individuals with early RA[3]. However, few studies have detected the radiographic damage in the whole upper extremity on the mechanical stress.

Objectives: To examine the relationship between mechanical stress and radiographic damage in RA, we evaluated the joint destruction of the dominant and non-dominant upper extremity.

Methods: The joint destruction of the upper extremity (shoulder, elbow, wrist, metacarpo-phalangeal, interphalangeal, proximal interphalangeal) in 295 patients with RA, who were from 25 years to 91 years (mean age 64 years, mean disease duration 183 months, 86% females and 97% right-handed), was assessed according to the eroded joint, which was defined as ≥ 2 by Larsen scores for radiographic damage. These were divided into the dominant and non-dominant upper extremity. The Wilcoxon signed-rank test was used to examine the difference between the eroded joint count (EJC) in the dominant and the non-dominant upper extremity.

Results: The mean EJC in the dominant and the non-dominant upper extremity was 2.4 and 2.05 respectively. The EJC was significantly more in the dominant than the non-dominant upper extremity. And, in regards to every joint of upper extremity, the eroded joint rate was higher in the dominant than the non-dominant
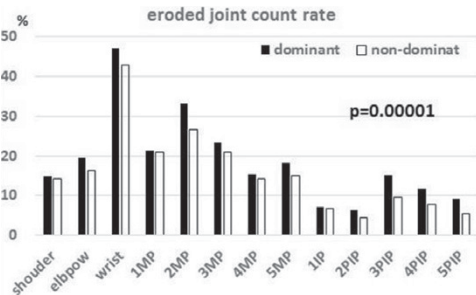
Conclusions: The eroded joint count of the upper extremity was significantly more in the dominant than the non-dominant one, therefore it was suggested that the mechanical stress influenced the radiographic damage in patients with RA

References:
[1] John H Bland, et al.: Hemiplegia and rheumatoid hemiarthritis. Arthritis Rheum (11) 1: 72–80, 1968.
[2] E N Glick, et al.: Asymmetrical rheumatoid arthritis after poliomyelitis. Br Med J 3: 26–28, 1967.
[3] Jung Hee Koh, et al.: Radiographic structural damage is worse in the dominant than the non-dominant hand in individuals with early rheumatoid arthritis. PLOS ONE, doi: 10. 1371/journal. Phone.0135409 August 6, 2015.

Disclosure of Interest: None declared

COMPARATIVE CARDIOVASCULAR SAFETY OF ABATACEPT AND TUMOR NECROSIS FACTOR INHIBITORS IN RHEUMATOID ARTHRITIS PATIENTS WITH AND WITHOUT CARDIOVASCULAR DISEASE: A POPULATION-BASED COHORT STUDY

Y. Jin[1], E.H. Kang[1,2], G. Brill[1], R.J. Desai[1], S.C. Kim[1,3]. [1] *Division of Pharmacoepidemiology and Pharmacoeconomics, Brigham and Women's Hospital, Harvard Medical School, Boston, United States;* [2] *Division of Rheumatology Department of Internal Medicine, Seoul National University Bundang Hospital, Seongnam, Korea, Republic Of;* [3] *Division of Rheumatology, Immunology and Allergy, Brigham and Women's Hospital, Harvard Medical School, Boston, United States*

Background: Rheumatoid arthritis (RA) patients have an increased risk of cardiovascular disease (CVD). Recent studies suggested that treatment of RA with tumor necrosis inhibitors (TNFi) can reduce the risk of cardiovascular events. However, it is unclear how abatacept, a selective costimulation modulator, affects cardiovascular risk among RA patients compared with TNFi.

Objectives: To evaluate the comparative cardiovascular safety of abatacept versus TNFi in RA patients with and without underlying CVD.

Methods: We identified RA patients aged ≥ 18 with ≥ 2 RA ICD-9 codes (714.xx) separated by ≥ 7 days but <365 days, from two large insurance claims data across the U.S.: Medicare (2008–2013) and Truven MarketScan (2006–2015). Only new users of abatacept or TNFi (adalimumab, etanercept, certolizumab, golimumab, and infliximab) were included. The primary outcome of interest was a composite endpoint of CVD including myocardial infarction (MI), stroke/transient ischemic stroke, or coronary revascularization. Secondary outcomes were each component of the primary outcome, incident heart failure (HF), and venous thromboembolism (VTE). 1:1 propensity score (PS) matching was performed separately in each database and each subgroups (with or without baseline CVD). Then the PS-matched subgroups were aggregated to form the overall matched cohort. Cox regression model was used to estimate the hazard ratio (HR) and 95% confidence interval (CI) of risk of each outcome. Estimates from two different databases were combined through an inverse variance-weighted fixed-effects model.

Results: After 1:1 PS matching, there were 6,102 patient pairs from Medicare

and 6,934 pairs from MarketScan. Among them, patients with baseline CVD were 35.3% in Medicare and 14.0% in MarketScan. Baseline characteristics were well balanced between two treatment groups after matching (standardized mean difference <0.1). In Medicare cohort, abatacept consistently showed a decreased risk of composite CVD compared with TNFi in overall and each subgroup (Figure). However, in MarketScan cohort, where the population was younger than Medicare cohort, there was no association between abatacept and CVD compared to TNFi. After combining the two databases, abatacept was significantly associated with reduced risk of composite CVD outcome vs. TNFi in overall cohort (HR =0.79, 95% CI=0.67–0.92) and baseline CVD subgroup (HR =0.79, 95% CI=0.64–0.98). We also observed similar trend for secondary outcomes, where abatacept had decreased risk than TNFi.

Figure. Risk of composite CVD outcome in Abatacept initiators versus TNF inhibitors: PS-matched analysis
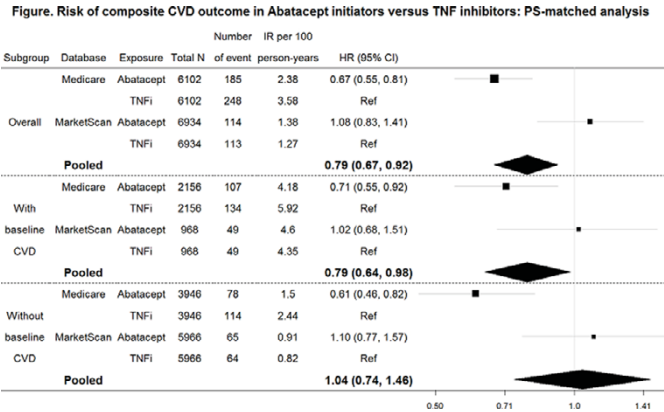
Conclusions: In this large multi-database population-based study of RA patients, abatacept treatment was associated with reduced risk of CVD compared to TNFi, especially among older population and patients with prior CVD conditions.

Disclosure of Interest: None declared

RESTORATION OF DECREASED LYMPHOCYTES, CD8+T CELL SUBSETS WITH TH1 SKEWED PHENOTYPE ASSOCIATE WITH SPONTANEOUS REGRESSION OF LYMPHO-PROLIFERATIVE DISORDERS IN RHEUMATOID ARTHRITIS PATIENTS TREATED WITH METHOTREXATE

S. Saito[1], K. Suzuki[1], K. Yamaoka[1], K. Amano[2], M. Tokuhira[3], T. Takeuchi[1]. [1] *Division of Rheumatology, Department of Internal Medicine, Keio University School of Medicine, Tokyo;* [2] *Division of Rheumatology, Saitama Medical Center, Saitama Medical University;* [3] *Division of Hematology, Saitama Medical Center, Saitama Medical University, Saitama, Japan*

Background: Lympho-proliferative disorder (LPD) is known as a relatively rare but life-threatening complication in RA patients under MTX administration. Spontaneous regression of LPD after MTX withdrawal is regarded as a distinct character of LPD under MTX administration. Previous study from our institution [1] and others [2] reported the link between decreased lymphocyte counts at LPD diagnosis and restoration after MTX cessation and the regression of LPD.

Objectives: To investigate the immunological factors including lymphocyte subsets which involved in spontaneous regression of LPD following MTX withdrawal.

Methods: We studied 35 RA patients complicated with LPD under MTX administration in our institution. Age, sex, RA disease duration matched control MTX-treated patients (N=35) were selected. LPD patients were divided into two groups regarding to the status of LPD after MTX cessation; regressive group (N=22) and persistent group (N=13). Clinical features were compared among 3 groups. Flowcytometric analysis of the whole blood sample and measurement of cytokine concentration in ELISA were conducted in a part of the patients (N=10, 7 regressive, and 3 persistent LPD) and controls (N=10). The time of MTX cessation, which was simultaneous with LPD diagnosis, was defined as week 0, and blood sample was collected at week 0, 4 and 12.

Results: At week 0, number of peripheral lymphocytes was significantly decreased in regressive group, compared to persistent group and control group. Flowcytometric analysis revealed significant decrease in proportion of effector memory CD8+ T cells (EMCD8+T), Epstein Barr Virus specific CD8+ T cells (EBV specific CD8+) and T helper 1 cells (Th1 cells) subset in regressive group compared to control group. Following MTX cessation, significant increase in proportion and absolute number of these subsets were observed only in the regressive group, but not in persistent group. Expansion of Th1 cells and EMCD8+ T cells significantly correlated with increase of serum IFN-γ, and expansion of EMCD8+ T cells inversely correlated with change of serum IL-15.

Conclusions: Restoration in proportion and absolute number of Th1 cells, EMCD8+T cells and EBV specific CD8+ T cells coincided with increase of IFN-γ, and associated with regression of LPD developed under MTX administration. Since changes of those immunological factors were not observed in persistent LPD, this study would be the first report to demonstrate the difference of

immune status between regressive and persistent LPDs developed under MTX administration.

References:
[1] Saito S, et al. Rheumatology. 2017; in press.
[2] Inui Y, et al. Leuk Lymphoma. 2015; 56(11):3045–3051.

FRI0169 | **GLOMERULAR FILTRATION RATE IS LOW IN RHEUMATOID ARTHRITIS COMPARED TO HEALTHY POPULATION: ESSENTIAL ROLE OF INFLAMMATION**

S. Hannawi[1], I. AlSalmi[2]. [1]*Rheumatology, Ministry of Health and Prevention of UAE, Dubai, United Arab Emirates;* [2]*Nephrology, Royal Hospital, Muscat, Oman*

Background: Rheumatoid Arthritis is associated with subclinical renal impairment which contributes to increase mortality and morbidity. The role of inflammation on kidney function in inflammatory arthritis is not well studied.

Objectives: To investigate the associations between estimated glomerular filtration rates (eGFR), traditional cardiovascular risk factors, and markers of inflammation in rheumatoid arthritis compared to healthy controls.

Methods: RA patient were recruited through a specialized rheumatology clinic at the Ministry of Health and Prevention of UAE, from January 2013 to January 2016. Healthy subjects recruited from the community through brochure advertisement. The Modification of Diet in Renal Disease Study (MDRD) formula was used to calculate the eGFR. ttest was used to compare the laboratory values and renal function parameters between two groups. Linear regression analysis used to look for the correlation between the eGFR and each of the traditional cardiovascular risk factors and inflammatory markers

Results: 98 RA patients and 82 controls were recruited. None of the patients has history of diabetes, atherosclerosis or renal impairment. The mean age for the total participants was 49±13 years (Min16 –Max 82). The mean eGFR of the inflammatory arthritis patients was 118±30 ml/min (range 60–227) and 128±37 ml/min (range 62–286) for the controls. Patients and control had no significant difference in Systolic and diastolic blood pressure.

Inflammatory arthritis patients had lower GFR, albumin (P<0.001), and total protein (p=0.03) levels, and had higher Erythrocyte Sedimentation Rate (ESR) (P<0.001), C-reactive protein (CRP) (P<0.001), and uric acid level (p=0.01), Negative linear relationships were found as follows:

Among RA patients and controls: There was a negative linear relationship between GFR and each of the age of the participants; (p<0.001, CI: -1.24, -0.40 for the patients and p=0.01, CI: -1.82, -0.26 for the controls), and the systolic blood pressure; (p=0.04, CI: -0.61 for the patients and, -.00 and p=0.022, CI: -0.61, -0.05 for the controls).

Among RA patiens: The GFR had a negative linear relationship with the age of the participants, age at RA onset (p=0.002, CI: -1.18, -0.29), diastolic blood pressure (p=-2.14, CI: -1.24, -0.05), ESR level (p=0.04, CI: -0.24,-0.01), C-reactive protein; CRP level (p=0.02, CI: -0.47, -0.04), uric acid level (p<0.001, CI -0.18, -0.05), and total protein (p=0.01, CI: -0.91, -0.16). There was a positive linear relationship between eGFR and albumin level (p=0.14, 2.35),

Conclusions: In RA non-traditional cardiovascular risk factors such as inflammatory markers are associated with sub-clinical presence of renal injury. Our data indicate that in RA, inflammation is involved in the early stages of impaired kidney function. Whether anti-inflammatory therapies are effective in slowing down the deterioration of kidney function in the arthritis diseases remain to be established.

References:
[1] Puttevils, D., et al., Increased cardiovascular risk in patients with rheumatoid arthritis: an overview. Acta Cardiol, 2014. 69(2): p. 111–118.
[2] Nordin, H. and L.M. Pedersen, Kidney function problems in rheumatoid arthritis. Ugeskr Laeger, 1996 158(22): p. 3137–40.

FRI0170 | **CARDIOVASCULAR RISK FACTORS AND DISEASE CHARACTERISTICS ARE CONSISTENTLY ASSOCIATED WITH ARTERIAL STIFFNESS IN RHEUMATOID ARTHRITIS**

S. Gunter[1], C. Robinson[1], G. Norton[1], A. Woodiwiss[1], L. Tsang[1], A. Millen[1], P. Dessein[1,2]. [1]*Cardiovascular Pathophysiology and Genomics Research Unit, University of Witwatersrand, Johannesburg, South Africa;* [2]*Rheumatology Division, Universitair Hospital Brussels, Brussels, Belgium*

Background: In the non-rheumatoid arthritis (RA) population, arterial stiffness contributes to cardiovascular disease risk beyond brachial blood pressure and other established cardiovascular risk factors. The increased cardiovascular disease risk in RA is now well documented. In this regard, how RA impacts on arterial stiffness remains uncertain.

Objectives: The aim of the present study was to identify potential determinants of comprehensively assessed arterial stiffness in a relatively large group of ethnically diverse patients with RA.

Methods: Relationships of traditional cardiovascular risk factors and RA characteristics with 9 arterial stiffness markers including central systolic and pulse pressure, pulse wave velocity, augmentation index, forward and reflected wave pressure, reflection magnitude, brachial-to-aortic pulse pressure amplification (a marker of reduced wave reflection) and peripheral pulse pressure were identified in multivariable backward regression models among 177 (118 white, 32 Asian, 22 black, 5 mixed ancestry) patients without established cardiovascular disease.

Results: Recorded characteristics explained 37% (pulse wave velocity) to 71% (reflected wave pressure) of the variability in arterial stiffness. RA duration (partial r=0.17, p=0.04), rheumatoid factor status (partial r=-0.19 to 0.20, p=0.01 to 0.03), leukocyte counts (partial r=0.16 to 0.19, p=0.02 to 0.05) and total cholesterol (-0.18 to 0.26, p=0.00 to 0.03) were associated with enhanced central systolic blood pressure and/or wave reflection markers. C-reactive protein (partial r=-0.24, -0.17 and -0.20, respectively, p≤0.05) was paradoxically related to reduced central pulse pressure, pulse wave velocity and forward wave pressure, and body mass index (partial r=-0.39 to 0.42, p=0.00 to 0.02) and insulin resistance (partial r=-0.21 to -0.20, p=0.00 to 0.01) to reduced wave reflection and peripheral pulse pressure. Exercise (partial r=0.19, p=0.02) and alcohol (partial r=-0.27, p=0.00) consumption were associated with increased pulse pressure amplification and decreased peripheral pulse pressure, respectively. Tumour necrosis factor-α inhibition (partial r=-0.25, p=0.00) was related to reduced pulse wave velocity and tetracycline use (partial r=-0.20, p=0.02) to reduced peripheral pulse pressure.

Conclusions: Traditional cardiovascular risk factors and disease characteristics are consistently associated with vascular hemodynamic alterations in RA. The role of arterial stiffness in cardiovascular disease risk in RA needs further study.

FRI0171 | **OBESITY CONTRIBUTES TO SUBOPTIMAL PHYSICAL FUNCTION IN RHEUMATOID ARTHRITIS**

A. Sirois[1], N. Chiarlitti[1], M. Inceer[1], M. Jones[2], C.O. Bingham III[2], S.J. Bartlett[1]. [1]*McGill University, Montreal, Canada;* [2]*Johns Hopkins, Baltimore, United States*

Background: Aggressive, early treatment of RA with new therapeutic agents has dramatically improved the management of RA. However, many studies have failed to show greater improvements in function or disability reduction between targeted control vs. less-aggressive care. The prevalence of obesity is increasing dramatically globally, and may be even higher in RA patients.[1]

Objectives: Because obesity is also a risk factor for disability, we evaluated the extent to which excess weight may independently contribute to poorer physical function (PF) in RA.

Methods: RA patients enrolled in an observational study at an academic Inflammatory Arthritis Clinic in Baltimore USA completed the patient global, pain VAS, and PROMIS measures assessing PF, pain, fatigue, sleep, and depression. RA clinical indicators were also collected at the visit. Outcomes were compared in obese and non-obese patients using t-tests and chi-square. Multiple regression was used to evaluate the effects of pain, fatigue, and BMI on PF, after controlling for age and disease activity.

Results: Participants were mostly female (82%) and white (83%) with mean (SD) age of 55 (13) years; 24% had ≤ high school; RA duration 12 (9). Mean CDAI was 8.1 (8.1). Most were in CDAI remission (n=57; 32%) or LDA (n=64; 36%); 40 (23%) were in MDA and 16 (9%) in HDA. 49 (28%) were classified as normal weight (BMI 18.5–24.9), 46 (26%) were overweight (BMI 25–29.9), and 82 (46%) were obese (BMI≥30). Men had a significantly higher mean BMI than women (33.6 [8.4] vs. 29.5 [7.2], p=0.006).

As compared to non-obese participants, obese participants had a significantly (p<.05) higher CDAI (6.1 [6.9] vs. 10.4 [8.9]; p=.000, respectively) and worse PF, pain, fatigue, sleep, and depression (mean differences -5.0, -4.6, 4.6, 3.8, 2.9, 3.6, and -4.7, respectively). In regression analyses, pain, fatigue, and BMI (but not sleep or depression) were inversely related to PF, after controlling for age and disease activity. In the final model, pain, fatigue and BMI were significantly and inversely related to PF (β=-.39, -.24, and -.153, respectively) after controlling for age and disease activity (F (5, 170) =55.5, p=.000, adjusted r²=.61).

Table. Independent Contributors of Function in RA.						
	Coefficient			Std	Collinearity	
	B	SE	Beta	Sig.	Tol	VIF
Pain	-.386	.064	-.405	.000	.500	2.001
Fatigue	-.240	.060	-.266	.000	.509	1.965
BMI	-.153	.058	-.128	.010	.932	1.073
Adjusted for age and CDAI.						

Conclusions: Our results suggest that excess weight also contributes to poorer PF in addition to pain and fatigue. As the prevalence of obesity continues to