptom duration of ≤12 months (mean duration of 3.8 months) had baseline serum MMP-3 levels tested. Patients had been DMARDs/glucocorticoid naïve, without visible X-ray erosions at the study entry. The subsequent structural joints damages, that were estimated by high frequency linear probe by ESAOTE My Lab 70 machine, as well as clinical markers of disease activity, in the first 2 years were followed. The presence of bone erosion was analyzed at the wrist, MCP2 and MCP5 joints of both hands, as well as at MTP5 joints according to OMERACT US group definition. In order to estimate progression of preexisting erosion the total volume (TV) of bone erosion were calculated by multiplying three diameters (mm): a-the length of erosion; diameter b- the width and diameter c-the depth of erosion. Anova statistical method was performed in data processing.

Results: 46 pts. had basal serum MMP-3 level higher than normal (MMP-3 positive). The 504 joints were assessed summary by US on each visit. The 122 bone erosions in total (1.9 per patient) were depicted at baseline and 213 bone erosions (4.3 per patient) at follow-up visit in whole group. After 24 months MMP3 positive pts. had significantly higher total number of US erosions than MMP-3 negative (3.8 vs. 2.4, p=0.039). The total volume of bone erosion (mm³) was higher in MMP-3 positive than MMP-3 negative pts. after 24 months of treatment, but without statistical significance (18.6 vs. 8.9, p=0.07). MMP-3 positive pts. had a significantly higher value of ESR, CRP and DAS28 than MMP-3 negative pts. at the baseline visit (53.6 vs.25.9, p=0.002; 36.6 vs. 9.5, p=0.005; 6.0 vs. 4.8, p=0.002, respectively). All of those parameters were significantly decreased after 24 months in a group of MMP-3 positive compared to MMP-3 negative pts. (p=0.009, p=0.021, p=0.028, respectively).

Conclusions: After 2-year of follow-up, US assessment showed a significantly higher number of new bone erosions in patients with early, treatment "naïve" RA and baseline MMP3 levels higher than normal (MMP3 positive) compared to patients with normal baseline levels of MMP3, as well as a bigger TV of bone erosions but not statistically significant. The parameters of RA activity (ESR, CRP, DAS28) significantly correlated with baseline MMP-3 levels of higher than normal.

Disclosure of Interest: None declared

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SAT0065 ACPA AGAINST DIFFERENT CITRULLINATED PEPTIDES IDENTIFY SPECIFIC PHENOTYPES OF RHEUMATOID ARTHRITIS

M. Brink¹, M. Hansson², L. Mathson-Alm³, M. Cornillet⁴, J. Rönnelid³, K. Skriner⁵, G. Serre⁴, R. Holmdahl⁶, L. Klareskog², S. Rantapää-Dahlqvist¹. ¹Public Health and Clinical Medicine, Rheumatology, Umeå University, Umeå; ²Rheumatology Unit, Dept of Medicine, Karolinska Institute, Stockholm; ³Immunology, Genetics and Pathology, Uppsala University, Uppsala, Sweden; ⁴U1056 Inserm, Univeristy de Toulouse, Toulouse, France; ⁵Medicine, Charité University, Berlin, Germany; ⁶Medical Inflammation Research, Karolinska Institute, Stockholm, Sweden

Background: Anti-citrullinated protein/peptide antibodies (ACPA) have been suggested to identify a more severe phenotype of rheumatoid arthritis (RA).

Objectives: In this study in an inception cohort of early RA we have analysed a number of antibodies against different citrullinated and/or mutated peptides using a multiplex platform in relation to the patients disease inflammation and radiological destruction

Methods: Patients with early RA (≤12 m of symptoms) fulfilling the 1987 ARA criteria (n=1022, 692f/330m, mean age56.7±14.0 years) were sampled at the time of diagnosis and assessed using disease activity score (DAS28) at baseline, 6, 12, 18 and 24 months. Radiographs were graded using Larsen score (baseline and at 24 m). Plasma sampled at baseline was analysed for presence of antibody reactivities against 21 different citrullinated peptides/proteins; Fibrinogen (Fib) α36–50, Fibα573, Fibα591, Fibα621–635, Fibβ36–52, Fibβ60–74, Fibβ62–78 (72), Fibβ62–78 (74), Filaggrin (Fil307–324), α-Enolase peptide 5–21 (CEP-1), Vimentin (Vim) 2–17, Vim60–75, F4-R-Cit, F4-Cit-Cit, F4- Cit-R), or mutated proteins (Bla26, Pept1, Pept5, PeptZ1, PeptZ2) and type II Collagen citrullinated or not using a custom-made microarray assay based on the ImmunoCAP ISAC system (Phadia AB, Sweden). Cut-off levels were at the 98th percentile of controls (n=477). Anti-CCP2 was analysed using ELISA (Euro Diagnostica, Sweden).

Results: The most frequent appearing ACPA were; Fibβ60–74 (63%), Vim60–75 (56.6%), Fibβ36–52 (55.1%), Fil307–324 (54.9%), CEP-1 (53.7%) and Pept5 (52.0%) besides CCP2 (67.5%). Adding all ACPAs gave additional 13.1% of positivity in the anti-CCP2 negative group, yielding a positivity of 77.5%. The median (IQR) number of positive ACPA-peptide was 8 (11). There was a high degree of correlation between the antibodies, e.g., anti-Fibβ60–74 vs. -Vim60–75, -Fibβ36–52 or anti-CCP2 antibodies and also anti-Fil307–324 vs. -Fibβ36–52, -F4 R-Cit or anti-CCP2 antibodies (rs 0.692–0.79). Positivity for all antibodies was associated with higher ESR (baseline and AUC₂₄). A number of antibodies were associated with both high DAS28 (baseline and AUC₂₄) and radiological findings/progression (anti-CCP2, -Fil307–324, -Vim60–75 and Vim 2–17, and -CEP1 antibodies), whilst some others were more associated with inflammation (DAS28, baseline and AUC₂₄) (anti-Fibβ60–74, -Pept5 and -F4R-cit antibodies) and others more with radiological destruction/progression (anti-Fibβ36–52, -Fibβ74, -PeptZ1, -F4 Cit-R antibodies). Partial least squares regression analyses confirmed the results with significant correlation between radiological progression and antibodies against Vim2–17, Fibβ36–52, CEP1, Fibα621–635, and CCP2 and between DAS28AUC₂₄ and Vim60–75, Vim2–17, Fibα621–635 and F4-R-Cit. Patients treated with biologics during the first 24 months (11.2%) were significantly more frequent positive for anti-CCP2, -Vim60-75, -Fibα36–50, -PeptZ1 and -PeptZ2 antibodies vs. being negative.

Conclusions: Analyses at baseline, of the ACPA specificity profiles allowed different patterns of disease activity and radiological progression during the first 24 months of the disease to be identified.

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SAT0066 HISTOLOGICAL AND ULTRASOUND SYNOVIAL PREDICTORS OF CLINICAL DIFFERENTIATION TO DEFINED ARTHRITIS IN PATIENTS WITH SERONEGATIVE UNDIFFERENTIATED PERIPHERAL INFLAMMATORY ARTHRITIS

S. Alivernini¹, L. Petricca¹, B. Toluoso¹, L. Bui², C. Di Mario¹, M.R. Gigante¹, G. Di Sante¹, R. Benvenuto², A.L. Fedele¹, F. Federico², E. Gremese¹, G. Ferraccioli¹. ¹Institute of Rheumatology; ²Institute of Pathology, Catholic University of the Sacred Heart, Rome, Italy

Background: Undifferentiated Peripheral Inflammatory Arthritis (UPIA) is a common diagnosis at the first clinical evaluation in rheumatological settings. However, the likelihood of developing a well-defined rheumatic disease in UPIA patients is still matter of debate.

Objectives: To examine the role of ultrasound (US) and histological parameters in the disease outcome of patients with seronegative UPIA.

Methods: Fourty-two patients with IgA/IgM-Rheumatoid Factor and anti-citrullinated peptide antibodies negative UPIA, naïve to any Disease-Modifying Anti-Rheumatic Drugs, underwent Gray Scale (GSUS) and power Doppler (PDUS) evaluation and US guided synovial tissue biopsy. Synovial expression of CD68, CD3, CD21, CD20 and CD31 was evaluated by immunohistochemistry. IL-6, VEGF-A and VEGF-D peripheral blood (PB) and synovial fluid (SF) levels were measured by ELISA. To exclude Reactive Arthritis, each patient underwent genital and throat swabs. Afterwards, each UPIA patient was treated with chloroquine 250 mg/daily and followed every 3 months for 1 year and classified as having