Astra-Zeneca, Astro, BMS, Celgene, Celltrion, Chugai, Gilead Glaxo, ILTOO, Janssen, Eli Lilly and Company, Medimmune, MSD, Novartis-Sandoz, Pfizer, Roche, Samsung, Sanofi-Aventis, UCB, G. Burmester Consultant for: Eli Lilly and Company, C. Walls Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, W.-S. Wu Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, C. Dickson Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, R. Liao Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, M. Genovese Grant/research support from: Eli Lilly and Company, AbbVie, Consultant for: Eli Lilly and Company, AbbVie,
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FRI0078 PROSPECTIVE FOLLOW-UP OF A COHORT OF PATIENTS WITH INTERSTITIAL LUNG DISEASEASSOCIATED WITH RHEUMATOID ARTHRITIS IN TREATMENT WITH DMARD

N. Mena-Vázquez[1], C. Gomez-Cano[2], L. Perez-Albaladejo[3], S. Manrique-Arija[1], C. Romero-Barco [4], C. Aguilar-Hurtado[5], I. Ureña-Garnica[6], F. Jiménez-Núñez[1], M. Ordóñez-Cañizares [1], C. Fuego[1], M. Padin-Martin[5], R. Caliz-Caliz[3], A. Fernández-Nebro.[7]. *[1]UGC de Reumatología, Instituto de Investigación Biomédica de Málaga (IBIMA) Hospital Regional Universitario de Málaga, Universidad de Málaga., Málaga*; *[2]Department of Rheumatology, Hospital Virgen de Valme, Sevilla*; *[3]Department of Rheumatology Hospital Virgen de las Nieves., Granada*; *[4]UGC de Reumatología, Instituto de Investigación Biomédica de Málaga (IBIMA), Hospital Clínico Virgen de la Victoria, Universidad de Málaga*; *[5]Department of Radiology Hospital Regional de Málaga*; *[6]Department of Rheumatology at the University Regional Hospital of Malaga (HRUM). Institute for Biomedical Research in Malaga (IBIMA). Malaga University*; *[7]UGC de Reumatología, Instituto de Investigación Biomédica de Málaga (IBIMA) Hospital Regional Universitario de Málaga, Departamento de Medicina y Dermatología, Universidad de Málaga., Málaga, Spain*

Objectives: To describe prospectively the evolution of interstitial lung disease (ILD) in RA treated with modifying antirheumatic drugs (DMARDs) in clinical practice.
Methods: Design: Multicenter prospective observational cohort. Patients: Patients with RA (ACR/$^{EULAR\ 2010}$ criteria) and ILD (American Thoracic Society) from different centres of Málaga, Valme Hospital of Sevilla and Virgen Nieves of Granada were included. Protocol: All patients with RA and ILD who visited clinic from 2015 to 2017 were recruited. They were reviewed according to a predetermined protocol for data collection. Resolution Computed Tomography (HRCT), Pulmonary function test (PFT) and echocardiogram were requested for all patients who did not have it in the last year. This visit was marked as v0 (index date). At 12 months (v12) the joint assessment (DAS28), echocardiogram, PTF and HRCT were again evaluated. HRCT's were assessed by the same radiologist. Outcomes: At v12:([1]improvement (ie improvement in FVC ≥10% or DLCO ≥15% and no radiological progression),[2] non-progression (stabilisation or improvement in FVC ≤10% or DLCO <15% and no radiological progression),([3] progression (worsening of FVC >10% or DLCO >15% and radiological progression), or[4] death due to ILD. Variables: Description of ILD type and lung function by PTF, HRCT. Presence of PTH by echocardiogram and dyspnoea. Disease activity by DAS28-ESR;Adverse events during the follow-up period. Statistical analysis: Descriptive analysis and Wilcoxon or T test between the v0 and v12. One factor ANOVA between sDMARD, bDMARD and combination therapy groups.
Results: The main characteristics at V0 of the patients (n=41) are shown in the table 1. Nine patients (21.9%) received a sDMARDs with a bDMARDs;25 patients (60.9%) monotherapy with sDMARD and 7 (17.0%) monotherapy with bDMARDs (table 1). Nine patients (21.9%) had improvement (2 with MTX, 1 with MTX +HCQ, 2 with RTX, 2 HCQ +RTX, 1 MMF +RTX and 1 with ABA); 24 patients (58.5%) remained stable (6 with MTX, 6 with LFN, 3 with HCQ, 1 AZA, 1 SSZ, 1 MMF, 1 TCZ, 2 ABA, 1 MTX +ETN, 1 HCQ +RTX, 1 HCQ +ADA, 1 RTX +MMF); and 7 (17.0%) got worse of ILD (2 with MTX developed lung nodules not known, 2 with LFN, 1 with LFN +IFX, 1 with ETN +MTX and 1 with SSZ). One patient died due to respiratory infection (with RTX). Two patients developed PPH. We did not find significant differences between Vo DAS28 and v12 (2.61 [0.74] vs 2.54 [1.12]; p=0.684) or in HAQ (1.12 [0.89] vs 1.23 [0.73],p=0.368). There were no significant differences in PTF, HRCT or DAS28 between sDMARD, bDMARD and combination therapy groups. During the follow-up period 27 patients had infections, the majority (53.7%) respiratory infection.

VARIABLES	Patients
Sex (male), n (%)	21 (51.2)
Age (years), mean (DE)	67.9 (7.8)
Smoker, n (%)	8 (19,5)
Ex-smoker, n(%)	32 (78)
Body mass index (BMI), mean (SD	28.7 (5.3)
Disease duration (months), median (p25-p75)	138.1 (77.7-285.3)
ILD duration (months), median (p25-p75)	41.4 (10.1-79.2)
Rheumatoid factor, n (%)	39 (95.1)
Anti–cyclic citrullinated peptide, n (%)	35 (85.4)
Erosions, n (%)	27 (66)
DAS28, mean (DE)	2.8 (0.8)
HAQ, mean (DE)	1.2 (0.8)
Treatment	
sDMARD, n (%)	34 (82.9)
Methotrexate, n (%)	13 (31.7)
Leflunomide, n (%)	9 (22.0)
Sulfasalazine, n (%)	2 (4.9)
Hydroxychloroquine, n (%)	6 (14.6)
Azathioprine	1 (2.4)
Micofenolato	3 (7.3)
MTX+HQC	2 (4.9)
bDMARDs, n (%)	16 (39.0)
Rituximab, n (%)	7 (17.1)
Abatacept, n (%)	3 (7.3)
Etanercept, n (%)	3 (7.3)
Infliximab, n (%)	1 (2.4)
Adalimumab, n (%)	1 (2.4)
Tocilizumab, n(%)	1 (2.4)

Conclusions: Most patients with RA and ILD who are receiving treatment with DMARD (80.5%) remained stable or improved after at least one year of both synthetic and biological DMARD treatment. More prospective studies are necessary to identify the influence of DMARDs in this evolution.
Disclosure of Interest: None declared
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FRI0079 ADIPONECTIN LEVEL, INSULIN RESISTANCE, ENDOTHELIAL DYSFUNCTION IN FEMALES WITH RHEUMATOID ARTHRITIS AND COMORBID HYPERTENSION

O. Sirenko, O. Kuryata, T. Lysunets. *State Establishment «Dnipropetrovsk medical academy of Health Ministry of Ukraine», Dnipropetrovsk, Ukraine*

Background : *Rheumatoid arthritis (RA) associates with accelerated atherosclerosis and high cardiovascular mortality. Cardiovascular risk assessment in RA pts with comorbid hypertension (HT) is do not fully reflected by traditional risk scales, thus additional factors searching is required.*
Objectives: We aimed to estimate the adiponectin level, insulin resistance, endothelial function in RA females with comorbid HT and its relationship with subclinical manifestations of atherosclerosis.
Methods: The study included 82 RA females with low disease activity and comorbid HT (mean age of 54.6 [49.7; 62.5] years) and 40 HT females without RA (control group). All pts received stable therapy of RA more than 6 months. Pts with coronary artery disease were excluded. The risk of fatal cardiovascular disease was calculated using mSCORE. RA disese activity was measured using DAS28 scale. Carotid ultrasound detection and endothelial-dependent flow mediated vasodilatation (EDVD) by Celermajer method were performed. The levels of adiponectin, insulin were measured using ELISA kit test, insulin resistance was estimated using HOMA2 index.
Results: Endothelial dysfunction was established in the majority of main group patients – 61 (74.4%), insulin resistance – in 70 (85.4%), elevated levels of adiponectin – in 35 (42.7%). Hypertensive females with RA had significantly higher adiponectin, insulin, insulin resistance levels compare to control (p<0.05). *Subclinical manifestations of atherosclerosis were established in* 64 (78.0%) *HT females with RA and* 10 (50%) *control group pts. While the median cardiovascular risk level was* 4.2 [2.7; 6.5]% match*ed by mSCORE.* The presence of atherosclerotic plaques in HT females with RA was associated with age (OR=1.242, p=0.004, 95% CI 1.007–1.78), glucocorticosteroid therapy >3 months (OR=1.56, p=0.001, 95% CI 1.22–2.45), endothelial dysfunction (OR=3.584, p=0.001, 95% CI 1.71–4.723), insulin resistance (OR=1.684, p=0.011, 95% CI 1.22–2.74), abnormal adiponectin level (OR=1.71, p=0.028, 95% CI 1.17–2.43). *AUROC index for prognostic role of adiponectin and HOMA2 in subclinical atherosclerosis develop were 0.79 (95% CI 0.64–0.95*; p*<0.05) and 0.76 (95% CI 0.61–0.92*; p*<0.05) respectively, that indicate a good quality of diagnostic models.*

Conclusions: Hypertensive females with rheumatoid arthritis are characterised by higher frequency of insulin resistance, endothelial dysfunction, adiponectin level changes which associates with subclinical atherosclerosis manifestations.
Disclosure of Interest: None declared
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FRI0080 THE ROLE OF PAIN IN RHEUMATOID ARTHRITIS (RA) PATIENTS' ASSESSMENTS OF THEIR HEALTH

P. Katz[1], Y.C. Lee[2], A. Quebe[3], L. Sun[3], H. Patel[3], C. Gaich[3], N. Boytsov[3], K. Michaud[4,5]. [1]*Medicine, University of California, San Francisco, San Francisco*; [2]*Northwestern University, Chicago*; [3]*Eli Lilly and Co, Indianapolis*; [4]*Forward/NDB, Wichita*; [5]*University of Nebraska Medical Center, Omaha, USA*

Background: Patients often describe pain as the most important symptom of RA. Despite advances in RA therapy to improve disease control, some patients continue to have significant pain[1,2]. The relative impact of pain on RA patients' evaluations of overall health and RA-specific global assessments is unknown.
Objectives: Determine the relative role of pain in RA patients' health assessments.
Methods: Data derived from the Forward (The National Databank for Rheumatic Diseases) longitudinal cohort, collected January-May 2017. Respondents (n=5471) have rheumatologist-confirmed RA. Two health assessments were examined:[1] overall satisfaction with health (SAT) measured by the item: "How satisfied are you with your health now?" with responses of very unsatisfied to very satisfied; and[2] patient global assessments of RA impact (GBL) measured using a numeric rating scale (NRS): "Considering all the ways that your RA affects you, rate how you are doing on a scale of 0–10, where 0=very well and 10=very poor." For regression analyses, SAT was dichotomized as "very satisfied" or "somewhat satisfied" vs. other responses. Current pain severity was rated on an NRS from 0 (no pain) to 10 (extreme pain). Spearman correlations examined the association of pain with SAT and GBL. Initial multiple regression analyses (table 1, Model 1) examined the following as predictors of SAT and GBL: age, sex, education, disease duration, obesity (BMI ≥30), conventional and biologic DMARD use, Rheumatic Disease Comorbidity Index[3] (RDCI), self-report of depression, fatigue, and functional limitations (Health Assessment Questionnaire [HAQ] score). Follow-up models (Model 2) added pain to determine its relative independent role in health assessments.
Results: The sample was 84% female, mean age 65 years, mean RA duration 22 years. 53% were satisfied with their health, and mean GBL was 3.6±2.5. Mean pain severity rating was 3.8±2.8. Correlations of pain with SAT and GBL were 0.58 and 0.71, respectively (each p<0.0001). Regression models predicting both SAT and GBL improved with the addition of pain (table 1). Pain was significantly and independently associated with both health assessments.

	Model 1 (excluding pain)	Model 2 (adding pain)
Satisfied with health		
Pain		0.81 (0.79, 0.84)*
Model R^2	0.37	0.40
Global assessment of RA		
Pain		0.33 (<0.0001)†
Model R^2	0.51	0.57

*Odds ratio (95% CI)
† b (p-value)

Regression models included age, sex*, education, disease duration, obesity, medications, RDCI, depression, fatigue*, and HAQ*. Variables noted with asterisk were also statistically significant (p<0.05) in both final models.
Conclusions: Pain plays a critical role in RA patients' assessments of general and RA-specific health. Analyses suggest that pain may be more important to RA global assessments than to overall health satisfaction, though the clinical relevance of this difference is not known. RA global assessments are included in some indices of disease activity. Future research should focus on distinguishing between non-inflammatory and inflammatory causes, which may lead to more accurate assessment of RA disease activity.

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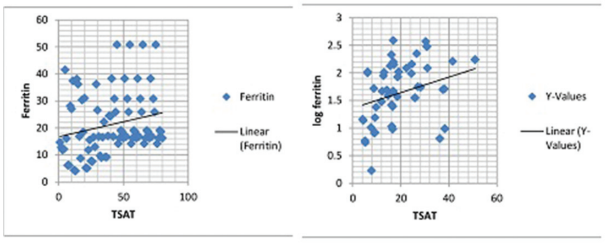
FRI0081 THE IMPORTANCE OF TRANSFERRIN SATURATION, SERUM FERRITIN, LOG FERRITIN AND TRANSFERRIN/ LOG FERRITIN IN DIFFERENTIATING IRON DEFICIENCY ANAEMIA FROM ANAEMIA OF CHRONIC DISEASE IN RHEUMATOID ARTHRITISPATIENTS

R.A. Abdel Noor[1], M.H. Abu-Zaid[2], S.A. Elshweikh[3], E.S. Rabee[4], G.E. Khedr[5]. [1]*Internal Medicine and Rheumatology*; [2]*Physical medicine, Rheumatology and Rehabilitation*; [3]*Internal Medicine and Hematology*; [4]*Clinical pathology, Faculty of Medicine, Tanta University*; [5]*Clinical pathology, Tanta Cancer Center, Tanta, Egypt*

Background: The most common types of anaemia in rheumatoid arthritis (RA) are iron deficiency anaemia (IDA) and anaemia of chronic disease (ACD). The differentiation between both is very important and challenging.
Objectives: is to select the most simple, cheap, accurate method differentiate IDA from ACD in RA patients.
Methods: This case control study was carried out on 80 RA patients. Group I 40 RA patients with anaemia Group II 40 RA patients without anaemia, complete blood count, assessment of disease activity using DAS 28 score, serum iron, total iron binding capacity (TIBC) "transferrin level", transferrin saturation (TSAT), serum ferritin, log ferritin and transferrin/log ferritin were tested, then we divided the patients in group 1 into 2 subgroups according to TSAT : group Ia (RA patients with anaemia and low TSAT) and group Ib (RA patients with anaemia and normal TSAT) and we compared the parameters.
Results: There was a statistically significant difference between anaemic and non anaemic RA patients as regard serum iron level and transferrin saturation and there was no significant difference as regard serum ferritin, log ferritin, transferrin and transferrin/log ferritin. Among the anaemic group (67.5%) had low TSAT (IDA) and only (32.5%) had normal TSAT (ACD). In these 2 subgroups there was no significant differences as regard DAS28 score, blood indices, serum ferritin and transferrin/log ferritin) except Log ferritin and there was positive correlation between TSAT and (ferritin and log ferritin) and significant negative correlation between TSAT and transferrin/log ferritin.

Abstract FRI0081 – Table 1. Clinical and laboratory parameters in both groups

Characteristic	Group I[40] RA +anaemia	Group II[40] RA without anaemia	P
DAS-28	2.99±1.07	2.35±0.77	0.002*
Hb (g/dl)	9.62±0.96	12.95±0.64	<0.0001*
MCV (fl)	79.71±7.29	83.0±9.34	0.083
MCH (pg/cell)	26.06±4.05	28.05±2.66	0.006*
MCHC (g/dl)	31.1±2.21	31.23±2.38	0.8
Serum iron (µg/ml)	0.66±0.37	0.89±0.38	0.0076*
Serum ferritin (ng/ml)	83.02±93.08	84.87±86.82	0.927
TIBC (µg/ml)	3.94±1.06	3.71±0.59	0.23
TSAT (%)	17.7±10.6	24.73±11.36	0.0054*
Log ferritin	1.61±0.75	1.7±0.46	0.52
Transferrin/Log ferritin	3.23±3.8	2.41±0.9	0.18



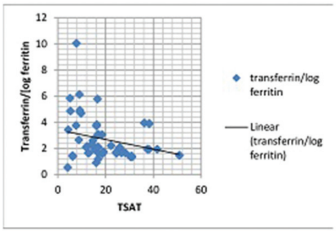


Figure 1 Correlation between TSAT and (serum ferritin, log ferritin and Transferrin/Log ferritin)