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

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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=0.03, 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.

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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 or/and wave reflection markers. C-reactive protein (partial r=-0.24, -0.17 and -0.20, respectively, $p\leq0.05$) 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.

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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 $\leq$ 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$\geq$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 r^2=.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

escalate in RA populations, weight loss may be increasingly important to improve not only physical health, disease activity, and response to treatment, but also pain, fatigue, and PF.

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FRI0172 PREVALENCE AND FEATURES OF THE "MASKED" ARTERIAL HYPERTENSION IN WOMEN WITH RHEUMATOID ARTHRITIS WITHOUT CARDIOVASCULAR DISEASES BASED TO AMBULATORY BLOOD PRESSURE MONITORING

T.A. Romanova, N.M. Nikitina, A.P. Rebrov. *Hospital Therapy Department, State Medical University of Saratov, Saratov, Russian Federation*

Background: Cardiovascular (CV) events are the main reason of increased mortality at patients with rheumatoid arthritis (RA). Arterial hypertension (AH) takes a leading position among CV risk factors in RA. Ambulatory blood pressure monitoring (ABPM) has to be executed for persons with high cardiovascular risk and can be used for evaluation of "masked" arterial hypertension (MAH), according to the current recommendations.
Objectives: to estimate the frequency of identification and peculiarities of the MAH according to ambulatory blood pressure monitoring in women with RA without CV diseases.
Methods: The study included 36 women with RA (ACR 1987 and/or EULAR/ACR 2010 criteria) without CV diseases and AH (according to the anamnesis and 3-fold "office" blood pressure (BP) measurement). Mean age of RA patients was 55±7.15 years; mean duration of RA was 10 [3; 17] years, mean DAS 28 – 5.25 [4.6; 5.7]. As controls were involved 39 women with RA and AH (mean age- 58.3±6.08 years; mean duration of RA - 8 [4;14] years, mean DAS 28- 5.08 [4.04; 5.85]) and 30 women with AH without inflammatory joint diseases (mean age 55.9±6.2). Exclusion criteria were smoking, diabetes mellitus, symptomatic AH (except controls), cardiovascular diseases.
ABPM was measured using the BPlab with the program VASOTENSE (Russian). Criteria for MAH were out-of-office BP $\geq$135/85 mmHg and/or average daily out-of-office BP $\geq$130/80 mmHg according to ABPM and considering optimal office BP.
The anamnesis, laboratory and instrumental methods of inspection were considered. Statistical analyze was performed with STATISTICA 7.0 (State Soft, USA).
Results: According to ABPM 24 of the 36 (66.6%) RA patients had optimal BP, 12 (33.3%) patients had the MAH phenomenon.
The patients with RA + AH and AH patients had the comparable levels of a daily BP [132.1/80.7 and 126.9/78.0 mmHg respectively, $p>0.05$].
The RA patients with MAH had statistically significant differences of BP in day and night hours [137.58/87 mmHg and 137/84 mmHg, respectively] compared to the RA + AH patients [132.1/80.7 mmHg and 128.3/73.8 mmHg, respectively, p=0.004] and AH patients [126.9/78 and 118/68.5 mmHg, respectively, p=0.001], what it is can be bound to absence of anti-hypertensive therapy at RA patients with MAH.
Nocturnal systolic BP correlated with ESR (Spearmen's r=0.64, $p<0.05$). Daily diastolic BP interrelated with SCORE/EULAR (r=0.86, $p<0.05$) and arterial stiffness index (r=0.86, $p<0.05$) in RA patients with MAH; nocturnal systolic BP correlated with C-reactive protein (r=0.36, $p<0.05$) in RA+AH patients.
More than 60% of the RA+AH patients and RA+MAH had high variability of BP. High variability of nocturnal systolic BP occurred at 34.3% and 44.4% of RA+AH and RA+MAH patients, respectively.
In RA patients were found correlations between indices of a BP variability, duration of RA (r=0.33, $p<0.05$); C-reactive protein (r=0.36, $p<0.05$); daily diastolic BP (r=0.35, $p<0.05$); pulse BP (r=0.31, $p<0.05$); and frequency of non-steroidal anti-inflammatory drugs intake (r=0.42, $p<0.05$).
Conclusions: In RA patients AH can proceed subclinically. Activity of RA and increased arterial stiffness can predict the masked arterial hypertension's development in RA patients.
ABPM measurement can be use full in early evaluation of AH and optimization of RA treatment.

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FRI0173 CORRELATION BETWEEN ASSYMETRIC DIMETHYLARGININE AND HOMOCYSTEINE LEVELS IN PATIENTS WITH RHEUMATOID ARTHRITIS

T. Dimitroulas[1,2], J. Hodson[3], A. Sandoo[4], K. Douglas[1], J. Smith[1], G. Kitas[5]. *[1]Department of Rheumatology, Dudley Group of Hospitals NHS Trust, Russells Hall Hospital, Dudley, United Kingdom; [2]4th Department of Internal Medicine, Aristotle University of Thessaloniki, Thessaloniki, Greece; [3]Wolfson Computer Laboratory, University Hospital Birmingham NHS Foundation Trust, Queen Elizabeth Hospital Birmingham, Mindelsohn Way, Birmingham; [4]School of Sport, Health and Exercise Sciences, Bangor University, George Building, Bangor; [5]Arthritis Research UK Epidemiology Unit, University of Manchester, Manchester, United Kingdom*

Background: Rheumatoid Arthritis is a chronic inflammatory condition associated with increased cardiovascular disease (CVD) morbidity and mortality due to high coronary and cerebral atherosclerotic burden. Assymetric dimethylarginine (ADMA) – the most potent endogenous inhibitor of nitric oxide synthase - is an emerging marker of endothelial dysfunction and CVD in several conditions such as coronary artery disease and diabetes mellitus. ADMA levels are higher in RA patients compared to controls suggesting a role in the development of atherosclerosis. In addition ADMA is involved in the metabolism of honocysteine (Hcy) which is considered a novel, non-traditional CVD risk factor contributing to excess CVD risk in this population.
Objectives: The aim of our study was to determine whether asymmetric dimethylarginine (ADMA) levels are associated with Hcy and methylenetetrahydrofolate reductase (MTHFR) C677T (rs1801133) gene variants in patients with RA.
Methods: Serum ADMA and Hcy levels were measured in 201 RA individuals [155 (77.1%) females, median age 67 years (interquartile range 59–73)]. The MTHFR C677T polymorphism was assessed by using the LightCyclerTM System. Initially, ADMA was compared across the categories of MTHFR using a one-way analysis of variance (ANOVA), followed by a multivariate model, which accounted for Hcy, age, erythrocyte sedimentation rate (ESR), and homeostatic model assessment (HOMA).
Results: In univariable analysis, ADMA differed significantly across the categories of MTHFR (p=0.037). Patients with the MTHFR 677TT genotype had the highest ADMA levels, with a mean of 0.62 (SE =0.03), significantly higher than either those patients carrying the MTHFR 677CT (0.55, SE =0.01) or the MTHFR 677CC (0.55, SE=0.01) genotype (p=0.042) in both cases. In the multivariable model, Hcy (p=0.022) and ESR ($p<0.001$) were found to have significant positive associations with ADMA but the relationship between MTHFR gene variants and ADMA was found to be non-significant (p=0.102).
Conclusions: The results of our study indicate an association between ADMA and Hcy in patients with RA without any genetic background as no relationship was established between ADMA and MTHFR gene variants. Abnormal metabolism of Hcy and dimethylarginines may interfere with each other resulting in endothelial dysfunction and accelerated atherosclerosis in RA individuals.

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FRI0174 A POPULATION BASED COHORT STUDY OF RHEUMATOID ARTHRITIS-ASSOCIATED INTERSTITIAL LUNG DISEASE: COMORBIDITY AND MORTALITY

C. Hyldgaard[1], O. Hilberg[2], A.B. Pedersen[3], S.P. Ulrichsen[3], A. Løkke[1], E. Bendstrup[1], T. Ellingsen[4]. *[1]Department of Respiratory Diseases, Aarhus University Hospital, Aarhus C; [2]Department of Medicine, Vejle Hospital, Vejle; [3]Department of Clinical Epidemiology, Aarhus University Hospital, Aarhus; [4]Department of Rheumatology, Odense University Hospital, Odense, Denmark*

Background: Interstitial lung disease (ILD) is an important reason for excess mortality among patients with rheumatoid arthritis.
Objectives: The aim of this study was to compare mortality risks in patients with rheumatoid arthritis-associated interstitial lung disease (RA-ILD) and patients with RA without ILD.
Methods: The study was conducted in Denmark, using nationwide, prospectively collected data from the national medical registries. Among patients with RA, diagnosed between 2004 and 2016, 679 patients with RA-ILD were matched for age, gender, and age at RA diagnosis with 11,722 patients with RA but without ILD. Mortality risks were assessed using Kaplan-Meier survival curves, and hazard rate ratios (HRR) for death were estimated using Cox proportional hazards regression models.
Results: The number of prevalent RA patients more than doubled from 15,352 to 35,362 individuals during the study period. RA-ILD was seen in 2.2% of incident RA patients. 34.0% of RA-ILD cases were diagnosed within one year prior to and one year after the RA diagnosis. One-year mortality was 13.9% in RA-ILD and 3.8% in non-ILD RA, three-year mortality was 28.0% and 10.9%, and five-year mortality was 39.0% and 18.2%, respectively. The HRRs for death were two to 10 times increased for RA-ILD compared with non-ILD RA, irrespective of follow-up period. Stratified analysis showed that the HRR for death was highest in the first months after the diagnosis of RA-ILD was made, especially in patients diagnosed with RA before diagnosis of ILD. HRR was higher in males and in patients without comorbidity as assessed by the Charlson Comorbidity Index.