obesity was defined by WHO criteria and the hand grip strength was evaluated by dynamometry. Sarcopenia was defined according to the European Working Group on Sarcopenia in Older People, as Skeletal Muscle Mass Index less than 5.45 kg/m² in women and less than 7.26 kg/m² in men, while sarcopenic obesity was defined by the presence of sarcopenia more abdominal obesity. The GCs use and dosage (prednisone and methylprednisolone) were analyzed reviewing the clinical records. Anti-cyclic citrullinated peptides (anti-CCP) antibodies, rheumatoid factor (RF) levels, high sensitivity C reactive protein (hsCRP), erythrocyte sedimentation rate (ESR) were determined. The morning stiffness, clinical activity of disease (DAS28-ESR score) and disability index functional (HAQ-DI) were measurement.

**Results:** In this study 86% of the population had sarcopenia and 44% sarcopenic obesity. The 62.8% under GCs therapy. The prednisone dosage was positively associated to sarcopenia (≥ 5 mg/day; OR: 4.3, p=0.003) and sarcopenic obesity (OR: 3.2, p=0.06). The intramuscular pulse of methylprednisolone (40 mg/kg) was associated to sarcopenia obesity phenotype (OR: 2.6, p=0.09). Regarding the clinical and serological markers in RA, high disease activity (DAS28-ESR score) was associated to sarcopenia (OR: 6.6, p=0.01) and sarcopenic obesity (OR: 6.3, p=0.02). The morning stiffness (p=0.03), RF (p=0.05), anti-CCP positive (p=0.03) and HAQ-DI score (p=0.04) were too associated, mainly to sarcopenic obesity.

**Conclusion:** Sarcopenia and sarcopenic obesity are associated to GCs dosage and with serological and disease activity markers in RA patients from southern Morocco. So that is needed promote monitoring and management of sarcopenia and sarcopenic obesity in RA patients.

**Reference:**


**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2019-eular.7872

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**Cognitive Impairment and Rheumatoid Arthritis in Moroccan Patients**

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**Background:** Cognition in rheumatoid arthritis is regaining interest in publications lately. A recent experimental study(1), have shown that low grade chronic periodontal infection by Porphyromonas gingivalis result in the development of neuropathology that is consistent with that of Alzheimer disease in adult mice. This same pathogen has been linked previously in many studies to the development of RA. These findings are raising existing doubt about an association between cognitive impairment and RA.

**Objectives:** Our study aims to assess cognitive impairment in RA patients in remission state.

**Methods:** It is a cross-sectional study realized at the rheumatology department of our University hospital. Our established RA patients regularly followed were asked to fulfill the Arabic version of the mini mental state examination (A-MMSE)(2) twice (initially and 15 days later) and the Patient Health Questionnaire (PHQ-9)(3) test for depression. Patients with neurological history (stroke, chronic infection and hematoma) have been excluded. Data about demographic, clinical (body mass index, Disease Activity Score of 28 joints, Health Assessment Questionnaire, modified total sharp score), biological (C-Reactive Protein, blood platelet count, Anti-Citrullinated Peptide Antibodies, rheotropin, vitamin B12, folic acid) characteristics were recorded. A-MMSE under 24 after adjustment for the level of education as recommended was considered abnormal. A cut-off of 10 of the PHQ-9 was considered as suggestive of depression. A descriptive study and regressions were done by SPSS 20 software and p<0.05 was considered as significant.

**Results:** A total of 80 patients were recruited, with an average age of 55.9 ± 8.5 years. Women were predominant (86.3%) in this population with an illiteracy rate of 66.3%. Our patients were ACPA positive in 55.9 ± 8.5 years. Women were predominant (86.3%) in this population.

**Conclusion:** It is certainly true that the interpretation and evaluation of cognitive functions is a difficult task, especially in an illiterate and disabled patient. However, in our study A-MMSE results were low even after test repetition, and in a state of disease remission. Although these results underscore the high frequency of cognitive impairment in RA patients, we estimate that further studies are eligible to confirm causality.

**References:**


**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2019-eular.7162
DURATION OF EXPOSURE TO BIOLOGICS AND STATINS MODERATES THE EFFECTS OF CUMULATIVE INFLAMMATION ON CORONARY ATHEROSCLEROSIS PROGRESSION IN RHEUMATOID ARTHRITIS

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Background: Higher inflammatory load associates with increased risk of cardiovascular events in patients with rheumatoid arthritis (RA). We recently reported that occult atherosclerosis burden on coronary computed tomography angiography (CCTA) predicted long-term cardiovascular events (CVE) in RA above and beyond cardiac risk factors or scores. We further showed that higher cumulative inflammatory burden independently predicted coronary plaque progression.

Objectives: To explore whether the duration of exposure to biologic DMARDs and/or statins during the study period mitigates the effect of cumulative inflammatory load on coronary plaque progression.

Methods: One hundred-one participants with a baseline CCTA underwent a follow-up evaluation in 83±3.6 months. Plaque burden was reported as segment involvement score (SIS, describing the number of coronary artery segments with plaque per patient) and coronary artery calcium (CAC), quantified by the Agatston method. Robust logistic and linear regression models evaluated associations of predictors on plaque (SIS) progression and CAC change, respectively. Predictors of interest were time-averaged c-reactive protein (CRP), duration of bDMARD exposure (years), duration of statin exposure (years), and their 2- and 3-way interactions. Models were controlled for age and baseline hypertension. Significant interactions were subsequently decomposed and examined based on a median split for the duration of biologic and statin exposure.

Results: A significant interaction between inflammation and statin exposure duration was observed for plaque progression ($p=0.019$) (figure 1A); in patients with shorter statin exposure (<1 year), higher inflammation predicted a greater likelihood of plaque progression ($OR=3.25, 95\% CI=1.02$ to 10.17, $p=0.012$). By contrast, longer statin exposure (>1 year) attenuates that risk. Higher inflammatory load associates with increased risk of treatment with bDMARDs, statins and their ultimate impact on coronary plaque progression in RA are highly nuanced. The effect of the duration of statin exposure on coronary plaque burden progression seems relevant only in the context of insufficient biologic exposure; shorter treatment with statins in that setting allows for significant coronary plaque progression in response to higher cumulative inflammatory load. In contrast, longer statin exposure attenuates that risk.

Conclusion: The relationship between cumulative inflammation, length of treatment with bDMARDs, statins and their ultimate impact on coronary plaque progression in RA are highly nuanced. The effect of the duration of statin exposure on coronary plaque burden progression seems relevant only in the context of insufficient biologic exposure; shorter treatment with statins in that setting allows for significant coronary plaque progression in response to higher cumulative inflammatory load. In contrast, longer statin exposure attenuates that risk.