Methods: We included 246 RA patients who met the ACR / EULAR 2010 rheumatoid arthritis classification criteria. We divided the patients into two groups according to the Charlson comorbidity index (scores 0-2 mild, > 2 moderate and severe) (3). We compared the functional status of the two groups assessed by the health assessment questionnaire disability index (HAQ-DI). In addition, we compared disease-related characteristics between groups. We performed the Mann-Whitney U and chi-square tests where appropriate. Lastly, we also adjusted the age when comparing the continuous variables with ANCOVA.

Results: The patients with moderate and severe co-morbidities were older ($p < 0.001$). All other demographic features were similar between the groups. Extra-articular involvement was more frequent in moderate and severe co-morbidity group ($p = 0.03$). All other disease features and treatment modalities were similar between the groups. Additionally, after adjusting the age, none of the continuous features were different between the groups.

Table 1. Disease and demographic features and treatment characteristics of the patients.

	Mild co-morbidity group (n=180)	Moderate and severe co- morbidity group (n=66)	P	p** (age adjusted)
Age (year)	44.5±9.2	65.0±7.0	P<0.001	N/A
Gender (M/F)	34/146	16/50	0.5	
Smoking (%)	24 (13.3)	7 (10.6)	0.63	
Charlson co-morbidity index	1.38±0.48	3.3±0.6	p<0.001	p<0.001
Disease duration (month)	65.2±77.1	65.1±75.3	0.97	0.23
Swollen joint	0.3±1.5	0.2±0.6	0.92	0.86
Tender joint	0.6±1.6	0.3±1.0	0.38	0.50
ACPA positivity (%)	84 (46.6)	29 (43.9)	0.92	
RF positivity (%)	93 (51.6)	66 (58.3)	0.39	
DAS28-CRP	2.2±1.2	2.1±0.7	0.85	0.37
HAQ-DI	0.16±0.20	0.27±0.39	0.05	0.64
Extra-articular involvement (%)	3 (1.6)	5 (7.5)	0.03	
Joint deformity (%)	21 (11.6)	7 (10.6)	0.77	
Number of received DMARD	1.3±0.5	1.3±0.4	0.55	0.48
Steroid dosage (mg)	1.7±2.4	1.4±1.9	0.73	0.60
Ever receiving bDMARD (%)	21 (11.6)	3 (4.5)	0.09	

M: Male; F: Female; ACPA: Anti-Cyclic Citrullinated Peptide Antibody; RF: Rheumatoid factor; DAS-28: Disease activity score-28; HAQ-DI: Health assessment questionnaire disability index; DMARD: Disease modifying drugs; bDMARD: Biologic disease modifying drugs. **For continuous variables. Continuous variables shown as Mean ± SD. $p < 0.05$ is significant

Conclusion: The only different disease feature was the increased frequency of extra-articular involvement in the more severe co-morbidity group. All other disease characteristics were similar. In addition, the severity of the co-morbidities may not change the doctors' decision to prescribe biological or synthetic DMARDs and steroid dosage.

REFERENCES:

- [1] Dougados M, Soubrier M, Antunez A, et al. Prevalence of comorbidities in rheumatoid arthritis and evaluation of their monitoring: results of an international, cross-sectional study (COMORA). *Ann Rheum Dis* 2014; 73:62-8.
- [2] Luque Ramos A, Redeker I, Hoffmann F, et al. Comorbidities in Patients with Rheumatoid Arthritis and Their Association with Patient-reported Outcomes: Results of Claims Data Linked to Questionnaire Survey. *J Rheumatol* 2019; 46:564-571.
- [3] D'Hoore W, Sicotte C, and Tilquin C. Risk adjustment in outcome assessment: the Charlson comorbidity index. *Methods Inf Med* 1993; 32:382-7.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2903

POS0561 OBESITY PHENOTYPES IN EARLY RHEUMATOID ARTHRITIS

Y. Gorbunova¹, I. Popkova¹, L. Kondrateva¹, M. Cherkasova², E. Nasonov³, A. Lila³. ¹VA Nasonova Research Institute of Rheumatology, Laboratory of Systemic Rheumatic Diseases, Moskva, Russian Federation; ²VA Nasonova Research Institute of Rheumatology, Laboratory of Immunology, Moskva, Russian Federation; ³VA Nasonova Research Institute of Rheumatology, Scientific Department, Moskva, Russian Federation

Background: Patients with rheumatoid arthritis (RA) and obesity have a lower risk of early death than patients with normal weight due to the so-called "obesity paradox". Available publications discuss three phenotypes of obesity: classical obesity, metabolically healthy overweight/obesity, and latent obesity (normal weight associated with metabolic disorders, most importantly - with insulin resistance and adipocytokines imbalance).

Objectives: To clarify the prevalence of obesity phenotypes and to identify factors predisposing to «latent obesity» in early RA without diabetes mellitus or hyperglycemia.

Methods: The study included 35 pts (23 women, 12 men) with early RA (criteria ACR / EULAR, 2010), 56 [43.0; 60.0] years old, naive to treatment with glucocorticoids

and disease-modifying anti-rheumatics, without established diabetes mellitus. Pts were seropositive for IgM RF and anti-CCP, with highly active RA (DAS28 5.9 [5.2; 6.4]; SDAI 35.4 [27.0; 45.8], and CDAI 31.0 [26.0; 44.0]) scores, and median disease duration of 8.0 [6.0; 15.0] months. Electrochemiluminescence assay Elecsys (Roche Diagnostics) was used to measure patients' insulin levels, and ELISA (DBS – Diagnostics Biochem Canada Inc.) - for evaluation of serum leptin concentrations. Insulin resistance (IR) was defined as Homeostasis Model Assessment of Insulin Resistance index (HOMA-IR) ≥ 2.77 . Leptin levels were considered elevated at values ≥ 11.1 ng/ml for women, ≥ 5.6 ng/ml for men. The overweight/obesity status was determined by World Health Organization criteria in pts with body mass index (BMI) ≥ 25 kg/m².

Results: Overweight/obesity were documented in 19 (54, 3%) pts with early RA, 4/19 (21%) had high leptin levels and IR, 11/19 (58%) had isolated hyperleptinemia. Normal BMI was found in 16 (45,7%) pts with early RA, 2/16 (12,5%) had elevated leptin levels and IR, and 6/16 (37,5%) – only hyperleptinemia. Four (21%) out of 19 overweight and 8 (50%) out of 16 pts with normal BMI did not show any metabolic disorders ($p = 0.02$). Classical obesity was found in 15 (43%), and latent obesity – in 8 (23%) pts with early RA. Leptin levels correlated with waist circumference ($r = 0.58$, $p = 0.02$), BMI ($r = 0.71$, $p = 0.0006$), ESR ($r = 0.5$, $p = 0.02$) in the overweight/obese pts, and with waist circumference ($r = 0.59$, $p = 0.03$), IgM RF ($r = 0.58$, $p = 0.03$), triglyceride levels ($r = 0.77$, $p = 0.003$), and atherogenic index ($r = 0.62$, $p = 0.03$) in pts with normal weight.

Conclusion: The classical obesity phenotype associated with abdominal obesity and inflammation was the most common and prevailing over other phenotypes in early RA. Latent obesity was less common and was associated with an unfavorable lipid profile and accumulation of abdominal fat, therefore increasing the risk of cardiovascular diseases in RA. Metabolically healthy overweight/obesity in early RA was really a rare phenotype.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2961

POS0562 PULSE PRESSURE AS AN INDICATOR OF SUBCLINICAL ATHEROSCLEROSIS IN RHEUMATOID ARTHRITIS

S. Lugo-Perez¹, I. J. Colunga-Pedraza², J. R. Azpiri-López¹, D. Á. Galarza-Delgado², N. Guajardo-Jauregui², A. B. Rodríguez-Romero², A. Cárdenas², A. C. Garza Acosta³. ¹Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Cardiology, Monterrey, Mexico; ²Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Rheumatology, Monterrey, Mexico; ³Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Radiology, Monterrey, Mexico

Background: Pulse pressure (PP) is defined as the difference between systolic and diastolic blood pressure and represents arterial compliance and reflective properties of blood flow (1). It is well known that gender, age, and race / ethnicity are intrinsic factors of the patient that influence PP. Brachial PP has recently been associated with markers of subclinical cardiovascular disease after adjustment with traditional cardiovascular risk factors in the general population (2). However, this relationship has not been studied in patients with rheumatoid arthritis (RA) and its identification would allow earlier adjustments of cardiovascular therapies in this high-risk group.

Objectives: The aim of the study is to analyze the difference in PP between patients with RA and healthy controls. Additionally, to analyze the difference between patients with carotid plaque (CP) and without CP.

Methods: A cross-sectional, observational, and comparative study of ninety-two patients with RA aged 40-75 years and who fulfilled ACR/EULAR 2010 classification criteria. Also, we included ninety-two controls without RA, matched by gender, age and comorbidities. A carotid ultrasound was performed in patients with RA and it was divided into two subgroups, with the presence of CP and without CP. A blood pressure measurement was taken after 15 minutes of rest on the right arm of all patients. Distribution was evaluated with the Kolmogorov-Smirnov test. Descriptive analysis was done using measures of central tendency. Chi square, Student's t test and Mann-Whitney U test were used for comparisons between groups. A p value < 0.05 was considered statistically significant.

Results: We found no statistical difference between groups regarding age, gender and, comorbidities (type 2 diabetes mellitus, hypertension, dyslipidemia and, active smoking) (Table 1). There was a significant difference in PP between patients with RA and controls (50 mmHg vs 41 mmHg respectively, $p = 0.032$). Patients with RA had a significant difference in PP of patients with CP and without CP (50 mmHg vs 44 mmHg respectively, $p = 0.008$) (Figure 1). When performing a binary logistic regression, it was found that PP was the only independent factor for the presence of CP in patients with RA, OR 1.054 (95% CI 1.008-1.101, $p = 0.020$).

Conclusion: Patients with RA had a higher PP than controls. Binary logistic regression showed PP as the only independent factor for the presence of sub-clinical atherosclerosis in patients with RA. PP is a parameter that all rheumatologists should consider when evaluating cardiovascular risk in patients with RA.

REFERENCES:

- [1] Winston GJ, Palmas W, Lima J, et al. Pulse pressure and subclinical cardiovascular disease in the multi-ethnic study of atherosclerosis. *Am J Hypertens* 2013;26(5):636-42. doi: 10.1093/ajh/hps092