earlier diagnosis from symptom onset ($p=0.0001$), higher titer of rheumatoid factor ($p=0.037$) and female gender ($p=0.019$). In early SpA pts the only predictor of shorter MTXm survival was younger age at diagnosis ($p=0.0,37$). Smoking, disease activity at diagnosis and BMI were not predictors of MTXm survival.

Conclusions: The MTXm survival is not influenced by VEA or EA diagnosis. The RA seems to be less responder to MTXm than SpA. It might be due to the difficult to capture the earliest phase of disease, as circulating antibodies might appear years before the onset of symptoms in RA, and biomarkers reflecting bone destruction are elevated before arthritis is present, as described in previous studies. In particular, might be pay attention to younger pts, female gender and pts with higher titer of rheumatoid factor as they seem to present a more resistant RA disease. A high dose of MTX and earlier interventions are suggested in RA to improve the clinical outcome of patients and the MTXm survival.

References:

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.4237

FRI0111 ANTI-CITRULLINATED PROTEIN ANTIBODY REACTIVITIES IN TREATMENT NAÏVE EARLY RHEUMATOID ARTHRITIS

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Background: An increase in the number of anti-citrullinated protein antibodies (ACPA) reactivities precede RA onset, and may be involved in the pathogenesis of the disease. The presence of ACPA is associated with radiographic progression in RA, and it has been suggested that ACPAs with different reactivities may be associated with different phenotypes of RA.

Objectives: To assess the prevalence of baseline ACPA reactivities in an inception cohort of early RA patients, including subgroups based on anti-CCP/RF status, and to compare the findings to healthy controls.

Methods: 217 DMARD-naïve early RA patients from the ARCTIC trial (1) were analysed. Radiographs were scored according to van der Heijde Sharp (vdHS) score. Anti-CCP status was analysed by FEIA (pos. if ≥ 10 IU/mL) and RF by ELISA (pos. if ≥ 25 IU/mL). ACPA titres (AU/ml) were considered pos. if above the 98-perc. of values in 619 non-RA subjects. Analysis of 13 ACPA reactivities targeting citrullinated peptides from fibrinogen, alpha-1 enolase, vimentin, fillagrin and histone was performed at baseline in patients and 94 controls (blood donors matched for age/gender/smoking), using a multiplex chip-based assay (2).

	anti-CCP+, n=178	anti-CCP-, n=39	RF+, n=154	RF-, n=63	All RA, n=217	Controls, n=94
Age, years ¹	50.8 (13.2)	55.0 (14.9)	51.9 (13.3)	50.8 (14.2)	51 (14)	52 (9.4)
Female ²	109 (61)	22 (56)	91 (59)	40 (64)	131 (60)	63 (59)
Ever-smoker ²	122 (69)	26 (67)	109 (71)	39 (62)	148 (68)	81 (64)
DAS ¹	3.4 (1.1)	4.0 (1.3)	3.5 (1.2)	3.5 (1.2)	3.5 (1.2)	NA
vdHS score ³	4.0 [1.5–8.0]	4.5 [2.0–10.0]	4.5 [2.0–8.0]	3.5 [1.5–10.0]	4.0 [1.5–8.0]	NA
Vim60–75 cit ⁴	152 (85)	7 (18)	130 (84)	29 (46)	159 (73)	5 (5)
H4 31–50 cit ⁴	142 (80)	3 (8)	119 (77)	26 (41)	145 (67)	1 (1)
CEP1 ⁴	137 (77)	3 (8)	117 (76)	23 (37)	140 (65)	1 (1)
Fil307–324 cit ⁴	134 (75)	2 (5)	113 (73)	23 (37)	136 (63)	0 (0)
Fib573 cit ⁴	121 (68)	2 (5)	99 (64)	24 (38)	123 (57)	0 (0)
Fib36–52 cit ⁴	116 (65)	1 (3)	96 (62)	21 (33)	117 (54)	2 (2)
H3 1–30 cit ⁴	106 (60)	1 (3)	93 (60)	14 (22)	107 (49)	0 (0)
H4 14–34 cit ⁴	103 (58)	2 (5)	90 (58)	15 (24)	105 (48)	3 (3)
H3 21–44 cit ⁴	94 (53)	2 (5)	80 (52)	16 (25)	96 (44)	1 (1)
Vim2–17 cit ⁴	87 (49)	1 (3)	80 (52)	8 (13)	88 (41)	0 (0)
Fib591 cit ⁴	66 (37)	3 (8)	56 (36)	13 (21)	69 (32)	1 (1)
Fib74 cit ⁴	60 (34)	6 (15)	54 (35)	12 (19)	66 (30)	3 (3)
Fib72 cit ⁴	24 (14)	3 (8)	21 (14)	6 (10)	27 (12)	2 (2)

¹Mean (SD), ²n (%), ³Median [IQR], ⁴ACPA reactivity, n positive (%).

Results: Baseline characteristics are presented in the table. The figure shows the prevalence of ACPA reactivities in the subgroups, with median [IQR] number of antibody reactivities in all patients 7 [3,10], compared to 0 [0,0] in controls ($p<0.001$). The corresponding numbers were 8 [5,10] and 0 [0,1] for the anti-CCP+ vs. anti-CCP- patients ($p<0.001$), and 8 [5,10] and 2 [0,7.5] for the RF+ vs. RF- patients ($p<0.001$). Positivity for ACPA reactivities was seen mainly in the anti-CCP+ and RF+ patients, but also occurred more frequently in RF- and anti-CCP- patients (table) than in controls (anti-CCP- vs controls $p=0.037$, RF- vs controls $p<0.001$).

Conclusions: Prevalence of ACPA reactivities differed in subgroups of DMARD-naïve early RA patients according to anti-CCP and RF status. All RA subgroups, including RF- and anti-CCP- patients, had higher prevalence of ACPA reactivities compared to healthy controls.

References:

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[2] Hansson et al *Arthr Res Ther* 2012.

Disclosure of Interest: M. Jonsson: None declared, A. Hensvold: None declared, M. Hansson: None declared, L. Mathsson-Alm Employee of: Thermo Fisher Scientific, A.-B. Aga: None declared, J. Sexton: None declared, B.-T. Fevang Grant/research support from: Novartis, S. Lillegraven: None declared, A. Catrina: None declared, E. Haavardsholm Grant/research support from: AbbVie, MSD, Pfizer, Roche, UCB

DOI: 10.1136/annrheumdis-2017-eular.2915

FRI0112 SERUM LEVELS OF IL-33 AND SST2 ARE ASSOCIATED WITH FUNCTIONAL DISABILITY IN RHEUMATOID ARTHRITIS

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Background: Interleukin 33 (IL-33) is a cytokine related to amplification of the articular inflammation in rheumatoid arthritis (RA) animal models. Elevated IL-33 serum levels have been described in RA patients, suggesting a possible participation of this cytokine in the pathophysiology of the disease.^{1,2} IL-33 soluble receptor (sST2) is a decoy receptor that functions as an inhibitor of the interaction of the transmembrane receptor with IL-33.³

Objectives: To identify the association between serum levels of IL-33 and its soluble receptor (sST2) with clinical and laboratory characteristics of RA.

Methods: Cross-sectional observational study in which RA patients were submitted to clinical and laboratorial evaluation. IL-33 and sST2 serum levels were measured by ELISA (R&D System Inc, Minneapolis, MN, USA).

Results: 102 RA patients were included, 92.5% women, mean age of 55.5±10 years and mean disease duration of 17.6±9.5 years. Eighty-four (82.4%) patients had seropositive RA. The median (interquartile range) IL-33 serum level was 69.1 pg/ml (31.6 - 114.5). Higher scores on the visual analogue scale (VAS) of disease activity assessed by the examiner were associated with higher IL-33 values (C95%: 0.01–0.05). In the group of patients with high titres of rheumatoid factor (RF), IL-33 levels were higher, compared to the group with negative RF (95% CI: 0.55 - 2.34). In 34 (33.3%) patients, IL-33 was undetectable and the presence of metabolic syndrome⁴ represented a 63% lower chance (OR =0.37, 95% CI: 0.15–0.90) of having IL-33 detected. In addition, 1-unit increase in HAQ-DI increased by 2.43 times the chance of detecting IL-33 (95% CI: 1.23 - 4.80). The median sST2 serum level was 469.8 pg/ml (336.3–651). sST2 was associated with worse functional capacity by the classification of Steinbrocker⁵ (C95%: 0.09 - 0.5), use (current or in the past) of tobacco (95% CI: 0.02 - 0.53) and use of leflunomide (95% CI: 0.05 - 0.53). There was no correlation between IL-33 and sST2 levels.

Conclusions: These findings may suggest that both IL-33 and its soluble receptor play a role as a marker of RA severity and functional disability. The negative association of IL-33 with metabolic syndrome is in agreement with the possible protective role of this cytokine in relation to lipid metabolism.⁶