Abstract THU0110 –Table 1.

Table 1. Baseline characteristics of the matched RA cohort

	Patients with RA + SS (n=283)	Patients with RA only (n=283)
Age, years, mean (SD)	58.4 (12.2)	58.7 (12.8)
Sex, female	235 (83.0)	235 (83.0)
Duration of RA, years, mean (SD)	10.5 (10.0)	10.3 (9.7)
Co-morbidities		
CV disease*	32 (11.3)	31 (11.0)
Malignancy†	29 (10.2)	31 (11.0)
Serious infections‡	24 (8.5)	24 (8.5)
Anti-cyclic citrullinated peptide +, n/m (%)	56/114 (49.1)	66/119 (55.5)
RF+, n/m (%)	91/137 (66.4)	80/144 (55.6)
Erosive disease, n/m (%)	74/216 (34.3)	64/197 (32.5)
Subcutaneous nodules, n/m (%)	69/283 (24.4)	73/283 (25.8)
CDAI score, mean (SD)	24.0 (14.9)	24.1 (14.7)
Number of prior b/tsDMARDs		
0	116 (41.0)	121 (42.8)
1	77 (27.2)	70 (24.7)
≥2	90 (31.8)	92 (32.5)
Number of prior csDMARDs		
0	16 (5.7)	16 (5.7)
1	116 (41.0)	110 (38.9)
≥2	151 (53.4)	157 (55.5)

Data are n (%) unless otherwise stated
 *History of coronary artery disease, myocardial infarction, congestive heart failure requiring hospitalisation, acute coronary syndrome, unstable angina, cardiac revascularisation procedure, cardiac arrest, ventricular arrhythmia, stroke, transient ischemic attack or other CV event
 †History of lung cancer, breast cancer, lymphoma, skin cancer (melanoma and squamous cell carcinoma) or other cancer
 ‡Infection required hospitalisation or IV treatment
 b/tsDMARD=biologic or targeted synthetic DMARD; csDMARD=conventional synthetic DMARD; CV=cardiovascular; n/m=number of patients/total number of patients with available data; SS=Sjögren's syndrome

Abstract THU0110 –Table 2.

Table 2. Mean (SD) change in CDAI score and PROs from index visit to 12 month follow-up visit

	Patients with SS + RA (n=283)	Patients with RA only (n=283)	p value*
CDAI	8.8 (14.6)	9.3 (14.3)	0.704
Patient pain	6.5 (29.2)	14.0 (28.0)	0.002
Patient fatigue	4.3 (29.9)	13.0 (29.9)	0.001
Patient global assessment	5.5 (29.1)	13.0 (28.5)	0.002
Morning stiffness (min)	19.8 (245.2)	21.2 (250.7)	0.947

*Estimated using Student's t-tests
 PRO=patient-reported outcome

Results: After PSM, baseline characteristics in 283 pairs of pts with and without SS were well balanced (Table 1). Mean change in CDAI score was numerically lower in SS than non-SS pts (8.8 vs 9.3; Table 2), but this did not reach statistical significance. Improvements in pain, fatigue and stiffness were 2–2.5-fold greater for non-SS than SS pts (Table 2).

Conclusion: Among those initiating b/tsDMARD treatment, improvements in RA-related PROs were lower in pts with RA with SS than non-SS pts. Physicians may want to consider SS status secondary to RA when managing pts with RA and specifically explore whether additional intervention is needed to improve the pt experience for pts with RA.

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THU0111

ANGIOGENIC T CELLS AS AN INDEPENDENT PREDICTOR OF VASCULAR STIFFNESS IN VERY EARLY RHEUMATOID ARTHRITIS

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Background: Angiogenic T cells (Tang) are responsible of vascular repair and homeostasis. Decreased Tang frequencies have been reported in rheumatoid arthritis (RA), in association with disease activity and previous history of cardiovascular (CV) disease. However, excess of CV risk is already present during the early stage of RA. Whether Tang cells are associated with subclinical vascular abnormalities remains unknown.

Objectives: To evaluate whether Tang cells could be associated with subclinical CV endpoints (subclinical atherosclerosis and vascular stiffness) in the very early stage of RA.

Methods: Patients were recruited upon early referral to the rheumatology department. All patients were untreated at the time of sampling. Tang (CD3+CD31+CXCR4+) frequency was assessed by flow cytometry in peripheral blood samples. Plaque occurrence, cIMT and stiffness parameters were analyzed by Doppler ultrasound in internal carotid, middle cerebral and basilar arteries.

Results: 53 patients were recruited, 47 fulfilling 2010 ACR/EULAR classification criteria for RA (36 women, 26 FR+ and 26 ACPA+) and 6 fulfilling criteria for Clinically Suspect Arthralgia (CSA) (6 women, 3 RF+ and 2 ACPA+). Tang frequency did not differ between RA and CSA (p=0.382) and it was negatively associated with DAS28 (r=-0.448, p<0.001) and SDAI (r=-0.385, p=0.006), but not with duration of the symptoms (r=0.053, p=0.713). Tang levels were not related to individual traditional CV risk factors, body mass index, waist circumference (all p>0.050) nor with the modified SCORE (r=-0.173, p=0.244) or Framingham Risk Score (r=-0.127, p=0.399). Neither atherosclerosis plaque occurrence (r=0.631) nor the cIMT (r=0.070, p=0.652) were associated with Tang levels.

Tang frequency paralleled carotid peak systolic velocity (r=0.399, p=0.008) and end diastolic velocity (r=0.332, p=0.034) in internal carotid artery. Moreover, Tang positively correlated stiffness parameters: vascular strain (VS, r=0.428, p=0.010), vascular distensibility (VD, r=0.479, p=0.004), vascular stiffness (VSf, r=-0.519, p<0.001) and pressure-strain elastic modulus (PSEM, r=-0.531, p<0.015) in left internal carotid artery. Finally, Tang frequency was an independent predictor of stiffness parameters after adjusting for mSCORE, body mass index, DAS28, RF and ACPA positivity: VS (β=0.451, p=0.045), VD (β=0.462, p=0.026), VSf (β=-0.422, p=0.035) and PSEM (β=-0.446, p=0.026).

Conclusion: Tang cells may have a very early role on vascular stiffness in RA. These results point to the Tang subset as the missing link between disease activity and vascular abnormalities during the first stage of the disease.

Disclosure of Interests: None declared

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THU0112

SECULAR TRENDS IN THE INCIDENT RISK OF ACUTE MYOCARDIAL INFARCTION IN RHEUMATOID ARTHRITIS RELATIVE TO THE GENERAL POPULATION

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Background: Recent studies have demonstrated a declining trend in RA mortality relative to the general population (1). This improvement in mortality could be due to improvement in incident risk of cardiovascular events that are the leading cause of excess deaths in RA (2).

Objectives: Our objective was to assess secular trends in ten-year incident risk of acute myocardial infarction (AMI) in incident cohorts of RA versus general population controls, using administrative health data.

Methods: We conducted a retrospective study of a population-based cohort of incident RA cases who first met previously published RA criteria between 01/01/1997 and 31/12/2004 in British Columbia, followed until 12/2014, with general population controls matched 2:1 on gender, age, and index year. Individuals were excluded if they had a diagnosis of MI prior to index date. Incident AMI was defined as first AMI during follow-up using ICD codes (ICD-9 code 410/ICD-10 code I21) in Hospital Discharge data or death certificate in Vital Statistics data. RA and general population cohorts were stratified according to year of RA incidence,