value <0,05 and $r = 0,486$, p value <0,01 respectively), depressed bronchial resistance SGaw ($r = 0,773$, p value <0,01) and increased RV ($r = -0,736$, p value <0,01), which determined BO severity.

Conclusions: More than half of patients with RA have BO predominantly in terminal bronchioles. Accepting significant decrease of Clara cell protein CC16 in patients with RA having BO we suppose pathogenic relationship between functional depression of Clara cell anti-inflammatory activity and BO in this category of patients.

References:

[1] Park HY. Club cell protein 16 and disease progression in chronic obstructive pulmonary disease (2013).

Disclosure of Interest: None declared

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SAT0112 PREVALENCE AND INCIDENCE OVER 3 YEARS OF DIFFERENT COMORBIDITIES IN RHEUMATOID ARTHRITIS (RA): A 3 YEAR LONGITUDINAL STUDY IN 769 ESTABLISHED RA PATIENTS

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Background: Comorbidities including cardiovascular (CV) risk, cancer and osteoporosis are frequent in RA.[1]

Objectives: To quantify at baseline and 3 years later, the prevalence (at baseline) and incidence (over 3 years) of some selected comorbidities.

Methods: This was an open long term (3 years) extension of the COMEDRA 6-month randomized controlled trial in which patients with definite, stable RA were visiting a nurse for comorbidity counselling.[2] Comorbidity status was assessed through face-to-face interviews and nurses provided advice on screening and management, at baseline and 3 years later. The frequency of comorbidities was assessed at both timepoints and incidence of new cases was assessed as overall % of patients and as relative increase in the given comorbidity.

Results: Of the 970 recruited patients, 776 (80%) were followed up at 2–4 years (15, 1.5%, had died) and 769 (79%) had available data for comorbidities at both timepoints: at baseline, mean ($\pm$ SD) age 58 ($\pm$ 11) years, mean disease duration 14 ($\pm$ 10) years; 614 (80%) were women and 538 (70%) were receiving a biologic with a mean DAS28 of 3.1 ± 1.3 .

At baseline, the most frequent comorbidities were history of fracture (31.9%) and high blood pressure (30.9%) and at 3 years the comorbidity which had most increased (i.e., incidence) in this population aged around 60 years, was high blood pressure (4%) whereas smoking had decreased (Table).

Table: frequency of comorbidities at baseline and 3 years later

Comorbidity	COMEDRA study baseline (month 0 for group I and month 6 for group II)	Follow-up at 3 years	Absolute increase, % of 769 patients (relative increase, % of baseline prevalence)
Myocardial infarct	13 (1.7)	18 (2.3)	0.6 (38.4)
Angina	9 (1.2)	17 (2.2)	1.0 (88.9)
Stroke	16 (2.1)	29 (3.8)	1.7 (81.2)
Obstructive pulmonary disease	8 (1.0)	14 (1.8)	0.8 (75.0)
Diabetes	39 (5.1)	50 (6.5)	1.4 (28.2)
High Blood Pressure	238 (30.9)	270 (35.1)	4.2 (13.4)
Hyperlipidemia	149 (19.4)	170 (22.1)	2.7 (14.1)
Obesity (BMI $\geq$ 30)	114 (14.8)	124 (16.1)	1.3 (8.8)
Smoking	120 (15.6)	104 (13.5)	-2.1 (-13.3)
Colon cancer	2 (0.3)	2 (0.3)	0 (0)
Breast cancer	23 (3.0)	27 (3.5)	0.5 (17.4)
Lung cancer	1 (0.1)	2 (0.3)	0.2 (100.0)
Prostate cancer	3 (0.4)	4 (0.5)	0.1 (33.3)
Uterus cancer	7 (0.9)	7 (0.9)	0 (0)
Skin cancer (all types)	26 (3.4)	36 (4.7)	1.3 (27.8)
Lymphoma	2 (0.3)	3 (0.4)	0.1 (50.0)
History of fracture	245 (31.9)	256 (33.3)	1.4 (4.5)

Conclusions: Comorbidities are frequent in RA though screening does not always address the most frequent or severe comorbidities. Efforts must be pursued to improve comorbidity screening and prevention.

References:

[1] Ref 1. Baillet A, Gossec L et al. Ann Rheum Dis. 2016;75(6):965–73.

[2] Ref 2. Dougados M, Soubrier M, et al. Ann Rheum Dis. 2015;74(9):1725–33.

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SAT0113 OBESITY AND METABOLIC SYNDROME INFLUENCE ON N-TERMINAL PRO-BRAIN NATRIURETIC PEPTIDE LEVELS IN RHEUMATOID ARTHRITIS PATIENTS

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Background: N-terminal pro-brain natriuretic peptide (NT-proBNP) is a recognized myocardial injury marker, a known predictor of heart failure and cardiovascular death. Rheumatoid arthritis (RA) patients (pts) were shown to have higher than in general population NT-proBNP concentrations, positively correlating with IL-6 levels. Adipose tissue is considered to be one of the sources of this pro-inflammatory cytokine. But on the other hand, metabolic syndrome (MS) and obesity, which also occur in RA pts, are usually associated with lower NT-proBNP levels in general population.

Objectives: To find out whether overweight/obesity or MS may modify NT-proBNP levels in RA pts.

Methods: A total of 68 early RA pts (72% females, 28% males) was enrolled in the study. Mean disease duration was 6 [4;8] months, and RA activity using DAS28 calculator was 5,6 [5,1;6,4] scores. The majority of pts had positive RF (87%) and ACPA (88%). All pts were glucocorticoids and disease-modifying antirheumatic drugs - naïve prior to inclusion. The overweight/obesity was determined by WHO criteria in patients with body mass index (BMI) ≥ 25 kg/m². National Cholesterol Education Program/Adult Treatment Panel III criteria were used to confirm MS. Serum levels of NT-proBNP (pg/mL) were measured using electrochemoluminescence test Elecsys proBNP II (Roche Diagnostics, Switzerland). NT-proBNP levels > 125.0 pg/mL were considered as elevated.

Results: Overweight and obesity were established in 54,4% RA pts. Overweight/obese RA pts were older than pts with normal BMI (57 [53;61] years vs 48 [34;61] years, $p=0,02$), had higher DAS28 scores (5,85 [5,20;6,57] vs 5,28 [4,76;5,69], $p<0,01$), higher CRP (38,8 [14,3; 47,9] mg/L vs 12,8 [2,2;28,8] mg/L, $p<0,01$). Elevated NT-proBNP concentrations were found in 62,2% overweight/obese pts vs 29,0% pts with normal weight ($p<0,01$), median NT-proBNP concentrations were 153,4 [87,3;236,9] pg/mL vs 75,2 [40,2;147,4] pg/mL, respectively ($p=0,02$). MS was established in 62,2% pts with BMI ≥ 25 kg/m² and in 25,8% pts with BMI < 25 kg/m² ($p<0,01$). Median NT-proBNP concentrations did not vary significantly in pts with and without MS, whether they were overweight/obese ($p=0,75$), or had normal BMI ($p=0,27$). Although higher rates of elevated NT-proBNP concentrations were documented in pts with MS (61,3% vs 35,1%, $p=0,03$). NT-proBNP levels positively correlated with waist circumference ($r=0,33$, $p<0,01$), ESR ($r=0,25$, $p=0,04$), and CRP ($r=0,3$, $p=0,01$).

Conclusions: Overweight and obesity in RA pts were associated with increased NT-proBNP levels. Abdominal obesity was identified as the only MS component, influencing NT-proBNP levels, presumably via up-regulation of the inflammatory process.

Disclosure of Interest: None declared

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SAT0114 POOR CARDIOVASCULAR RISK MANAGEMENT IN RHEUMATOID ARTHRITIS PATIENTS DESPITE AN EXPLICIT CARDIOVASCULAR RISK MANAGEMENT PROGRAM

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Background: In 2011, we started a cardiovascular (CV) risk management program for rheumatoid arthritis (RA) patients visiting Reade in the Netherlands. We previously reported the presence of under treatment of hypercholesterolemia and hypertension [1].

Objectives: To assess the effectiveness of our CV risk management program after one year.

Methods: CV risk screening was performed at baseline and we informed the general practitioner (GP) about the results, including advices regarding the initiation of cardio preventive drugs. In high risk patients, antihypertensives were recommended when systolic blood pressure > 140 mm/Hg and statins were recommended when low-density lipoprotein > 2.5 mmol/L. The decision to start preventive medication was left to the GP. CV risk screening was repeated after one year. Patients completed a questionnaire about the actions that were taken following the results of the initial screening.

Results: Of the 266 patients 202 (76%) were female, the mean age was 58 ± 11 years. After one year, 88 out of 134 patients who received inadequate or no treatment at baseline were still untreated or undertreated. Of the 188 (71%) patients who were at high CV risk and who did have an indication to start therapy, only 7.5% was contacted by their GP and another 6.8% arranged an appointment themselves. While the 10-year CV risk did not decrease in the group as a whole, a risk reduction was found in the patients that started medication. Remarkably, 42% of patients reported lifestyle changes, including more exercise (20%), diet adaption (16%) and weight loss (9%).

Conclusions: It is striking that one year after the introduction of our CV risk

Table 1. Cardiovascular risk factors at baseline and after one year

	Baseline	Follow-up
CV disease history	20 (7.5)	23 (8.6)
CV risk <10%	43 (16.2)	52 (19.5)*
CV risk 10–20%	60 (22.6)	62 (23.3)
CV risk >20%	143 (53.8)	129 (48.5)*
SBP in mm/Hg	135±17	135±18
Body Mass Index in kg/m ²	26.4±5.0	26.3±4.8
TC in mmol/l	5.5±1.0	5.2±1.0**
Triglycerides in mmol/l	1.4±0.8	1.4±0.8
LDL-cholesterol in mmol/l	3.2±0.9	3.0±0.9**
HDL-cholesterol in mmol/l	1.6±0.5	1.6±0.5
TC/HDL-ratio	3.7±1.2	3.7±1.2**
Current smokers	60 (22.6)	53 (20.1)
Diabetes mellitus	12 (4.5)	12 (4.5)
SBP >140 mm/Hg	95 (35.7)	96 (36.2)
LDL >2.5 mmol/l	208 (78.8)	180 (68.4)**
TC ≥6.5 mmol/l	37 (13.9)	26 (9.8)
Antihypertensives	81 (30.5)	88 (33.1)
Statins	38 (14.3)	57 (21.4)**

CV = Cardiovascular, HDL = high-density lipoprotein, LDL = low-density lipoprotein, TC = total cholesterol, SBP = Systolic blood pressure. Results are presented as mean ± standard deviation or n, (%). *p<0.05, **p<0.01.

management program, only 14.3% patients with an indication for preventive treatment visited their GP and only 14% started with CV risk lowering drugs. This is mainly caused by the small percentage of high risk patients that contacted their GP. On the other hand, a high percentage reported the start of healthy lifestyle. These results underscore the need for a short-term follow-up, in close collaboration with primary care providers to ensure appropriate CV risk management. This is a future implementation project that will be initiated shortly.

References:

- [1] Ann Rheum Dis 2015;74:192.
Disclosure of Interest: None declared
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SAT0115 EFFECT OF BIOLOGICAL DISEASE MODIFYING ANTI-RHEUMATIC DRUGS (BDMARDS) ON CARDIOVASCULAR DISEASE (CVD) RISK IN RA PATIENTS:THREE YEARS RESULTS FROM A CANADIAN COHORT

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Background: RA patients have a higher risk for developing CV Diseases (e.g. IHD and stroke) than the general population. We reported previously (ACR, EULAR) the results CV risk in our patients treated with bDMARDs for 1 & 2 years.

Objectives: To assess the effect of bDMARDs treatment on 10-year CV disease risk in patients with RA after 36 months and evaluate the impact of their use on the incidence of CV events

Methods: A cohort of RA patients was followed prospectively. Demographics, disease activity parameters, inflammatory markers, CV events, CVD 10 year risk assessment [Framingham Risk Score (FRS)] were ascertained at the baseline and every six months, up to 36 months. Statistical analysis utilized SPSS (IBM version 24) software.

Results: 321 patients were included, of those 270 patients were followed for three years. Eight bDMARDs were included evaluating their effect on disease activity and CV risk in 233 patients who continued on their prospective biologic for the three years.

Significant overall reduction in disease activity in all groups was obtained as compared to baseline. The whole group DAS improved from 3.996 to 3.118.

The HAQ also improved significantly (1.201 to 0.857) and the CRP improved significantly (12.91 mg/L to 7.3).

Overall, Total cholesterol (TC) changed from 5.195 mmol/L at baseline to 4.992 (P 0.001). High Density Lipoprotein Cholesterol (HDL-C) increased significantly at 36 months from 1.336 to 1.376. However, the Atherogenic Index (AI) for the whole cohort changed from 3.636 to 3.845. The overall 10 year risk of cardiovascular event increased from 12.878 to 13.8

The cohort included 321 patients. Eight bDMARDs were evaluated to assess their effect on disease activity and CVD risk. The outcomes in 233 patients who continued their prospective biologic for the three years were reported (table). 77% were females, median RA duration was 11 ys. Significant reduction in disease activity in all groups was achieved vs. baseline. The DAS improved from 3.996 to 3.118, the HAQ improved significantly (1.201 to 0.857) and the CRP also improved significantly (12.91 mg/L to 7.3). Overall, Total cholesterol (TC) changed from 5.195 mmol/L at baseline to 4.992 (P 0.001). High Density Lipoprotein Cholesterol (HDL-C) increased significantly at 36 months from 1.336 to 1.376. The Atherogenic Index (AI) for the cohort changed from 3.636 to 3.845. The overall 10 year risk of cardiovascular event increased from 12.878 to 13.8.

Table 1

	Rituximab n=40	Tocilizumab n=42	Golimumab n=24	Adalimumab n=42	Abatacept n=35	Etanercept n=39
DAS 28	4.3 vs 3.7 P 0.014	4.2 vs 2.8 P<0.001	4.1 vs 3.4 P 0.028	3.4 vs 2.8 P 0.011	4.3 vs 3.1 P<0.001	4.0 vs 2.8 P 0.0015
HAQ	1.3 vs 1.1	1.0 vs 0.9	1.2 vs 1.0	1.7 vs 0.5	1.2 vs 1.0	1.1 vs 0.7 P<0.001
Swollen Joint Count	3.8 vs 2.1 P 0.005	5.0 vs 1.8 P<0.001	5.3 vs 6.7 P 0.005	3.6 vs 1.7 P 0.010	3.7 vs 3.6	3.9 vs 1.7 P<0.001
AI	4.0 vs 3.7 P 0.045	4.7 vs 4.0	3.2 vs 3.94	3.8 vs 3.7	4.1 vs 3.9	4.2 vs 3.7
10 yr CV Risk	15.9 vs 17.9	13.5 vs 14.4	14 vs 15.7	10.1 vs 13.2 P 0.009	13.5 vs 14.1	11.2 vs 12.2

Conclusions: The bDMARDs studied were effective in controlling the disease activity of RA. Although the overall CVD risk was not reduced in the group as a whole at 36 months, bDMARDs may still play a role in reducing the CVD risk in these patients

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SAT0116 PREVALENCE OF UNACCEPTABLE PAIN IN PATIENTS WITH LONG-STANDING RA

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Background: Pain in patients with rheumatoid arthritis (RA) is the most common and troublesome symptom, occurs frequently in many sites in the musculoskeletal system, causes fatigue and functional impairment. The origin of pain in RA may be inflammatory and/or other, such as increased sensitivity to pain, fibromyalgia and osteoarthritis.

Objectives: To study unacceptable pain in an inception cohort of patients with RA at 15 years follow-up

Methods: This study concerns 477 patients from the BARFOT inception cohort study, consisting of 839 patients with early RA, who have passed the pre-defined 15 year follow-up visit. Unacceptable pain (UP) was defined as ≥40 mm on a continuous VAS pain scale of 100 mm with the ends "no pain" and "worst possible pain" (1). ADL- function was assessed by the HAQ test, physical function by the SOFI test and joint damage by the van der Heijde modified Sharp method. Remission was defined as DAS28 ≤2.6.

The prevalence of UP over time as well as the relationship of UP with clinical variables were assessed. Baseline predictors of UP after 15 years were evaluated by logistic regression.

Results: The mean baseline VAS pain was 45 mm, decreased after 6 months to 29 and remained after that fairly stable through the years ending in 31 at 15 years. UP followed a similar course with 56% of the patients at baseline, which decreased to 30% after 6 months and then behaved stable until 15 years, 34%. Seventy-seven per cent of the patients with UP were not in remission at 15 years, while 23% had UP in spite of being in remission.

Patients with UP who were in remission at the 15 years visit had higher mean age (p=0.004), ESR (p=0.001) and SOFI (p=0.001) than patients without UP.

Patients with UP who were not in remission at this point in time were more often women (0.008) and had more tender joints (p=0.001) than patients who did not have UP.

In both remission groups the mean DAS28, patient global assessment and HAQ were higher in patients with UP compared with patients without. The means of the x-ray joint damage scores - total score, erosion and joint space narrowing scores - were very similar among patients with or without UP, statistically not significant.

A logistic regression analysis of the ability of baseline variables to predict UP at 15 years was performed. It was shown that female gender and baseline pain independently predicted unacceptable pain at 15 years in patients not in remission while in patients in remission high age and ESR were independent predictors.