ftTB were associated with disseminated (miliary) tuberculosis (**mTB**) in 6 (3.7% of 161; 28.57% of 21) **RA** patients with **ftTB** or **ftTB**.

Demographics, onset and duration of disease of 161 **RA** patients with **TB**, **ftTB**, **ftTB**, or **mTB** are summarized in Table.

Sex	Number of autopsies	Mean age in years at death $\pm$ SD	Range of age (in years)	Mean age at onset of disease $\pm$ SD	Disease duration (in years) $\pm$ SD
RA patients (total)	161	65.32 $\pm$ 12.95	16–88	50.83 $\pm$ 16.96	14.43 $\pm$ 10.51
without TB	140 of 161	64.77 $\pm$ 13.28	16–88	50.22 $\pm$ 16.99	14.36 $\pm$ 10.13
with TB	21 of 161	69.00 $\pm$ 9.70	50–84	54.19 $\pm$ 16.39	14.81 $\pm$ 12.41
with fibrous TB	12 of 21	70.92 $\pm$ 8.48	59–84	52.33 $\pm$ 16.01	18.58 $\pm$ 12.76
with fibrocasseous TB	9 of 21	66.44 $\pm$ 10.59	50–80	56.67 $\pm$ 16.55	9.78 $\pm$ 9.91
with miliary TB	6 of 21	68.33 $\pm$ 11.09	50–82	58.67 $\pm$ 8.24	9.67 $\pm$ 4.85

The mean age of **RA** patients was higher with **TB**, **ftTB**, **ftTB** or **mTB** in comparison to total population or to the patients without **TB**, but the difference was only significant between patient cohorts with **ftTB** and **RA** patients without **TB** (70.92 years versus 64.77, $p < 0.043$).

There was a definitely shorter duration of **RA** in patients with **ftTB** or **mTB** compared to the total population.

Proliferative or exudative epithelioid granulomatous **mTB** involved different organs, such as lung in 5, liver in 3, spleen in 2, lymph nodes in 2, adrenal gland in 1, synovial membrane in 1, vertebrae in 1 and pituitary gland in 1 of 6 **RA** patients with active **mTB**.

There was a significant correlation between: **TB** and **mTB** ($c=1$, $\chi^2=33.96$, $p<0.00000001$), **TB** and **ftTB** ($c=1$, $\chi^2=78.36$, $p<0.00000001$), **TB** and **ftTB** ($c=1$, $\chi^2=55.69$, $p<0.00000001$), or **ftTB** and **mTB** ($c=0.99$, $\chi^2=56.89$, $p<0.000001$). The link between **ftTB** and **mTB** was not significant ($c=0.45$, $\chi^2=0.007$, $p<0.9333$ – NS).

Conclusion: **TB**, **ftTB**, **ftTB**, or **mTB** complicated **RA** in both sexes, and at any time in the course of the disease.

The mean age at death was higher in all forms of **TB**. Significant difference was only in mean age of **ftTB** compared to the patients' cohort without **TB**. Fibrocasseous tubercle or miliary dissemination of tuberculosis reduced definitely the survival time (and disease duration) of aged **RA** patients.

Post-primary **TB** especially **ftTB** (mostly in the lungs) represents a high risk of miliary dissemination in **RA**. In our autopsy material **mTB** was the consequence of endogenous exacerbation of **TB** (and was not due to an exogenous reinfection), based on the high values of Youle's association coefficients between **TB** or **ftTB** and **mTB**.

The **mTB** may be considered as a terminal phenomenon, because of the limited numbers of granulomas involving only a few organs. The exudative character – beside proliferative epithelioid granulomas – must be regarded as histological evidence of impaired immune reactivity, an unfavorable prognostic sign in elderly patients, forecasting the possible fatal outcome of **RA** associated **TB**.

Disclosure of Interests: None declared

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FRI0056

LUNG COMPROMISE SCREENING IN PATIENTS WITH EARLY RA. A MULTICENTRIC CROSS SECTIONAL STUDY

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Background: Rheumatoid arthritis (RA) affects 0.4-1.3% of general population (1). It can affect lungs in different ways, with interstitial lung disease (ILD) as the most severe. Clinically evident ILD has been reported in 10-42% of patients, with a great impact in prognosis (2).

Objectives: To identify the prevalence of lung involvement in early rheumatoid arthritis patients (ERA) without previous known lung disease and describe the association between high resolution computed tomography (HRCT), lung functional tests (LFT) and clinical findings.

Methods: Cross sectional multicentric study. We included ERA patients (1 year or less since diagnosis) consecutively. Patients with previous RA related lung disease or biologic/targeted synthetic Dmard treatment were excluded. HRCT,

immunological tests (rheumatoid factor, anti-CCP, ANA), LFT and clinical evaluation were performed.

Results: We included 74 patients, 63 (85.1%) woman, mean (SD) of 47 (17.7) years. Thirty-seven patients (50%) were current or former smokers. Abnormal findings in HRCT were found in 62 patients (88.6%): ILD in 6 (8.6%), airway involvement in 40 (70%) and emphysema in 7 (10%). Ten patients (13.5%) had abnormal auscultation (2 sibilances, 2 roncus, and 6 crackles). Six patients (8.1%) had digital clubbing. Regarding immunological tests, 54/61 (88.5%) patients were positive for Anti CCP, and 53/61 (86.9%) were positive for RF. We compared features of patients with findings related to RA in HRCT (interstitial and/or airway) with those without them. We found no differences in the mean (SD) of DAS-28 [4.74 (1.38) vs 4.32 (1.39); $p=0.27$]. The prevalence of anti-CCP was not higher in patients with abnormal HRCT [38/44 (86.3%) vs 16/17 (94.1%); $p=0.39$]. Patients with abnormal HRCT were older [median (IQR) 50.5 years (44.5-59.5) vs 43 years (32-51); $p=0.008$] and showed higher VSG values [mean (SD) 39.09 (24.03) vs 27.38 (17.6); $p=0.043$]. Abnormal physical examination or dyspnea (class 2 mMRC or higher) was significantly associated with HRCT abnormalities [26 (50%) vs 3 (13.6%); $p=0.003$] and the presence of ILD on HRCT was significantly associated with crackles on the auscultation [4/68 (6.25%) vs 2/6 (33.33%); $p=0.023$].

Conclusion: This study shows a high prevalence of lung involvement in ERA patients of less 1 year from diagnosis. Also, we showed a significant association between HRCT and physical examination findings. This data highlights the importance of the clinical examination in Rheumatoid Arthritis patients. More studies with bigger samples and longitudinal follow up are needed to confirm and complete our results.

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FRI0057

A MODEL FOR QUANTIFYING THE EFFECT OF INFLAMMATION ON CARDIOVASCULAR DISEASE RISK PREDICTION IN RA PATIENTS

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Background: Patients with rheumatoid arthritis (RA) are at increased risk for cardiovascular disease (CVD)[1]. Quantifying the effect of inflammation on CVD risk is important because rheumatologists can reduce inflammation with effective RA medications. A new score has been developed for predicting the risk for a CVD event (MI, stroke or CV death) in RA patients. It combines serological measures of inflammation (the multi-biomarker disease activity [MBDA] score, a measure of RA disease activity; and three individual biomarkers [TNF-RI, MMP-3 and leptin]), with age and four conventional CVD risk factors (smoking, hypertension, diabetes and history of a high-risk CVD condition)[2]. To gain insight into the potential effect that treating inflammation may have on the CVD risk score, it would be useful to know how the score is affected by the level of inflammation.

Objectives: Explore the quantitative contribution of inflammation to CVD risk score in individual RA patients.

Methods: To quantify the effect of inflammation on the CVD risk score across a range of MBDA scores, a commercial dataset of 177,486 RA patients with ≥ 2 MBDA tests between October 2010 and June 2019 was split 2:1 into training and validation datasets. Curves showing variation in the CVD risk score across the spectrum of all possible MBDA scores (1-100) were generated for canonical patient types differing in the number of conventional risk factors (0 to 4) and age (45, 55, 65, 75, 85 years). To generate these curves, the contributions of TNF-RI, MMP-3 and leptin to the CVD risk score were treated in aggregate (denoted the molecular score) and estimated using a linear regression model of the difference in molecular scores vs. the difference in MBDA scores. This model for the molecular score was fit in the training dataset, then in the full dataset, with dataset (training or validation) and the interaction between dataset and change in MBDA score included as additional predictor variables. The method was considered validated if the F-test for the interaction variable was not significant at the 0.05 level.

Results: The model for estimating the molecular score from the MBDA scores was validated and shown to fit the data well (Figure 1). The estimated molecular

score was applied to the CVD risk score algorithm to generate curves that show how CVD risk score varies with MBDA score for several distinct patient types. These curves demonstrate that the predicted 3-year CVD risk increases continuously and markedly with increasing level of inflammation, as represented by the MBDA score (Figure 2). Age and the number of conventional risk factors also affected the predicted CVD risk, with older patients (Figure 2a) and those with more conventional risk factors (Figure 2b) being at higher risk for a CVD event.

Conclusion: The level of CVD risk predicted by a new prognostic test for RA patients depends not only on conventional risk factors, which are relatively time invariant, but also varies greatly due to inflammation, which can potentially be reduced with RA treatment.

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Figure 1: Predicted versus observed molecular score in the validation dataset (N=59,162). The model derived to determine predicted molecular scores (denoted m) was: $mref + \beta(MBDA - MBDAref)$, where MBDAref and mref are fixed reference values and β is the coefficient estimated in the training dataset. The dashed line represents predicted molecular score = observed molecular score.

Figure 2: Relationship between inflammation and predicted CVD risk. A) Predicted 3-year CVD risk by MBDA score for hypothetical patients having diabetes, hypertension and the indicated ages. B) Predicted 3-year CVD risk by MBDA score for hypothetical 55-year old patients having 0 to 4 conventional risk factors, as indicated. Curves were derived using the CVD risk prediction algorithm across the range of all possible MBDA scores with molecular scores assigned by the model described above. For these CVD risk curves, MBDAref was fixed at 39 (the median MBDA score in the validation dataset), and mref was fixed at 0.07 (the median molecular score observed in patients with an MBDA score of 39).

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FRI0058 THE IMPORTANCE OF SPECKLE TRACKING ECHOCARDIOGRAPHY IN THE EVALUATION OF CARDIAC FUNCTIONS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is an inflammatory disease that includes chronic, progressive joint arthritis and also has multi-systemic involvement. It is known that the acceleration of many cardiovascular diseases causing mortality and morbidity, especially atherosclerosis and heart failure, is increased in RA patients.

Objectives: In this study, it was aimed to analyze the layer-specific (endocardial, transmural and epicardial) strain values obtained by speckle tracking echocardiography method in the determination of subclinical cardiac dysfunction in RA patients and to determine the correlation between anti cyclic citrullized peptide (Anti-CCP) titers, disease activity score (DAS-28), disease duration and strain values.

Methods: This study was performed with 63 RA patients and 31 healthy participants. The patients were grouped as <5 years, 5-10 years and >10 years according to their disease duration. DAS28-CRP was used to determine disease activation. The standard assessment included complete serum concentration of C-reactive protein, Anti-CCP, Romatoid faktör (RF), N-terminal pro b-type natriuretic peptide (NT-proBNP) and homocysteine. Endocardial, transmural and epicardial strain values were analyzed by M-mode, 2D, tissue doppler and speckle tracking echocardiography.

Results: When the groups were compared in terms of laboratory data, NT-proBNP value of RA patients was higher than the control group (p=0.044), homocysteine level was similar (p>0.05). When the groups were compared in terms of conventional echocardiographic parameters, ejection fraction of the control group was similar (p>0.05). E/A and E/E' ratios were found to be significantly different (p<0.001, p=0.015). When the groups were compared in GLS values obtained by speckle tracking echocardiography, endocardium, transmural and epicardium GLS values were lower in RA patients (p<0.05) (Table 1). As the disease duration increased, GLS values were found to be worse (p<0.05). There was a significant correlation between RA disease activity scores level and LV GLS value, increasing levels of disease activity was associated with worse LV GLS (r=0.583, p<0.01 and r=0.681, p<0.01 ve r=0.689, p<0.01 for endocardium, transmural and epicardium respectively. There was a significant correlation between anti-CCP, RF and LV GLS value, higher Anti-CCP and RF titers were associated with worse LV GLS (r=0.467, p<0.01 and r=0.509, p<0.01 and r=0.551, p<0.01) for endocardium, transmural and epicardium respectively.

Table 1. Comparison of layer-specific GLS values of groups

	<5	5-10	>10	Control	p
GLS endocardium Mean ± SD	-23,98±1,84	-23,29±1,59	-21,71±1,93	-24,95±0,73	Y: 0,000 β: 0,000 P: 0,001
GLS transmural Mean ± SD	-21,78±1,71	-21,20±1,66	-19,85±1,50	-22,98±1,17	Y: 0,001 β: 0,020 †: 0,017 P: 0,001
GLS epicardium Mean ± SD	-20,05±2,02	-19,23±1,77	-17,98±1,38	-20,83±0,70	β: 0,000 ‡: 0,023 P: 0,000 P: 0,001

SS: Standard Deviation, Y: 1-2, β: 2-3, P: 3-4, †: 1-3, ‡: 1-4, P: 2-4, GLS: Global longitudinal strain

Conclusion: The layer-specific global longitudinal strain values obtained by speckle tracking echocardiography were found to be decreased in RA patients. This study, which has been shown to decrease strain values before the reduction