Medcalc 12.5.0.0 (USA), including standard methods of parametric and nonparametric analysis. Differences were considered significant at $p < 0.05$.

Results: Levels and frequency of occurrence of anti-RA33, anti-CCP, RF IgM and anti-Sa were analysed for the examined patients depending on disease duration. Patients were divided into two groups: patients with early RA (disease duration less than 12 months) and patients with established RA (disease duration over 12 months). In the analysis it was found that the incidence of anti-Sa in established RA are much higher in comparison with the early RA (77.98% vs. 52.00% respectively, $p < 0.05$). The levels of anti-Sa autoantibodies were lower in early RA than in established RA 21,57 U/L; 95% CI 12,10–27,42 vs. 33,10 U/L; 95% CI 27,58–37,71 respectively, $p < 0.05$). Frequency of occurrence of anti-CCP and RF IgM is no different in the early and developed stage of RA. The frequency of occurrence of anti-RA33 in early RA was higher than in established RA (52.00% vs. 32.45% respectively, $p < 0.05$).

Conclusions: Thus, anti-RA33 is a perspective independent biomarker of RA, which has its own potential. Further study of the anti-RA33 as a diagnostic biomarker of early stage RA is promising. Based on preliminary studies the anti-RA33 could have diagnostic and prognostic value in diagnosis and evaluation of patients with early RA, and its differentiation from other small joint disorders, particularly when the other serologic tests are negative.

To clarify the diagnostic and pathogenic value of anti-RA33 further research with a large sample size, comparison of immunological data with genetic factors, the results of other laboratory and instrumental studies, clinical manifestations of the disease are required. The obtained data will allow us to more thoroughly study the pathogenesis of RA and contribute to the search for new therapeutic options.

Disclosure of Interest: None declared

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AB0369 ADULT-ONSET STILL'S DISEASE TREATMENT PREDICTORS AT 1-YEAR FOLLOW-UP IN A SINGLE RHEUMATOLOGIC CENTRE EXPERIENCE

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Background: Adult-onset Still's disease (AOSD) is a rare systemic inflammatory disorder mainly characterised by persistent high spiking fevers, evanescent rash, and joint involvement. Data on the efficacy of biologic therapy in the management of AOSD¹ is increasing and represents a breakthrough in the management of patients with AOSD refractory to corticosteroids (CS) or conventional (c) DMARDs.

Objectives: We aimed to evaluate possible predictor on the need to use a biologic agent in the management of AOSD patients.

Methods: In this retrospective monocentric study we evaluated AOSD patients followed in our outpatient's clinic since 2010 with at least 1 year follow-up. Clinical manifestations, joint involvement, CS and c- or bDMARD use were main outcome measures. T-test or ANOVA and Chi-squared were used to compare continuous or categorical data, when appropriate, and the odds ratio (OR) for which condition had an influence on clinical outcome was calculated.

Results: We evaluated 28 AOSD patients (mean age 43 ± 14 years; median disease duration 3 (95%CI 2.5–17.3) months). All patients at baseline were treated with a median CS of 18.7 (95%CI 14.3–24.8) mg/die prednisone equivalent dose, and median methotrexate (MTX) dose of 15 (95%CI 13.4–16.28) mg/week. After 1 year follow-up, in the 8 patients (28.6%) that needed to start a bDMARD (4 anti-IL1; 3 anti-IL6 and 1 anti-TNF), we observed that baseline joint involvement was the more prevalent manifestation of the disease with higher DAS28 compared to those patients still on CS +MTX (DAS28 3.8 ± 1.2 vs 2.8 ± 1.1 , respectively $p = 0.03$). Moreover, we showed that clinical manifestations of systemic involvement (i.e. fever, rash, organomegaly and anaemia) were associated with a protective risk to start a bDMARD at 1 year (OR 0.4 95% CI 0.2–0.76). Consistently, in those patients with concomitant elevated ESR (> 20 mm/h), CRP (> 15 mg/L) and ferritin (> 250 ng/ml) levels at baseline, we observed a low risk to start a bDMARD at 1 year follow-up (OR 0.4 95% CI 0.18–0.87).

Conclusions: In our cohort about one third of AOSD patients required to be treated with a bDMARD at 1 year follow-up, mainly if joint involvement was the predominant clinical manifestation at disease onset. Systemic manifestations of the disease seem to benefit from high dose CS+MTX combination therapy. Further studies with more extensive cases are necessary.

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AB0370 LOW SERUM IGF1 IS ASSOCIATED WITH HIGHER CARDIOVASCULAR RISK IN THE MIDDLE-AGED WOMEN WITH RHEUMATOID ARTHRITIS INDEPENDENTLY OF THE DISEASE RELATED PARAMETERS

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Objectives: To analyse the relation between serum levels of IGF1 and cardiovascular risk (CVR) in women with rheumatoid arthritis (RA).

Methods: The risk of dying of CV disease within 5 years was calculated using the strategy proposed by Pocock et al. BMJ 2001 in 185 women with RA (mean age 51.7 years) with no previous history of CV events. The CVR and characteristics related to it were analysed with respect to serum IGF1. IGF1 levels below the median of the total cohort were considered low.

Results: The RA women with low IGF1 ($n = 91$, mean 104 pg/ml) had significantly higher CVR compared to those with normal IGF1 ($n = 94$, mean 194 pg/ml) with the predicted risk of 0.51% and 0.17%, respectively ($p > 10^{-5}$). Among the traditional CVR factors, the low IGF1 group was 10 years older (mean 56.8 vs. 46.9 y, $p = 10^{-5}$), lower in height (165 vs. 168 cm, $p = 0.013$, 21% of the patients being < 160 cm) and had high prevalence of hypertension (24% vs. 8.5%, $p = 0.004$), while current smoking was similar between the groups (15% vs. 14%).

The low IGF1 group displayed the unfavourable metabolic profile with higher BMI ($p = 0.002$) and obesity in 22%, higher predicted body fat content (mean 39.5 vs. 35%, $p = 10^{-5}$), and higher total and LDL cholesterol ($p = 0.0014$ and $p = 0.0053$, respectively). The levels of adiponectin ($p = 0.032$) and HDL-cholesterol ($p = 0.25$) tended to be higher in the low IGF1 group, which resulted in the comparable total cholesterol to HDL ratio between the groups. This could also explain that the prevalence of diabetes mellitus and metabolic syndrome were low.

With exception of the disease duration, the groups displayed no significant difference in the RA-related CVR factors such as the disease activity measured by DAS28 (3.29 vs 2.99), systemic inflammation measured by serum IL6 (mean 8.46 vs. 5.99 pg/ml) and IL1 β (mean 19.47 vs. 23.1 ng/ml), and the prevalence of seropositivity (91% vs. 92%). The prevalence of treatment with MTX monotherapy was higher in the low IGF1 group (56% vs. 39%, $p = 0.024$), while the use of TNF-inhibitors and other biologic and synthetic anti-rheumatic drugs was similar within the groups.

Conclusions: Serum IGF1 levels in the normal low range are associated with higher CVR in RA female patients. This increase in CVR seems to be independent of the RA-related characteristics. The combination of low height and hypertension argues for the important role of congenital factors in defining serum IGF1 levels in the studied RA women.

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AB0371 THE EFFECT OF CUMULATIVE EXPOSURE TO CIGARETTE SMOKING ON VASCULAR DAMAGE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Smoking is described as a classic cardiovascular (CV) risk factor and we also know the beneficial effect on the CV system of smoking cessation. However, there is contradictory data about its effect in patients with rheumatoid arthritis (RA). It is possible that the measure of cumulative exposure to tobacco expressed in pack-year gives us more information than the smoking status.

Objectives: To explore the relation between smoking exposure, measured in pack-year, and subclinical vascular damage, mortality and vascular events in patients with RA.

Methods: Observational ambispective study. We included, consecutively, RA patients controlled in a tertiary hospital. We gathered demographic (sex, age, body mass index [BMI]), clinical (characteristics of RA, classic CV risk factors and history of vascular events) and analytical variables (CRP, ESR). We estimated the modified SCORE. We explored the extracranial branches of the carotid artery