

inflammation, traditional CVD risk factors and metabolic disease have been suggested, but no full explanation is currently present.

Objectives: In this prospective case-control study, we investigated how the progression of subclinical atherosclerosis is associated with CVD risk factors and parameters of inflammation in patients with RA compared with matched controls.

Methods: By the time of diagnosis, patients from northern Sweden diagnosed with early RA are consecutively recruited into an ongoing prospective study. From these, a subgroup aged ≤ 60 years was consecutively included for ultrasound measurements of intima media thickness (IMT) of a. carotis communis at inclusion (T0) (n=79), after 5 years (T5) (n=71) and after 11 years (T11) (n=55). 44 age-sex-matched controls were included and 31 could be reevaluated at T11. Pharmacological treatment, previous CVD, markers of inflammation, lipid status, blood pressure, body mass index as well as measurements of disease activity were registered. Any previous CV events were verified by medical records. European Systematic Coronary Risk Evaluation (SCORE) and Reynolds Risk Score were calculated and Larsen score (of hands and feet) were registered. IMT progression rate (Δ IMT T0-T11) was calculated by subtracting baseline values from IMT after eleven years follow up.

Results: IMT increased significantly between T0 and T11 among patients with RA (IMT T0: 0.51 (0.12) T11: 0.68 (0.16) $p < 0.0001$) and controls (IMT T0: 0.54 (0.12) T11: 0.63 (0.13) $p < 0.0001$). There was a higher progression rate between T0 and T11 in the RA group compared with the controls ($p < 0.05$). In simple regression models, IMT T11 was significantly associated with several traditional CVD risk factors as well as Larsen score at T0 among RA patients (table 1). Moreover, in simple regression models Δ IMT T0-T11 was significantly associated with Larsen score and age at T0 (both $p < 0.01$) among patients with RA. A multiple regression model, with Δ IMT T0-T11 as dependent variable, including traditional CVD risk factors at T0 (age, systolic blood pressure (BP), cholesterol and smoking), resulted in a R^2 of 0.32 where age and cholesterol ($p < 0.01$ for both) were significantly associated with Δ IMT T0-T11. When also adding CRP and Larsen score the R^2 increased to 0.50 and age ($p < 0.05$) and Larsen score ($p < 0.01$) were significantly associated with IMT T11.

Abstract THU0158 – Table 1. Simple regression models among 55 patients with RA with IMT after 11 years of follow up as the dependent variables.

| Variable | IMT T11 | | |
|-------------------------|----------------|------------------|--------|
| | β | 95% CI | p |
| Age, T0 | 0.104/year | 0.071–0.136 | <0.001 |
| Systolic BP, T0 | 0.035/ mmHg | 0.009–0.061 | 0.009 |
| SCORE, T0 | 0.534/unit | 0.261–0.807 | <0.001 |
| Reynolds risk score, T0 | 0.182/unit | 0.048–0.315 | 0.009 |
| Larsen score, T0 | 0.122/unit | 0.004–0.239 | 0.043 |
| DAS 28, T0 | –0.236/unit | –0.771– 0.298 | 0.364 |

Conclusions: In this prospective study, we found that there was an increased progression of atherosclerosis among RA patients, compared with controls, eleven years after diagnosis, and that this increase is associated with Larsen score and age at baseline.

Disclosure of Interest: None declared

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THU0159 BARRIERS TO RHEUMATOID ARTHRITIS TREATMENT OPTIMISATION: REAL-WORLD DATA FROM THE ARTHRITISPOWER REGISTRY

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Background: Few research studies have investigated treatment (Tx) goals in rheumatoid arthritis (RA) from the patients' (pts) perspective, including factors preventing achievement of Tx targets and reasons why pts tolerate sub-optimal disease control.

Objectives: To identify barriers to Tx optimisation, using real-world data from the ArthritisPower registry. Secondary objectives were to understand pts' Tx goals and why pts tolerate active disease.

Methods: This was an observational, cross-sectional sub-study of pts in the ArthritisPower registry. Pts were aged ≥ 19 years, had physician-diagnosed RA, no change to Tx within 3 months of baseline, and had access to a computer/smartphone. Pt-reported outcomes (PROs) included pain, fatigue, sleep, physical function, and general health. Pts also completed an online survey on barriers to Tx escalation, and were classified into 3 groups based on physician and pt attitudes to Tx change (change not offered, change offered and accepted, change offered

and rejected). Disease activity was reported using Routine Assessment of Patient Index Data 3 (RAPID3) scores.

Results: 257 pts met the inclusion criteria (table 1). 195/257 (76%) pts were treated with DMARDs (non-biologic or biologic). 180/257 (70%) pts had high disease activity by RAPID3 (median 18.0 on 0–30 scale), of which only 67/180 (37%) were offered a Tx change at their last physician visit. Most of these pts accepted the Tx change (48/67 [72%]). There were few differentiating factors in demographics, RA-related features, and background therapy among pts who were offered a Tx change versus not. Most pts (33/44 [75%]) who intensified Tx did so because their symptoms remained bad or worsened, whereas only 16/44 (36%) changed because they did not reach pre-defined Tx goals. The most common reason (21/32 [66%]) for deciding not to change therapy was the rheumatologist's satisfaction with the current therapy; pt concerns related to safety of the new therapy were less common (8/32 [25%]). There was a weak correlation between the RAPID3 score and pts' self-reported perception of their own disease activity. The majority of pts (176/257 [69%]) valued being actively involved in making decisions with their doctor about Tx.

Abstract THU0159 – Table 1. Patient demographics at baseline

| | Demographics | | |
|----------------------------------|-----------------------------|-------------------------------|--|
| | Survey participants (N=257) | High disease activity (n=180) | Moderate or low disease activity, or near remission (n=77) |
| Age, mean years (SD) | 51.9 (11.0) | 51.1 (10.8) | 53.7 (11.3) |
| Females, n (%) | 236 (91.8) | 167 (92.8) | 69 (89.6) |
| Ethnicity, n (% white) | 231 (89.9) | 157 (87.2)* | 74 (96.1)* |
| Years since diagnosis, mean (SD) | 11.4 (9.9) | 10.9 (9.2) | 12.4 (11.3) |
| PRO scores, median (IQR) | | | |
| RAPID3 | 15.0 (12.0–19.0) | 18.0 (15.0–20.0)* | 9.0 (6.0–11.0)* |
| Pain | 63.2 (60.0–66.9) | 65.7 (62.7–67.6)* | 58.7 (54.5–61.5)* |
| Fatigue | 64.0 (58.7–68.0) | 64.0 (62.3–69.3)* | 57.1 (50.7–62.5)* |
| Physical function | 37.3 (33.9–40.7) | 36.3 (32.5–38.6)* | 42.6 (38.8–45.9)* |
| Sleep | 59.4 (54.3–63.0) | 61.2 (55.8–64.8)* | 55.8 (50.4–61.9)* |

*Statistical significance between patient groups, $p < 0.05$; p values were nominal in nature and should be interpreted in an exploratory manner. Chi square tests were used to compare patient groups. IQR: Interquartile range; PRO: patient reported outcome; RAPID3: Routine Assessment of Patient Index Data 3; SD: standard deviation.

Conclusions: Despite treat-to-target recommendations,¹ about two-thirds of RA pts with high disease activity in this sample were not offered a Tx change by their rheumatologist. Only a minority changed because they had not met predefined targets for disease control. Pts commonly followed their rheumatologist's decision that no Tx change was needed and placed greater importance on their doctor's Tx goals than their own. These findings suggest that pts may be more deferential to their physicians' satisfaction with poor RA disease control than is appropriate. Encouraging pts (not just physicians) to overcome the status quo by changing medications and striving for low disease activity/remission may be worthwhile, but traditional metrics (e.g. RAPID3) may not reflect the most relevant target for pts' goals for therapy.

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THU0160 JUXTA-ARTICULAR BONE HEALTH AFFECTS NEW CAROTID PLAQUE FORMATION INDEPENDENT WITH GLUCOCORTICOID THERAPY IN POSTMENOPAUSAL PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a chronic and systemic inflammatory disease.

The incidence of cardiovascular (CV) disease is increased in patients with RA, compared with the general population, which is related to the fact that atherosclerosis has an inflammatory etiology.