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 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=0.002) and sarcopenic obesity (OR=3.2, p=0.06). The intramuscular pulse of methylprednisolone (40 mg/kg) was associated to sarcopenia obesity phenotype (OR=2.61, 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=0.04) 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 Mexico. So that is needed promote monitoring and management of sarcopenia and sarcopenic obesity in RA patients.

**REFERENCE:**


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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 neuropathy that is consistent with that of Alzheimer dementia 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 hemotoma) have been excluded. Data about demographic, clinical (body mass index, Disease Activity Score of 28 joints, Health Assessment Questionnaire, modified total sharp scoring, biological (C-Reactive Protein, blood pressure, heart rate), Anti-Citrullinated Peptide Antibodies, threoprotein, vitamin B12, folic acid) charactersitics 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 taken 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 93.8% of the cases, with mean disease duration of 11.4±6.5 years. They were receiving dCDA/MARDs in 96.3%. Their average DAS28CRP was 2.19 ± 0.92, and their mean HAQ was 1.14±0.89. Five patients had thyroid disease and were receiving treatment, 20% of the population had anemia but with correct vitamin B dosage (essentially iron deficiency). The A-MMSE was abnormal in 57% at the first time, and in 38.8% of the cases at the second try fifteen days later, while 27.5% of the patients had a PHQ9>10. The fields of altered A-MMSE were: attention and calculation (46.3%), copying (53.8%), orientation (38.8%), recall (25%), and language (41.3%). The immediate recall part was little concerned. No association was found for low MMSE in regressions with age, gender, menopause, BMI, disease parameters (HAQ, DAS, CRP, ACPA, tMSS) or other possible influencing factors (uric acid, creatinim clearance). An elevated PHQ was not correlated to A-MMSE in our patients (P=0.22).

Only their level of education was significantly related to A-MMSE (P=0.001).

**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:**


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**CARDIOVASCULAR RISK IN THE RHEUMATOID ARTHRITIS PATIENTS OF THE GULF CORPORATION GUC-C: WHAT CONTRIBUTE TO THE CARDIOTID INTIMA MEDIA THICKNESS**

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**Background:** Rheumatoid arthritis (RA) is a common inflammatory joints disease that occurs in 1-3% of population. RA patients are at higher risk of cardiovascular disease (CVD). This accelerated atherosclerosis can’t be fully explained by the traditional CVD risk factors. The CVD risk factors had never been investigated in the RA patients of Gulf Corporation Council (GCC).

**Objectives:** For the first time, this study assesses the CVD as manifest by carotid intima media thickness (cIMT) and the CVD risk factors (traditional and non-traditional) in RA GCC-population.

**Methods:** 216 RA (179 (83%) F and 37 (17%) M) GCC patients, who were free of atherosclerosis (CVD & Cerebrovascular diseases) included over 5 years (2013-2018). Diabetic, hypertensive, gout, renal and thyroid patients, pregnant, current smokers and those with history of smoking, and patients on diuretics medications were excluded. cIMT ultrasound (US) measurements were obtained using a real-time US scanner equipped with a 7.5-MHz linear probe. Blood tests (full blood counts, liver function, renal profile, and inflammatory markers), demography details, and body mass index (BMI) had been obtained within the same week of the cIMT scan. The correlation between cIMT and other variables were calculated using simple linear and multivariate regression analysis.

**Results:** The mean cIMT was 0.58 ± 0.11 mm (Min 0.28, Max 0.98). The mean age was 48 ± 13 years (48 ±12 yrs for females, 50±16 yrs for males, p= 0.279).

Univariate regression analysis showed a positive linear relationship between cIMT and age of the participants (p=0.001, Cl 0.00, 0.01), hemoglobin (Hb) (p=0.006, Cl: 0.004, 0.023), hematocrit (p=0.006, Cl: 0.001, 0.008), mean cell volume (MCV) (p=0.027, 0.000, 0.004), mean cell hemoglobin (p=0.04, Cl: 0.000, 0.009), platelet (p=0.000, 0.001, 0.000), monocytes (p=0.02, Cl: 0.001, 0.018), eosinophils (p=0.011, Cl: 0.002, 0.018), erythrocyte sedimentation rate (ESR) (p=0.04, Cl: 0.000, 0.001), creatinine (p=0.002, Cl: 0.000, 0.002), uric acid (p=0.002, Cl: 0.001, 0.004), triglycerides (p=0.033, Cl: 0.002, 0.044), low density lipoprotein (LDL) (p=0.002, Cl: 0.010, 0.045), C-reactive protein (p=0.000, Cl: 0.001, 0.002), ferritin (p=0.000, Cl: 0.000, 0.001), body weight (kg) (p=0.018, Cl: 0.000, 0.002), body mass index (Kg²/Ht²) (p=0.026, 0.000).
Conclusion: For the first time we show that high hemoglobin, transferrin level and eosinophils count seem to have a role as a CVD risk factors in RA. The glucose fasting level is important CVD risk factor even in non-diabetic RA patients. The rest of the analyzed CVD risk factors were not related to RA CVD risk in RA GCC population like what had been demonstrated in other population (older age, high Sbp, ESR, LDL, and GFR level).

REFERENCE:

Disclosure of Interests: None declared


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 [odds ratio (OR)=1.90, 95% confidence interval (CI)=1.15 to 3.14, p=0.012]. By contrast, longer statin exposure (>1 year) attenuated that effect [OR=1.37, 95% CI=0.67 to 2.82, p=0.391]. Notably, significant three-way interactions were observed between time-averaged CRP, duration of bDMARD exposure and duration of statin exposure on plaque progression [p=0.050] and CAC change [p=0.006]. In patients with shorter biologic exposure (<5 years), longer exposure moderated the effect of cumulative inflammation on plaque and CAC change. Specifically, cumulative inflammation positively associated with plaque progression [OR=3.25, 95% CI=1.02 to 10.38, p=0.047] and CAC change [OR=0.57, 95% CI=0.30 to 0.88, p=0.001] in patients with shorter treatment with statins (<1 year) [figure 1B and 1C, respectively]. By contrast, in patients with longer exposure to statins (>1 year), inflammation was not related to plaque progression [OR=1.25, 95% CI=0.43 to 3.31, p=0.63] or CAC change [p=0.18, 95% CI=0.43 to 0.21, p=0.52]. In patients with longer biologic exposure (>5 years) the length of treatment with statin did not moderate the effect of cumulative inflammation on plaque progression [p=0.229, figure 1].

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.

Figure 1. Interaction between cumulative inflammation, length of treatment with bDMARDs and statins on coronary plaque progression in RA.

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BODY COMPOSITION IN MEN WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a chronic immune inflammation of the joints, leading to early disability of patients at high risk of cardiovascular events and osteoporotic fractures. This problem is important today in men with RA, because, due to more frequent severe disease and increased mortality in the year after the fracture. Reduction of bone mineral density (BMD) and muscle mass are significant predictors of fractures, which leads to the high importance of studying the state of BMD and body composition.

Objectives: Improving the diagnosis of osteoporosis in patients with RA of the male gender with the regard of BMD and body composition.

Methods: The study involved 110 male patients with a documented diagnosis of RA at the age of 59 [53; 65] years. Depending on the taking of glucocorticosteroids (GCS) all patients was allocated two groups: I subgroup - 60 patients who are not taking GCS and subgroup II - 50 patients who are taking GCS. The control group consisted of 30 healthy men comparable by the age and body mass index.

The study of BMD at the lumbar spine (L1-L4) and femoral bone was measured by dual-energy x-ray absorptiometry using a bone mineral density «STRATOS dX» (OMS, France). Assessment of body composition was carried out, using the «Whole body» (=The whole body»). Sarcopenia was diagnosed as a decrease in lean mass index of less than 7.26 kg/m².

Results: 63.6% of patients with RA showed a reduction of BMD corresponding osteopenia/OP. OP was diagnosed in 28 (25.5%) patients with RA, and osteopenia in 42 (38.2%). The detection rate of OP in the subgroup was significantly higher (p=0.05), than in the first subgroup (48% and 5% respectively). The most significant decrease in BMD was observed in the neck of the femur in the main group as a whole and in individual subgroups. There was a negative correlation degree of activity of RA and indicators of BMD of the lumbar spine (r=-0.4, p<0.05) and proximal femur (r=-0.38, p<0.05). Assessment of body composition showed that patients of the main group were significant decrease in the total lean mass (LM) of the body, and the trunk and limbs of LM compared with patients of the control group (p<0.05). Sarcopenia detected in 66 (60%) of RA patients, whereas in the control group it was absent. In 44 (66.7%) male patients with RA with sarcopenia decreased BMD to the level of osteopenia (34.9%) and OP (31.8%). Obtained a negative