binding their Ig-Fc portion. However, it has been demonstrated that FcγR single nucleotide polymorphisms (SNP) leads to low binding affinity alleles (FcγRIIA-131R, IIA-158V and IIB NA2). Therefore, we can make an assumption that the presence of these risk alleles could influence the outcome of immune mediated disease such as rheumatoid arthritis (RA).

Objectives: The aim of this study was to assess the relationship between functional SNP of FcγR IIA H13R, IIA V158F and IIB NA1/NA2 and disease severity scores in RA.

Methods: We assessed disease severity in RA patients based on the Health Assessment Questionnaire (HAQ) and Sharp/van der Heijde (mSharp) method. To reduce selection bias, all recruited patients were treated with conventional DMARDs. We considered patients with HAQ score ranged from 2 to 3 as having severe disability. For radiographic evaluation, since there is not validated threshold for joint damage severity, we took mSharp score median value of our population as a reference. Patients underwent FcγRIIA, FcγRIIA and FcγRIIB SNP study using PCR-SSP and direct sequencing process. Then, biallelic polymorphisms were examined for association with HAQ and mSharp score.

Results: Fifty-eight RA patients were enrolled. The mean duration of the disease was 11.77 years [0.5-24]. Among these patients, 34 (58.62%) had severe disability according to HAQ score. The mSharp median value was 43.5 [0-232] and 28 (48.28%) patients had a score above this value.

Genotypic study showed that FcγR IIB-NA2 was significantly associated with severe disability ($p=0.021$) (Table 1).

Table 1. Correlation between FcγR biallelic polymorphisms and disability level

Gene	Genotype	HAQ<2	HAQ≥2	OR (95% CI)	P-value
FcγR IIB	NA1/NA1	10 (41.7%)	5 (14.7%)	1.00	0.021
	NA1/NA2 – NA2-NA2	14 (58.3%)	29 (85.3%)	4.14 (1.19-14.44)	
FcγR IIA	V/V	4 (16.7%)	7 (20.6%)	1.00	0.71
	F/F-F/V	20 (83.3%)	27 (79.4%)	1.30 (0.33-5.04)	
FcγR IIB	H/H	3 (12.5%)	5 (14.7%)	1.00	0.81
	H/R-R/R	21 (87.5%)	29 (85.3%)	0.83 (0.18-3.86)	

Table 2. Correlation between FcγR biallelic polymorphisms and radiographic evaluation

Gene	Genotype	Sharp < 43.5	Sharp ≥43.5	OR (95% CI)	P-value
FcγR IIB	NA1/NA1	7 (23.3%)	8 (28.6%)	1.00	0.65
	NA1/NA2 – NA2-NA2	23 (76.7%)	20 (71.4%)	0.76 (0.23-2.47)	
FcγR IIA	V/V	5 (16.7%)	6 (21.4%)	1.00	0.64
	F/F-F/V	25 (83.3%)	22 (78.6%)	1.36 (0.37-5.09)	
FcγR IIB	H/H	5 (16.7%)	3 (10.7%)	1.00	0.51
	H/R-R/R	25 (83.3%)	25 (89.3%)	1.67 (0.36-7.74)	

Furthermore, FcγRIIA-158F and IIA-131R carriers were more frequent in patients with severe disability but the association was not statistically significant. Finally, no correlation was found between radiographic evaluation and FcγR SNP study (table 2).

Conclusion: FcγR low affinity alleles seem to confer susceptibility to severe functional impairment in RA but not to structural damage. However further studies on larger population must be done to corroborate these findings.

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Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.8299

AB0268

MORTALITY RATE OF RHEUMATOID ARTHRITIS PATIENTS EVEN IN THE NEW ERA OF BIOLOGICS IS HIGHER THAN THE CONTROL GROUP: EIGHT YEARS OF THE TOMORROW STUDY

Koji Mandai^{1,2}, Tatsuya Koike^{1,3}, Yuko Sugioka³, Kentaro Inui¹, Tadashi Okano¹, Yutaro Yamada¹, Masahiro Tada⁴, Kenji Mamoto^{5,6}, Shohei Anno⁵, Hiroaki Nakamura¹. ¹Osaka City University Graduate School of Medicine, Orthopaedic Surgery, Osaka city, Japan; ²Osaka social medical center Hospital, Orthopaedic Surgery, OSAKA, Japan; ³Osaka City University Medical School, Center for Senile Degenerative Disorders, Osaka city, Japan; ⁴Osaka City General Hospital, Orthopaedic Surgery, Osaka city, Japan; ⁵Yodogawa Christian Hospital, Orthopaedic Surgery, Osaka, Japan; ⁶Cleveland clinic, Biomedical Engineering, Program of Advanced Musculoskeletal Imaging (PAMI), Cleveland, United States of America

Background: Patients with rheumatoid arthritis (RA) have a high mortality rate compared to the general population¹. However, the mortality rate of patients with RA might be improved by advances in therapy².

Objectives: We investigated the risk factors for mortality in patients with RA from TOMORROW study.

Methods: This study included 413 participants, comprising 208 patients with RA and 205 age- and sex- matched healthy volunteers (Vo) from the prospective "TOMORROW" cohort study that has been ongoing since 2010 were included in this study (women, 84%; mean age, 58 years old). Median disease duration was 10.3 years.

Results: The rate of accomplishment for 8 years was 83.2% in the RA group and 92.7% in the Vo group. There were 14 deaths in the RA group (8.7/1000 person-years) and two in the Vo group (1.2/1000 person-years) ($p=0.0025$) for 8 years. Infection was the most common cause of death in the RA group (43%). Cox proportional hazard analysis showed that having RA was significant risk factor for death (hazard ratio [HR]: 6.9, 95%CI: 1.6 - 30.4, $p=0.01$) adjusting by age (Figure). In the RA group, glucocorticoid (GC) use (HR: 3.6, 95%CI: 1.1 -10.4, $p=0.032$) was significant risk factor for death. Disease activity, duration of disease, use of biological products, use of methotrexate, presence of cardiovascular disease, and smoking were not significant factor.

Fig: survival curve of RA and control

Figure 1

Conclusion: RA was still significant risk factor of death. GC use was independent factors of death in RA patients.

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Disclosure of Interests: Koji Mandai: None declared, Tatsuya Koike Speakers bureau: AbbVie, Astellas Pharma Inc., Bristol-Myers Squibb, Chugai Pharmaceutical, Eisai, Janssen, Lilly, Mitsubishi Tanabe Pharma Corporation, MSD, Ono Pharmaceutical, Pfizer, Roche, Takeda Pharmaceutical, Teijin Pharma, and UCB, Yuko Sugioka: None declared, Kentaro Inui Speakers bureau: Takeda Pharmaceutical, Pfizer Japan, Daiichi-Sankyo Co.Ltd., Abbvie, Mitsubishi Tanabe Pharma Corporation, Janssen Pharmaceutical, Chugai Pharmaceutical, Ono Pharmaceutical, Eisai Co. Ltd., Eli-Lilly, Nippon Kayaku Co.,Ltd., Maruho Co.,Ltd, Kaken

Pharmaceutical Co.,Ltd., Tadashi Okano Speakers bureau: AbbVie, Yutaro Yamada Speakers bureau: Abbvie, Chugai, Mitsubishi Tanabe, Masahiro Tada Speakers bureau: Abbvie, Astellas Pharma, Bristol-Myers Squibb, Chugai Pharmaceutical, Eisai, Janssen Pharmaceutical, Mitsubishi Tanabe Pharma Corporation, Ono Pharmaceutical, Pfizer Japan, Takeda Pharmaceutical, Kenji Mamoto: None declared, Shohei Anno: None declared, Hiroaki Nakamura: None declared

DOI: 10.1136/annrheumdis-2019-eular.1317

AB0269

COMPARISON BETWEEN DIFFERENT DISEASE ACTIVITY SCORES IN ELDERLY ONSET RHEUMATOID ARTHRITIS

Melania Martínez-Morillo, Águeda Prior-Español, Anahy Brandy-García, Susana Holgado, María Aparicio-Espinar, Laia Gifre, Anne Riveros-Frutos, Clara Sangüesa-Gomez, Jordi Camins-Fàbregas, Ivette Casafont-Solé, Annika Nack, Alejandro Olive, Lourdes Mateo Soria. *Hospital Germans Trias i Pujol, Rheumatology, Badalona, Spain*

Background: Elderly onset rheumatoid arthritis (EORA) has several peculiarities. Which activity index should we use in this age group is still an unresolved issue. The use of ESR in DAS28 is widespread, but it is well known that ESR could increase with age, overestimating activity. DAS28-PCR or SDAI, which uses PCR, or CDAI that does not use analytical data, could be more suitable alternatives.

Objectives: To describe disease activity scores evolution in EORA and to analyse the correlation and concordance between them.

Methods: Longitudinal observational study in naïve treatment EORA patients (ACR/EULAR 2010 criteria). Study visits were carried out basally (pre-treatment) and at month 1, 3, 6 and 12. DAS28 VSG, DAS28 PCR, SDAI and CDAI were calculated at each visit, and ACR/EULAR remission index at 12 months. The same rheumatologist made all visits and explorations. Correlations were analysed using the Spearman or Pearson correlation index (p value ≤ 0.001). The concordance was evaluated with the kappa index. The statistical study was performed with Stata 15.1.

Results: 45 patients with EORA were enrolled. Table 1 and 2 summarize the clinical and analytical characteristics.

All the scores had a very good linear correlation both at baseline and at follow-up (correlation score: 0.83-0.98). A moderate concordance was observed from the baseline visit between DAS28-VSG and DAS28-PCR ($k=0.43-0.52$, $p < 0.001$), which decrease to low at 12 months ($k=0.32$, $p < 0.001$). A concordance of 100% was observed at baseline analysing DAS28-VSG and SDAI ($k=1$; $p < 0.001$), but worsened during follow-up being moderate ($k=0.40-0.56$; $p < 0.001$). A very good concordance was observed comparing DAS28-VSG to CDAI at the baseline visit ($k=0.88$, $p < 0.001$), which decreased to good-moderate during follow-up ($k=0.40-0.63$, $p < 0.001$). Between DAS28-PCR and SDAI a moderate concordance

N=45						
Epidemiological data						
Women n (%)	21 (46.7)					
Age (mean ± σ)	74.8 ± 7.5					
Symptoms n (%)						
Shoulder girdle	34 (75.6)					
Pelvic girdle	20 (44.4)					
Peripheral joints	45 (100)					
Oedema	21 (46.7)					
Small joints of hands	42 (93.3)					
Analytical and radiological data n (%)						
FR +	15 (33.3)					
ACPA +	13 (28.9)					
FR and ACPA +	10 (22.2)					
Bone erosions	4 (8.8)					
Treatments n (%)						
Glucocorticoids	43 (95.5)					
DMARDs	40 (91.1)					
	Baseline N = 45	Month 1 N=45	Month 3 N=44	Month 6 N=44	Month 12 N=44	% remission Month 12 N=44
DAS28-VSG (mean ±σ)	6.3 ± 1.0	2.9 ± 1.7	3.0 ± 1.4	2.8 ± 1.3	3.1 ± 1.3	40.9
DAS28-PCR (mean ±σ)	5.7 ± 1.0	2.5 ± 1.4	2.4 ± 1.3	2.2 ± 1.1	2.5 ± 1.0	70.4
SDAI (mean±σ)	41.0 ± 13.8	9.3 ± 11.1	7.9 ± 10.7	6.9 ± 8.7	7.5 ± 8.0	31.8
CDAI (mean±σ)	36.9 ± 11.6	8.7 ± 10.4	7.4 ± 10.5	6.4 ± 8.5	6.8 ± 7.9	31.8
ACR/EULAR remission						18.2

was observed up to 6 months ($k=0.43-0.57$, $p < 0.001$), which worsened to low at 12 months ($k=0.32$, $p < 0.001$). The concordance between DAS28-PCR and CDAI was low-moderate ($k=0.26-0.48$, $p < 0.001$). On the other side, the agreement was very good between SDAI and CDAI throughout the follow-up ($k=0.86-0.93$, $p < 0.001$). The ACR/EULAR index had a very low concordance with DAS28-PCR ($k=0.17$, $p < 0.001$), moderate with DAS28-VSG ($k=0.49$, $p < 0.001$) and the results are good with SDAI and CDAI ($k=0.64$, $p < 0.001$).

Conclusion: The correlation between the different scores was good. However, the concordance between all the scores decreases as time passes, according to a higher percentage of patients in remission or low activity. This low-moderate concordance is demonstrated even between DAS28-PCR and DAS28-VSG, and when these are compared with CDAI and SDAI. CDAI and SDAI are the only ones that maintain a good concordance between them, even when the number of patients in remission or low activity increases. ACR/EULAR only maintains an acceptable concordance with the most restrictive scores, SDAI and CDAI. By contrast, agreement is very low with DAS28, especially by DAS-PCR.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.3453

AB0270

POSITIVE ACPA WITHOUT EVIDENCE OF ARTHRITIS – A RARITY?

Daniela Martins¹, Graça Sequeira². ¹Centro Hospitalar Universitário do Algarve, Faro, Portugal; ²Centro Hospitalar Universitário do Algarve, Rheumatology, Faro, Portugal

Background: Rheumatoid Arthritis (RA) follows a variable disease course in regards to joint damage and functional outcome, therefore an early diagnosis is crucial for its management. However, occasionally, this diagnosis can be a challenge. Rheumatoid Factor (RF) and Anti-citrullinated peptide antibodies (ACPA) are the most useful biomarkers in the diagnosis of RA and, although individually they do not dictate the final diagnosis, they present high specificity and may be positive even before clinical manifestations of the disease occur.

ACPA is detected in approximately two thirds of the patients with RA and presents a superior diagnostic specificity when compared to RF. This specificity of 95-98% may present an increased value if it is three times superior to its value of reference.

Objectives: Share with the medical community and alerting them for the existence of patients with positive ACPA with no clinical evidence of RA.

Methods: By consulting the electronic clinical records of patients observed by a Rheumatologist, we identified and analyzed the patients that presented positive ACPA in their blood tests and no evidence of signs or symptoms of RA, during at least six years of follow-up. Patients with other rheumatic diseases, history of tuberculosis or chronic pulmonary diseases were excluded.

Results: We identified seven patients (average of 61 years old) with positive ACPA in at least two different blood tests, of which five presented a value three times superior to its normal value (<15 U/mL). It should be noted that, four patients presented both positive ACPA and RF. Clinically, all patients referred mechanical symptoms that varied between cervical, low back, shoulder and knee pain and did not present any record of swollen joints without trauma. Radiologically, in most cases we observed joint degenerative damage. Six years after the first positive ACPA, none of the patients developed any manifestation of inflammatory joint disease.

Conclusion: Studies reveal that positive ACPA in healthy patients may precede the clinical diagnosis of RA. The specificity of positive ACPA and RF one and a half years before the diagnosis of RA is approximately 99-100%. However, as we demonstrated, it is not uncommon to find during clinical practice individuals with positive ACPA without evidence of arthritis, which leads us to emphasize the importance of a detailed anamnesis with a meticulous physical examination before establishing a diagnosis based on the patient's blood tests.

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