Table 1. Multiple regression models

<table>
<thead>
<tr>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>n= 865</td>
<td>n= 2, 251</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ERS-RA score</th>
<th>Coefficient 95% CI, p</th>
<th>Coefficient 95% CI, p</th>
</tr>
</thead>
<tbody>
<tr>
<td>hs-CRP every 10 mg/L increment</td>
<td>0.005  (0.000 to 0.100, 0.043, 0.005) 0.000 to 0.011, 0.005</td>
<td>0.000 to 0.011, 0.005</td>
</tr>
<tr>
<td>hsCRP every 10 mg/L increment</td>
<td>-0.002 (-0.005 to 0.001) -0.000</td>
<td>-0.002 to 0.002</td>
</tr>
<tr>
<td>CRP</td>
<td>0.199</td>
<td>0.963</td>
</tr>
<tr>
<td>CRP</td>
<td>0.002 (-0.003 to 0.007, 0.394002) -0.002 to 0.006, 0.371</td>
<td></td>
</tr>
<tr>
<td>CRP</td>
<td>0.07</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Prob >F, model with only 0.03 0.03

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

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POS0215

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

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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 suberosclerosis presence, progression and cardiovascular event risk in general patients.

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 1.79 [95% CI 1.03-3.11], p= 0.035).

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 cardiovascular 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:


POS0216

GREATER HIGH-DENSITY LIPOPROTEIN LEVELS OVER TIME ARE LINKED TO DECREASED CORONARY PLAQUE FORMATION AND PROGRESSION AND STABILIZATION OF HIGH-RISK LESIONS IN RHEUMATOID ARTHRITIS

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Background: The relationship between serum lipoproteins and cardiovascular disease risk in rheumatoid arthritis (RA) is complex1. Their levels and function may vary based on disease activity and medication use. Beneficial effects on high-density lipoprotein (HDL-C) levels, structure and behavior, in response to treatment have been described. However, the impact of HDL-C levels over time on coronary atherosclerosis progression in RA is unknown.

Methods: One hundred one RA patients without symptoms or history of cardiovascular disease who participated in a computed tomography angiography study of coronary atherosclerosis had repeat assessments after 6.9±0.3 years to evaluate plaque progression. Clinical, laboratory and medication data were recorded at baseline and regular outpatient follow-up visits thereafter. Time-averaged HDL-C was calculated for each patient using available consecutive HDL measurements between baseline and follow-up. Robust logistic regression assessed the association between time-averaged HDL-C and likelihood of new plaque formation in segments without plaque at baseline, and transition of prevalent mixed plaque to calcified plaque. Robust multinomial logistic regression evaluated the effect of time-averaged HDL-C on likelihood of new non-calcified, mixed or calcified plaque formation in segments without plaque (compared to remaining without plaque), and non-calcified plaque regression or transition to mixed or calcified plaque at follow-up (compared to remaining non-calcified).

Results: Participants were mostly female (n=87, 86.1%), with a mean ± standard deviation (SD) age of 51.5±10.3 years and time-averaged HDL-C of 51.7±13.9. Ninety-seven new plaques formed in segments without plaque at baseline, 20 were non-calcified, 21 were mixed, and 56 were calcified. Time-averaged HDL-C...
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:

Table 1. Efficacy comparison of treatment with Bristol-Myers Squibb (BMS) drug.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No. of coronary segments (%)</th>
<th>Adjusted OR (95%CI) per 1-SD increment in time-averaged HDL</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>None to none</td>
<td>1337 (93.3)</td>
<td>1.00 (reference)</td>
<td></td>
</tr>
<tr>
<td>None to non-calcified</td>
<td>20 (1.4)</td>
<td>0.56 (0.35-0.92)</td>
<td>0.021</td>
</tr>
<tr>
<td>None to mixed</td>
<td>21 (1.5)</td>
<td>0.93 (0.65-1.33)</td>
<td>0.678</td>
</tr>
<tr>
<td>None to calcified</td>
<td>56 (3.9)</td>
<td>0.91 (0.59-1.41)</td>
<td>0.673</td>
</tr>
</tbody>
</table>

*Adjusted for Framingham D’Agostino cardiac risk score, proximal segment location, time-averaged CRP, biologic DMARD treatment duration, statin treatment duration, cumulative prednisone dose, waist-to-height ratio, and time-averaged triglycerides.

Figure 1. Impact of HDL-C over time on coronary plaque progression in RA.


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POS0218 TUMOR NECROSIS FACTOR INHIBITORS IMPROVE AORTIC STIFFNESS IN PATIENTS WITH LONGSTANDING RHEUMATOID ARTHRITIS

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Background: Major cardiovascular disease (CVD) benefits of disease-modifying anti-rheumatic drugs (DMARDs) therapy occur in early RA patients with treat-to-target strategy. However, it is unknown whether long-term DMARDs treatment in established RA could be useful to improve CVD risk profile.

Objectives: The aim of this study was to comparatively describe aortic stiffness progression in patients with longstanding and established RA treated with tumor necrosis factor inhibitors (TNFi) or conventional synthetic DMARDs (csDMARDs).

Methods: Ultrasound aortic stiffness index (AoS) has to be considered a proxy for arterial stiffness. AOsI was measured in 64 RA patients with untreated long-standing RA and 64 AoS in a group of RA patients on long-term treatment with TNFi or csDMARDs. Eligible participants were assessed at baseline and after 12 months; changes in serum lipids, glucose and arterial blood pressure were measured. All patients were on stable medications during the entire follow-up.

Results: We included 107 (64 TNFi and 43 csDMARDs) RA patients. Most patients (74%) were on biologic DMARDs or in remission; 17.5% were active disease activity and had some CVD risk factors. The AS gradient was calculated as carotid-femoral (cf)PWV/carotid-radial (cr)PWV ratio and its elevation ≥1 was considered as AS mismatch. p<0.05 was considered 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 0.9±0.2, respectively (p=0.001 for trend). cfPWV elevation ≥10 m/s was observed in 34.1% pts with RA and 32.5% of controls: 6.7 and 6.3% of normotensives and 49.8 and 45.3% of hypertensives, respectively (p=0.06). The frequency of AS mismatch in RA was significantly higher compared to the controls in both normotensive and hypertensive subgroups: 76.7% vs 43.8% (p=0.03) and 94.5% vs 72.9% (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 51.3% (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.

Disclosure of Interests: None declared.

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POS0217 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. Troitskaya1, S. Velmakin1, R. Osipyants1, A. Arbuzova1, V. R. Espinoza2, A. G. Moiseev3, Y. S. Saitov3, M. V. Timoshin4, A. A. Arbuzov5, A. G. Troitskaya1, E. Troitskaya1

1University of Verona, Rheumatology Section, Department of Medicine, Verona, Italy; 2University of Verona, Internal Medicine and Hypertension Unit, Department of Medicine, Verona, Italy

Background: Rheumatoid arthritis (RA) have high CV risk and may benefit from early detection of CVD risk factors. However, it is unknown whether long-term DMARDs treatment in established RA could be useful to improve CVD risk profile.

Objectives: The aim of this study was to comparatively describe aortic stiffness progression in patients with longstanding and established RA treated with tumor necrosis factor inhibitors (TNFi) or conventional synthetic DMARDs (csDMARDs).

Methods: Measurement of pulse wave velocity (PWV) is considered to be a gold reference values and the frequency of AS mismatch in RA

Disclosure of Interests: None declared, Matthew Budoff: None declared.

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