Table 1. Multiple regression models

<table>
<thead>
<tr>
<th>Model</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 865</td>
<td>n = 1, 251</td>
</tr>
<tr>
<td>ERS-RA score</td>
<td>Coefficient 95% CI, p</td>
<td>Coefficient 95% CI, p</td>
</tr>
<tr>
<td>hs-CRP every 10 mg/L</td>
<td>0.005</td>
<td>0.000 to 0.100</td>
</tr>
<tr>
<td>bDMARD use</td>
<td>-0.002</td>
<td>-0.005 to 0.001,</td>
</tr>
<tr>
<td>csDMARD use</td>
<td>0.199</td>
<td>0.199, 0.722</td>
</tr>
<tr>
<td>Prob &gt;F, model with only 0.03</td>
<td>CRP</td>
<td>0.03</td>
</tr>
<tr>
<td>Prob &gt;F, full model</td>
<td>0.07</td>
<td>0.08</td>
</tr>
</tbody>
</table>

A multiple linear regression (ENTER method) was performed for the dependent variable ERS-RA score using a listwise deletion analysis (Model 1) and a multiple imputation analysis (Model 2).

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2021-eular.2643

POS0215 DIRECT AND CONDITIONAL EFFECTS OF EPICARDIAL ADIPOSE TISSUE VOLUME ON CORONARY PLAQUE PROGRESSION IN RHEUMATOID ARTHRITIS

G. Karpouzas1, S. Ormseth1, E. Hernandez2, M. Budoff3, The Lundquist Institute, Rheumatology, Torrance, United States of America; The Lundquist Institute, Cardiology, Torrance, United States of America

Background: Epicardial adipose tissue volume (EATv) predicts coronary atherosclerosis presence, progression and cardiovascular event risk in general patients1,2. Our group recently reported that EATv associated with greater sub-erosclerosis presence, progression and cardiovascular event risk in general patients.3 In rheumatoid arthritis (RA), the relationship was stronger in RA patients with lower disease duration, no traditional cardiac risk factors, and who were not obese.3

Objectives: To evaluate the predictive value of EATv on long-term coronary atherosclerosis development, and moderators of the association between EATv and plaque formation.

Methods: This single-center observational cohort study included 100 patients without symptoms or diagnosis of cardiovascular disease who underwent computed tomography angiography for evaluation of EATv and coronary atherosclerosis at baseline and repeat assessments 6.9±0.3 years later to evaluate plaque progression. New plaque formation in segments without plaque at baseline was the main outcome. Robust multivariable logistic regression evaluated the effect of high versus low EATv (based on median) on likelihood of new plaque formation, accounting for clustering of segments within patients. Potential moderator effects of prespecified predictors were also assessed.

Results: High EATv (>107 cm³) predicted new plaque formation in segments without baseline plaque (OR 2.77 [95% CI 1.43-5.37], p= 0.003; however, significance was lost in the multivariable model. Importantly, high EATv associated with formation of higher-risk non- and partially calcified plaque after adjusting for Framingham DAgostino risk score, obesity, segment location, time-averaged CRP, duration of bDMARD and statin treatment and cumulative prednisone dose (adjusted OR 2.57 [95% CI 1.02-6.48], p= 0.045). RA duration (<10 versus >10 years), cardiac risk factor burden (≥1 versus >1), presence of mixed/calcified plaque in other coronary segments at baseline, and statin exposure (≤1 versus >1 year, based on median) moderated the effect of EATv on all new plaque formation (all p for interaction ≤ 0.021). Specifically, high EATv predicted new plaque formation in patients with RA duration <10 years (adjusted OR 5.75 [95% CI 1.77-18.67]), those with ≥1 cardiac risk factors (adjusted OR 3.40 [95% CI 1.46-7.90]), those without calcification at baseline (adjusted OR 2.65 [95% CI 1.11-6.31]) and those with statin treatment <1 year (adjusted OR 3.33 [95% CI 1.13-9.77]). This was not the case for patients with RA >10 years, ≥ 2 cardiac risk factors, calcification at baseline and statin treatment >1 year (figure 1).

Conclusion: High baseline EATv independently predicted future higher-risk non-calcified and mixed coronary plaque in RA. Moreover, it conditionally promoted new plaque formation overall in patients with earlier disease, low cardiac risk factor burden, who had little or no atherosclerosis at baseline and who had limited exposure to statin therapy. These findings indicate the need for a larger prospective evaluation of the role of EATv as a biomarker of coronary atherosclerosis development in RA.

REFERENCES:

had no effect on new total plaque formation (adjusted odds ratio-OR 0.88 [95% CI 0.64-1.21]). However, each 1-SD increase in time-averaged HDL-C associated with a 44% reduced likelihood of new non-calciﬁed plaque formation at follow-up (adjusted OR 0.56 [95% CI 0.35-0.92], Figure 1). In contrast, there was no effect of time-averaged HDL-C on new mixed or calciﬁed plaque formation. Of 98 non-calciﬁed plaques at baseline, 42 did not change at follow-up, 32 regressed (disappeared), 16 transitioned to mixed and 8 to calciﬁed plaques. Each SD increase in time-averaged HDL-C yielded a 2.2-fold greater likelihood of non-calciﬁed plaque regression (adjusted OR 2.21 [95% CI 1.02-4.83]). Sixteen of 52 mixed plaques present at baseline transitioned to more stable calciﬁed lesions, and time-averaged HDL-C (per 1-SD increment) predicted a 3.5-fold increased likelihood of transition of mixed to fully calciﬁed plaque (adjusted OR 3.56 [95% CI 1.25-10.17]).

Conclusion: Higher HDL-C over time predicted regression of existing and decreased formation of new higher-risk non-calciﬁed plaque. It also associated with transition of vulnerable mixed plaque to more stable fully calciﬁed plaque. These effects were independent of RA treatment duration, prednisone dose and statin exposure.

REFERENCES:

Impact of HDL-C over time on coronary plaque progression in RA

Disclosure of Interests: George Karpouzas Speakers bureau: Sanofi/Gene-

zyme/Regeneron, Consultant of: Sanofi/Genezyme/Regeneron, Grant/research

support from: Pfizer, Sarah Ormseth: None declared, Elizabeth Hernandez:

zyme/Regeneron, Consultant of: Sanofi/Genzyme/Regeneron, Grant/research

support from: Pfizer, Sarah Ormseth: None declared, Elizabeth Hernandez:

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3889

Impact of HDL-C over time on coronary plaque progression in RA

Disclosure of Interests: George Karpouzas Speakers bureau: Sanofi/Gene-

zyme/Regeneron, Consultant of: Sanofi/Genezyme/Regeneron, Grant/research

support from: Pfizer, Sarah Ormseth: None declared, Elizabeth Hernandez:

zyme/Regeneron, Consultant of: Sanofi/Genzyme/Regeneron, Grant/research

support from: Pfizer, Sarah Ormseth: None declared, Elizabeth Hernandez:

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3889

PULSE WAVE VELOCITY ELEVATION ABOVE
INDIVIDUAL REFERENCE VALUES AND AORTIC-
BRACHIAL STIFFNESS MISMATCH AS EARLY
MARKERS OF ARTERIAL STIFFNESS INCREASE IN
PATIENTS WITH RHEUMATOID ARTHRITIS

E. Troutskaya1, S. Velmakin1, R. Osypants1, A. Arbusova1, V. R. Espinoza2,
Z. Kobalava1, 1Peoples’ Friendship University of Russia (RUDN University),
Internal diseases, Moscow, Russian Federation; 2Peoples’ Friendship University
of Russia (RUDN University), Int, Moscow, Russian Federation

Background: Arterial stiffness (AS) is a known predictor of cardiovascular (CV)
disease. The measurement of pulse wave velocity (PWV) is considered to be a
good standard of AS assessment but the recommended threshold of 10 m/s1 may
not take into account multiple factors influencing PWV. Use of the proposed individual
reference values may help to identify patients with AS increase despite PWV level
below this threshold2. The impact of AS on CV outcomes may be mediated by the
reversal of the aortic-brachial stiffness (AS gradient)3. One small study in patients
with type 2 diabetes has shown that the aortic-brachial stiffness mismatch (hereaf-

ter AS mismatch) was an earlier marker of AS compared to PWV elevation4. Patients
with rheumatoid arthritis (RA) have high CV risk and may beneﬁt from early detection of
AS increase. Both approaches have not been studied in RA previously.

Objectives: To evaluate the incidence of PWV elevation above individual reference
values and the frequency of AS mismatch in RA

Methods: Study group included 85 patients (pts) with RA (females 77.6%, aged
59.7±14.3 years, HTN 65%, mean DAS-28(CRP) 3.7±1.1) and control group (40
pts matched by gender, age and risk factors). Parameters of AS were measured
by applanation tonometry. Individual PWV reference values were assessed6. The
AS gradient was calculated as carotid-femoral (cf)PWV/carotid-brachial (cr)PWV
ratio and its elevation ≥1 was considered as AS mismatch. p<0.05 was consid-
ered signiﬁcant.

Results: In pts with RA with and without history of HTN mean cfPWV was
10.3±3.1 and 7.3±1.5 m/s, respectively, mean AS gradient – 1.4±0.4 and
1.1±0.1 (p<0.001 for trend); in controls – 9.6±1.9 and 6.7±1.4 m/s and 1.3±0.3
and 0.99±0.2, respectively (p<0.001 for trend). cfPWV elevation ≥10 m/s was
observed in 31.4% pts with RA and 32.5% of controls: 6.7 and 6.3% of non-
motensives and 40.6% and 46.3%, respectively (p<0.05) in controls. AS gradient
above individual reference values was observed in 41.2% RA pts and
75.2% of controls (p=0.03): in 40% and 63% of normotensives (p=0.02) and
41.8% and 47.7% of hypertensives, respectively. After adjustment by age, gen-
der and systolic BP cfPWV elevation above individual reference values in
normotensive RA pts was independently associated with BMI (beta=0.39, p<0.02)
and dyslipidemia (beta=0.48, p=0.01). The frequency of AS mismatch in RA was
signiﬁcantly higher compared to the controls in both normotensive and hyper-
tensive subgroups: 76.7% vs 43.8% (p=0.03) and 94.5% vs 79.2% (p=0.04),
respectively. The same trend was observed in a subgroup with normal cfPWV:
AS mismatch was present in RA and controls in 82.1% vs 51.9% (p<0.004) in pts
with PWV ≤ 10 m/s and in 82% and 51.7% (p=0.04), respectively in pts with PWV
below individual reference values.

Conclusion: Patients with RA are characterized by higher frequency of cfPWV
l......