Methods: From the observational PSARA study, we examined 64 RA patients starting with MTX monotherapy (n=34) or anti-TNF with MTX co-medication (n=30) due to active disease. All patients starting with anti-TNF regimens had been previously unsuccessfully treated with MTX. s-NT-proBNP (ELISA), EndoF (measured by finger plethysmography), and other laboratory and clinical parameters were evaluated at baseline and after 6 weeks and 6 months of treatment.

Results: Median age was 57 years (range 28-79), and 73% were women. 17 (27%) patients had CVD (history of angina, MI, heart surgery, PTA, cerebrovascular disease, thromboembolism, aortic aneurysm, peripheral artery disease). None of the patients had known/symptomatic HF. There were no statistically significant differences between s-NT-proBNP levels at baseline (median 2241 ng/L [IQR 9002]) and after 6 weeks (median 2300 ng/L, [IQR 8960]) and 6 months (median 2358 ng/L [IQR 7772]) of antirheumatic therapy (p=0.992 and p=0.528, respectively). There were no significant differences in the effects of MTX monotherapy and anti-TNF regimens on s-NT-proBNP levels ($p_{\text{baseline-6weeks}}=0.779$; $p_{\text{baseline-6months}}=0.421$).

At baseline, 57 (89%) patients had s-NT-proBNP >125 ng/L, and 44 (69%) had high s-NT-proBNP levels (s-NT-proBNP >450 ng/L in patients <50 years old and >900 ng/L in patients ≥50 years old), and these frequencies did not significantly change with antirheumatic treatment. s-NT-proBNP was not related to EndoF.

Conclusion: A large proportion of RA patients without known HF had elevated s-NT-proBNP levels, which might indicate subclinical impairment of cardiac function. s-NT-proBNP levels were not influenced by six-month MTX and/or anti-TNF treatment. Thus, in contrast to some previous studies, our data does not support the notion that anti-inflammatory treatment protects against HF, and that anti-TNF treatment has negative effect on cardiac function in RA. Nevertheless, definitive conclusions cannot be drawn by our study, e.g. due to limitations of s-NT-proBNP as surrogate marker of HF. Cardiac function in terms of s-NT-proBNP levels was not related to EndoF.

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AB0344

COMPARATIVE ANALYSIS OF SIDE EFFECTS BETWEEN PATIENTS TREATED WITH AND WITHOUT CORTICOSTEROID AT DMARDS-STARTING POINT. SINGLE CENTER RETROSPECTIVE REAL WORLD DATA ANALYSIS

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Background: In the EULAR recommendation on rheumatoid arthritis (RA) treatment, short-term steroid combination should be considered at the start of DMARDs treatment. In this study, we analyzed the present condition of steroid combined use in Japan and the actual situation of side effects in recent years.

Objectives: RA patients who started treatment with DMARDs at our hospital during July 2020 to April 2018,

Methods: A new incidence of hypertension (HTN), diabetes (DIB), dyslipidemia (HLP), infection (INF) were compared between the two groups of steroid combined use group (D_PSL) and noncombined group (D_nonPSL) and analyzed by Cox regression analysis. The cases had there complications before the start of RA treatment were excluded from the analysis.

Results: The number of cases of analysis was 573 (D_PSL = 216, D_nonPSL = 357, average observation period: 5.22±2.70 year), and the new incidence of each complication was INF 50/561 (D_PSL 16.2%vs. D_nonPSL 4.56%[16/351], p=0.000094), HTN 48/527 cases (13.3%[25/188] vs.6.78%[23/339], P = 0.0148), DIB 24/558 (6.16%[13/211] vs. 3.17%[11/347], p = 0.136), HLP 72/542 (15.8%[32/202] vs. 11.8%[40/340], p = 0.198). The incidence of INF was high in D_PSL group in both groups over 65 and under 65 years old (p = 0.00658, p = 0.00267), HTN was high in D_PSL group only in group over 65 years old (p=0.0276, p=0.0554). Average starting amount of steroids was 6.07±10.2 mg, cases of non-withdrawal of steroid at 1, 2, 3 years later is 79.7%, 72.2% and 66.3% respectively.

Conclusion: Combined use of steroid at the start of DMARDs increased the rate of mortality of infection requiring hospitalization and hypertension.

More than half of them were difficult cases of withdrawal of steroids. From the viewpoint of side effect, it is necessary to study the optimization of steroid therapy in the future.

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AB0345

A PILOT ATTEMPT TO USE THE CONSTANT GENETIC MARKERS IN CARDIOVASCULAR RISK STRATIFICATION IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is associated with early atherosclerosis and high mortality. Routine riskometers developed for a common population usually underestimate a true cardiovascular (CV) risk in RA-patients. For early prevention additional criteria is needed to evaluate CV risk in RA-patients. It's known, that early CV accidents in relatives relate to elevated risk. Nevertheless, specific constant genetic markers don't use in risk stratification. Single nucleotide polymorphisms in regulatory regions of genes, participating in immune interaction, can potentially play a role in progression atherosclerosis and used in CV-prevention algorithms.

Objectives: to develop the model for cardiovascular risk stratification in RA-patients including constant genetic markers.

Methods: Two hundred and twelve Caucasian patients with RA (age – 58.0 yrs [48.3; 65.0]; DAS28 4.96 [3.86; 5.85]) were included in our study. Patients had American College of Rheumatology (ACR)-defined RA (1987 classification criteria). All patients gave written informed consent before enrollment. Traditional and “non-traditional” (e.g. RA-associated factors) were analyzed [1]. Carotid atherosclerotic plaques had been found by ultrasonography. Single nucleotide polymorphisms were determined by restriction fragment length polymorphism (TNFA G-308A (rs1800629), TNFA C-863A (rs1800630), TNFA G-238A (rs361525), IL6 G-174C (rs1800795), VEGFA C-2578A (rs699947), MMP3 5-1171 6A (rs3025058), MMP9 -1562T (rs3918242)) and polymerase chain reaction (IL1B T-31C (rs1143627), IL4 C-590T (rs2243250), IL10 C-592A (rs1800872), IL10 A-1082G (rs1800896), VEGFA C+936T (rs3025039), MMP2 C-1306T (rs2438650)). Descriptive statistics, Chi-squared test, logistic regression with Wald statistics were used for data analysis. Results are presented as median and 25th/75th percentiles (Me [25th percentile; 75th percentile]).

Results: Atherosclerotic plaques (API) were found in 59 (27.8%) patients. API revealed were strongly associated with age (66.0 yrs [59.0; 73.0] with API vs 55.0 yrs [42.0; 61.0] without API, p<0.001) and sex (51.6% for man vs 23.8% for woman, p<0.001). Then, a logistic regression was performed to determine which variables analyzed are predictors of a carotid atherosclerotic lesion. Regression results had demonstrated that the model was statistically reliable in distinguishing between patients with API and without [-2 Log Likelihood = 146.53, p<0.01]. The model correctly classified 85.3% of cases. The Wald statistics showed that at least 6 parameters were principal – age (B=0.123, p<0.001), hypertension (B=3.114, p<0.001), smoke (B=2.167, p<0.001), positive rheumatoid factor (B=1.674, p=0.038), body mass index (B=-0.098, 0.019) and recessive allele in TNFA C-863A (B=1.338, p=0.005). Using parameters obtained the ROC-curve was constructed. Area under ROC curve were 0.900 (SE 0.022; 95%CI 0.857-0.942, P<0.001). Sensitivity and specificity calculated by Youden's Index were 79.7% and 87.5%, respectively.

Conclusion: Age, hypertension, smoke, positive rheumatoid factor, low body mass index and recessive allele in TNFA C-863A were associated with carotid atherosclerosis.

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