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 CVD risk factors in RA GCC population are like what had been demonstrated in other population (older age, high Hb, ESR, LDL, and GFR level).

REFERENCE:

Disclosure of Interests: None declared

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 longitudinal 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 the effect of SIS, plasma levels 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) but not for CAC progression (p=0.391). 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 10.38, p=0.047) and CAC change [Δ=0.57, 95% CI 0.05 to 1.08, p<0.001] in patients with shorter treatment with statins (<1 year) [figure 1B and 1C respectively]. 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 [Δ=0.57, 95% CI 0.05 to 1.08, 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 0.93 to 3.31, p=0.18] or CAC change [Δ=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.

Disclosure of Interests: George Karpouzas: Grant/research support from: Pfizer, Consultant for: Sanofi-Genzyme-Regeneron, Janssen, Roche-Genentech, Pfizer, Speakers bureau: BMS, Sanofi-Genzyme-Regeneron, Janssen, Roche-Genentech, Sarah Ormseth: None declared, Elizabeth Hernandez: None declared, Matthew Budoff: None declared

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 DPA» (DMS, 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 osteoporosis/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 significantly 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