Conclusion: Current evidence shows an increased risk of vaccine-preventable infections in patients with AIIRD, emphasizing that prevention of infections is essential in these patients.

REFERENCES:

[1] Falagas ME, Manta KG, Betsi GI, et al. Clin Rheumatol 2007;26(5):663-70.

Disclosure of Interests: Victoria Furer: None declared, Christien Rondaan: None declared, Marloes W. Heijstek: None declared, Ulf Müller-Ladner Grant/research support from: Projekt supported by an unrestricted educational grant from Celgene GmbH., Sander van Assen: None declared, Jacob M. van Laar Grant/research support from: Genentech, Consultant for: F. Hoffmann-La Roche, Marc Bijl: None declared, Ori Elkayam: None declared

DOI: 10.1136/annrheumdis-2019-eular.7706

FRI0659

REMISSION PERSISTENCE IN RHEUMATOID ARTHRITIS, PSORIATIC ARTHRITIS AND AXIAL SPONDYLOARTHRITIS UNDER BIOLOGIC TREATMENT

Pedro Ávila-Ribeiro^{1,2}, Joao Eurico Fonseca^{1,2}, Elsa Vieira-Sousa^{1,2}. ¹Hospital de Santa Maria, CHULN, Centro Académico de Medicina de Lisboa, Portugal, Serviço de Reumatologia e Doenças Ósseas Metabólicas, Lisbon, Portugal; ²Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Portugal, Unidade de Investigação em Reumatologia, Lisbon, Portugal

Background: Patients with rheumatoid arthritis (RA), psoriatic arthritis (PsA) and axial spondyloarthritis (axSpA) under biologic treatment are increasingly achieving prolonged remission and tapering treatment is becoming standard practice. However, the optimal timing to start tapering remains unclear and predictors of loss of remission (LOR) are missing. Guidelines disagree on whether to wait for 6 or 12 months of sustained remission before tapering.

Objectives: To determine whether longer sustained remission (6 versus 12 months) influences subsequent LOR rates and to identify predictors of LOR.

Methods: We used the Rheumatic Diseases Portuguese Register (Reuma.pt), to identify RA, PsA and axSpA patients on stable biologic treatment in a single center and retrospectively analyze those who achieved sustained remission for at least 6 and 12 months. Survival analysis was used to characterize stability of remission and identify predictors of LOR. The Cox proportional hazards regression was stratified by diagnosis and adjusted for age, gender, smoking, baseline disease activity, type of biologic, previous switches and starting year of biologic treatment.

Results: 195 patients (100 RA, 51 PsA and 44 AxSpA) of 1078 patients (785 RA, 116 PsA, 177 axSpA) registered in Reuma.pt at a single center and treated with biologics between 1999 and 2018, had at least one remission period with a minimal duration of 6 months. This corresponded to 310 individual remission periods longer than 6 months, 232 of which (74.8%) were longer than 12 months. Median remission time (since the start of remission period) was 78.6 weeks overall vs. 99.0 weeks for patients with a minimum 12 months remission (difference in median survival: 20.4 weeks). PsA patients showed significantly longer remission periods ($p<0.0001$), followed by axSpA and RA. We identified active smoking (HR 1.96, $p=0.008$ for the total population; HR 1.53, $p=0.20$; HR 7.42, $p=0.01$, HR 0.74, $p=0.79$ for RA, PsA and axSpA, respectively), and infliximab use (HR 2.23, $p=0.005$ for the total population; HR 4.07, $p<0.001$, HR 3.20, $p=0.36$, HR 0.62, $p=0.62$ for RA, PsA and axSpA, respectively; subcutaneous TNF inhibitors (TNFi) used as index category) to be significantly associated with LOR. A sensitivity analysis excluding infliximab patients further suggested female gender (HR 3.21, $p=0.005$) and duration of disease until first biologic (HR 1.05, $p=0.031$) as important co-variables.

a)

b)

Figure 1. – time to loss of remission considering a single flare (a) vs. persistent flare (b), all patients (minimum 150 days in remission). Number of failure events indicated in parenthesis

a)

b)

Figure 2. – time to loss of remission considering a single flare (a) vs. persistent flare (b), only patients with minimum 320 days in remission. Number of failure events indicated in parenthesis.

Figure 3. – time to loss of remission considering a single flare (a) vs. persistent flare (b) by diagnosis, all patients (minimum 150 days in remission).

Conclusion: 6 vs. 12 months of sustained remission did not influence the subsequent rate of LOR. Smoking, the type of TNFi, female gender and duration of disease until biologic treatment were identified as predictors of LOR.

Disclosure of Interests: Pedro Ávila-Ribeiro: None declared, Joao Eurico Fonseca: None declared, Elsa Vieira-Sousa Grant/research support from: MSD, Novartis

DOI: 10.1136/annrheumdis-2019-eular.8034

FRI0660 LONG- AND SHORT-TERM ASSOCIATION OF LOW-GRADE SYSTEMIC INFLAMMATION WITH CARDIOVASCULAR MORTALITY

Jan Leipe¹, Anna-Isabelle Kaelsch², Marcus Kleber², Hubert Schramagl³, Wolfgang Sattler³, Winfried März⁴, Ernst Malle³, Bernhard Kraemer². ¹University Medical Centre Mannheim, Division of Rheumatology, Mannheim, Germany; ²University Medical Centre Mannheim, Department of Medicine V, Mannheim, Germany; ³Medical University of Graz, Graz, Austria; ⁴Synlab Academy, Mannheim, Germany

Background: Evidence suggests a critical role for inflammation in the pathogenesis of coronary artery/heart disease (CAD/CHD), implicating inflammatory molecules as central mediators of chronic inflammatory processes within the vascular wall. In this regard, patients with chronic inflammatory disease such as RA and SLE are at increased risk, which was attributed to the high levels of inflammatory mediators. However, less is known about the association of low-grade systemic inflammation with the cardiovascular risk.

Objectives: The aim of the present study was therefore to evaluate biomarkers representing low-grade systemic inflammation and their association with mortality in a large cohort of patients.

Methods: The Ludwigshafen Risk and Cardiovascular Health (LURIC) study included 3316 consecutive patients undergoing coronary angiography between June 1997 and May 2001 with a median follow-up of 9.9 years. Before coronary angiography fasting blood samples were collected and plasma levels of interleukin-6 (IL-6), C-reactive protein (CRP) and serum amyloid A (SAA) were measured by immunonephelometry. IL-6 and SAA polymorphisms were genotyped.

Results: During a median observation time of 9.9 years 949 deaths (30.3%) occurred, of these 597 (19.2%) died from cardiovascular events. Plasma levels of IL-6, CRP and SAA were associated with unstable CAD

as well as established risk factors including type 2 diabetes, smoking, lower eGFR, lower triglycerides and lower HDL-C. In addition, IL-6 correlated with age, use of lipid lowering therapy and hypertension, SAA correlated with female gender and hypertension, and CRP correlated with female gender, lipid-lowering therapy and BMI. After adjustment for established cardiovascular risk markers and the other two inflammatory markers, SAA was found an independent risk factor for cardiovascular mortality after a short-term follow-up (1 year) with a HR of 1.41 (1.03-1.93, $p=0.030$), whereas IL-6 was identified as an independent risk factor for long-term follow-up (3, 5-9.9 years) with HRs of 1.21 (1.02-1.43, $p=0.006$), 1.22 (1.06-1.40, $p=0.006$) and 1.18 (1.07-1.31, $p=0.001$), respectively. Although 6 SNPs in the SAA gene were significantly associated with SAA plasma concentrations (none of the IL-6 SNPs associated with IL-6 levels), the genetic risk score was not associated with mortality.

Conclusion: In our large, long-term LURIC cohort study we demonstrate for the first time prospectively that plasma levels of IL-6 and SAA are not only associated with cardiovascular risk factors and the prevalence of CAD but also independently predicted cardiovascular mortality. These findings underline the importance of low-grade systemic inflammation for the cardiovascular risk and the prognostic relevance of inflammatory biomarkers, independently predicting cardiovascular mortality. Although it was not the focus of this study, our findings might suggest that even low-grade inflammation is unfavourable in chronic rheumatic disease.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.7919

FRI0661 ASSOCIATIONS OF CURRENT AND CHILDHOOD SOCIOECONOMIC STATUS WITH PATIENT-REPORTED HEALTH OUTCOMES AMONG PATIENTS WITH KNEE OR HIP OSTEOARTHRITIS IN A FAMILY PRACTICE SETTING IN MEXICO CITY

Julio Pisanty Alatorre¹, Maria Victoria Goycochea Robles². ¹Instituto Mexicano del Seguro Social, HGZMF 8, Mexico, Mexico; ²Instituto Mexicano del Seguro Social, Unidad de Investigación en Epidemiología Clínica HGR1, Mexico, Mexico

Background: The life-course approach is a useful framework to understanding socioeconomic inequalities in the prevalence and outcomes of chronic disease,¹ and in particular in osteoarthritis (OA).² Previous studies have measured Socioeconomic Status (SES) in 2 points of the life-course (childhood and current) and found consistent relationships between them and both prevalence and presence of worse outcomes,²⁻⁴ but this relationship has been little explored in the Latin-American region where exposure to low SES throughout the life-course is higher than in developed countries.

Objectives: Our aim was to investigate the association of current and childhood SES with functional status, quality of life and disability among patients with knee or hip OA.

Methods: We recruited patients with self-reported doctor-diagnosed knee or hip OA in a social security family practice setting, excluding patients with other rheumatic diseases liable to causing secondary OA. We measured current SES by education, occupation type and estimated income using a validated tool;⁵ and childhood SES by parental education and occupation type. We measured functional status, quality of life and disability using Spanish-language versions of the WOMAC,⁶ AMICAL⁷ and HAQ-DI⁸ tools. We used Spearman correlations, ANOVA and multiple linear regression to test for significance, with a $p<0.05$ cutpoint.

Results: We recruited 146 patients with a mean age of 69.4 yr. 80% were female; 92%, 30% and 45% suffered from knee, hip and other joint OA, respectively, while 60% had incomes in the upper 2 quintiles of the general Mexican population. We found current income to be the best predictor of WOMAC (Spearman's $\rho=-0.354$, $p<0.01$), AMICAL ($\rho=-0.362$, $p<0.01$) and HAQ-DI scores ($\rho=-0.335$, $p<0.01$), and education to be correlated with all three scores as well. Occupation type was significantly associated with AMICAL and HAQ-DI but not WOMAC scores. Paternal and maternal education, and maternal occupation type, were significantly associated only with AMICAL scores.

In multiple linear regression models were income and maternal education were entered separately, after adjusting for age, sex, BMI and presence of Diabetes and Hypertension, both variables were significantly associated with WOMAC, AMICAL and HAQ-DI scores (adj r^2 0.076-0.157, $p<0.05$). When both variables were entered into multiple linear regression models (adj r^2 0.112 and 0.110), significant associations remained between current income level and WOMAC and AMICAL scores ($p<0.01$), as well as between maternal education and AMICAL scores ($p<0.05$). Associations between both variables and HAQ-DI scores did not reach statistical significance ($p=0.07$).