targeted-synthetic DMARDs. The use of any conventional synthetic DMARD was associated with lower HAQ-DI indices ($p: 0.0243$), while the use of any biologic DMARD or targeted-synthetic DMARD was related to greater functional impairment ($p: 0.0018$). By evaluating separately, abatacept ($p: 0.0046$), rituximab ($p: 0.0001$) and tocilizumab ($p: 0.0441$) were associated with higher levels of HAQ-DI. The results are summarised in table 1.

Abstract AB0200 – Table 1. Health assessment questionnaire-disability index score position and dispersion measurements and result of comparison between groups

Variable	Category	N	Mean	SD	Median	p-value
Rheumatoid factor	High	612	0.98	0.80	0.88	0.0244 ^c
	Low	247	0.89	0.73	0.75	
	Negative	235	0.82	0.74	0.75	
ACPs	High	262	0.96	0.77	0.88	0.3927 ^c
	Low	86	0.89	0.84	0.63	
	Negative	111	0.88	0.75	0.68	
Bone erosion	No	493	0.77	0.72	0.63	<0.0001 ^a
	Yes	599	1.06	0.80	1.00	
Methotrexate	No	373	0.96	0.80	0.88	0.4028 ^b
	Yes	739	0.91	0.76	0.75	
Lefunomide	No	735	0.93	0.77	0.88	0.8936 ^b
	Yes	377	0.93	0.78	0.88	
Hydroxychloroquine	No	993	0.94	0.78	0.88	0.2696 ^b
	Yes	119	0.85	0.75	0.75	
Sulfasalazine	No	1,058	0.93	0.78	0.88	0.6727 ^b
	Yes	54	0.87	0.75	0.81	
Conventional synthetic DMARDs	No	115	1.12	0.89	1.00	0.0243 ^b
	Yes	997	0.91	0.76	0.75	
Adalimumab	No	1,059	0.92	0.77	0.88	0.7728 ^b
	Yes	53	1.00	0.86	0.75	
Infliximab	No	1,064	0.93	0.77	0.88	0.8659 ^b
	Yes	48	0.94	0.78	0.81	
Etanercept	No	1,046	0.94	0.77	0.88	0.1317 ^b
	Yes	66	0.79	0.75	0.69	
Certolizumab pegol	No	1,095	0.93	0.78	0.88	0.3074 ^b
	Yes	17	0.72	0.67	0.50	
Golimumab	No	1,075	0.93	0.78	0.88	0.8372 ^b
	Yes	37	0.85	0.70	1.00	
Tocilizumab	No	1,058	0.92	0.78	0.75	0.0441 ^b
	Yes	54	1.08	0.66	1.00	
Abatacept	No	1,040	0.91	0.77	0.75	0.0046 ^b
	Yes	72	1.16	0.78	1.00	
Rituximab	No	1,063	0.91	0.77	0.75	0.0001 ^b
	Yes	49	1.34	0.77	1.00	
Tofacitinib	No	1,103	0.93	0.77	0.88	0.9211 ^b
	Yes	9	0.93	0.76	0.75	

Conclusions: In our prospective cohort, patients with high levels of RF, bone erosion, in use of any biologic DMARD or targeted-synthetic DMARD, abatacept, rituximab or tocilizumab had worse functional capacity indexes. When compared to non-use, the use of any conventional DMARD was associated with better rates of HAQ-DI.

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AB0201

ASSOCIATION BETWEEN BASELINE CALPROTECTIN SERUM LEVELS AND RESPONSE TO BIOLOGICAL THERAPY IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Calprotectin (CLP) is an important proinflammatory factor of innate immunity released from activated granulocytes and macrophages during inflammation, which has also been identified in synovial fluid. It is a potential pro-inflammatory biomarker reflecting joint damage. CLP levels in synovial fluid are well correlated with levels in plasma, which allows measuring it easily in patients with rheumatoid arthritis (RA). However, the additional value of CLP over other biomarkers is unclear

Objectives: To study the association of baseline CLP serum levels with the clinical response and serum drug levels in patients under biological therapy at 6 and 12 months after starting treatment.

Methods: Prospective observational study including 109 patients with RA who started biological treatment (Infliximab(Ix); Adalimumab(Ada); Etanercept(Etn); Certolizumab(Ctz); Golimumab(Glm); Tocilizumab(Tcz) and Rituximab(Rtx) in a tertiary hospital since 1999 to 2016. Serum CLP levels were measured by ELISA

with the commercial kit CALPROLAB (Lysaker, Norway). Levels of biological drug and ADA were measured by capture and bridging ELISA respectively, except Certolizumab which was measured by Sanquin Diagnostic Service. Clinical response was assessed by DAS28. Serological and clinical parameters were evaluated at baseline, 6 and 12 months. For analysis, linear regression models adjusted for confounders (age, sex, BMI, DMARDs, smoking status) and with standardised beta as estimate were employed.

Results: Out of 109 studied patients, 22% patients received Ix, 6% Ada, 16% Etn, 18% Ctz, 5% Glm, 25% Tcz and 9% Rtx. Most patients were woman (84%), with a median (IQR) age of 62 (52.3–73.1) and disease duration of 9.5 (4.8–15.3). At baseline, CLP levels directly correlated well with CRP and DAS28 ($r=0.411$; $p<0.001$ and $r=0.308$; $p<0.001$). On the other hand, an inverse correlation between baseline CLP levels and the presence of drug at 6 ($\beta=-0.325$; $p=0.01$) and 12 months ($\beta=-0.397$; $p<0.001$) was also found. Moreover, in the adjusted analyses a trend for this correlation at 6 months ($\beta=-0.199$; $p=0.07$) and a significant correlation at 12 months ($\beta=-0.381$; $p<0.001$) was observed. Additionally, in the non-adjusted models, baseline CLP levels significantly directly correlated with the clinical response at 6 months ($\beta=0.194$; $p=0.04$) while the correlation at 12 months was not significant ($\beta=0.186$; $p=0.06$). However, after adjusting for confounders, no significant correlation was found at any study point ($\beta=0.213$; $p=0.06$ at 6 months and $\beta=0.206$; $p=0.07$ at 12 months).

Conclusions: In patients with RA initiating biological therapy, baseline CLP concentrations are inversely correlated with the circulating drug levels along the treatment. However, the additional value of CLP as a predictor of clinical response remains unclear.

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AB0202

GLUCOCORTICOIDS IN THE INITIAL TREAT-TO-TARGET STRATEGY OF EARLY RHEUMATOID ARTHRITIS

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Background: As stated in the 2013 update of RA recommendations, glucocorticoids (GCs) should be used as bridging therapy for up to 6 months, ideally tapering them at earlier time points.¹

Objectives: To evaluate whether initial combination therapy with GCs and disease modifying anti-rheumatic drugs (DMARDs) influences clinical and radiological outcome in the real-life practice of a cohort of early rheumatoid arthritis (ERA) patients.

Methods: A total of 367 ERA patients with less than 12 months of disease duration were enrolled in the study. ERA patients fulfilled the 2010 ACR criteria for RA and were followed according to the treat-to-target strategy. The mean follow-up (FU) was 38.2±32.8 months. At baseline, and every three months, the ACR/EULAR core data set variables were recorded. At baseline and every year, hand and foot radiographs were examined according to modified Total Sharp score (mTSS). At each visit, clinical improvement and remission were evaluated according to EULAR criteria. The achievement of CDC (28-joint Disease Activity Score using C reactive protein <2.6, Health Assessment Questionnaire <0.5 and change from baseline in mTSS ≤0.5) was assessed every year of follow-up.

Results: At baseline 291 (71.9%) ERA patients started GCs at a dosage of 0.2 mg/Kg, gradually tapered and withdrawn as rapidly as clinically feasible. As expected, these patients presented higher values of acute phase reactants ($p=0.001$), and higher levels of disease activity scores ($p<0.001$) and disability index ($p=0.001$), compared to the 76 subjects (18.8%) who had not been prescribed GCs. Patients not treated with GCs were in higher percentages anti-citrullinated peptide antibody (ACPA) (75.0%) and IgM-rheumatoid factor (RF) (64.5%) positive, compared to subjects taking GCs (62.9%, $p=0.05$; 49.8%, $p=0.02$, respectively). There were no differences regarding age, disease duration, BMI, smoking habit and presence of erosions at onset. The mean duration of GC treatment was 7.5±7.9 months and the mean dosage during FU was <5 mg prednisone per day. During the FU, in the two groups a similar percentage of patients started a combination therapy with biological (b)-DMARDs and no differences were observed regarding radiographic progression.

A higher rate of remission defined according to DAS values, 2011 ACR/EULAR criteria, and CDC criteria, was registered in patients not treated with GCs compared to subjects who required corticosteroid therapy. ERA patients that didn't manage to stop GCs at the sixth month (38.8%) had a higher BMI ($p=0.04$) and a lesser chance of achieving remission defined according to DAS values, 2011 ACR/EULAR criteria, and CDC criteria during follow-up. Moreover, a higher percentage of them required a combination therapy with bDMARDs during FU ($p<0.0001$).

Conclusions: In our cohort ERA patients initially treated with GCs had higher disease activity scores at onset compared to subjects without GCs. The lesser chance of achieving remission and the higher rate of bDMARD therapy in ERA

patients not able to stop GCs, reflect a more aggressive disease, refractory to conventional drugs.

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AB0203

CLINICAL PHENOTYPE AND ULTRASOUND CHARACTERISTICS OF RHEUMATOID ARTHRITIS FLARE AFTER DISCONTINUATION OF CONVENTIONAL SYNTHETIC DMARDS

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Background: Current protocols based on early and intensive treatment with csDMARDs in rheumatoid arthritis (RA) have allowed the achievement of remission in a considerable proportion of the patients and opened the perspective, in selected cases, of a drug-free monitoring scheme. Treatment discontinuation can lead, however, to possible recurrence of joint inflammation and clinical flare. Understanding the dynamics acting upstream these events remains a fundamental research task with direct clinical and patho-biologic implications.

Objectives: To delineate the clinical, serological and ultrasonographic changes associated to a drug-free flare in patients discontinuing csDMARD after achievement of stable remission. Co-primary objective was to compare, through a retrospective analysis in the same patients, these changes with early features of the pathology at onset, before treatment introduction.

Methods: 92 RA patients in stable DAS28 remission following a DAS-steered treatment strategy with MTX were recruited in our Centre and introduced to a drug-free monitoring scheme according to the following inclusion criteria: a) treatment introduced within 12 months from symptoms' onset, b) at least 24 months of continuative treatment, c) DAS28 <2.6 for at least 6 months in the absence of glucocorticoids. After discontinuation, all patients were follow-up at three months intervals across 24 months through complete clinical, ultrasonographic (power Doppler ultrasound –PDUS- in hands-feet and tendons) and serological analyses. Treatment was re-introduced upon occurrence of moderate disease activity (DAS28 ≥3.2) in a single occasion.

Results: A total drug-free follow-up of 1398 person-months was analysed with a median (IQR) of 15^{6–24} months. Thirty-eight patients (27/38 in ACR/EULAR Boolean remission, 16/38 with PD score=0 at withdrawal visit) required treatment re-introduction after a median (IQR) time from discontinuation of 6^{3–9} months (range 3–18). DAS28 variations at re-treatment showed a mean (SD) increase of 2.26 (1.03), reflecting significant differences in all DAS components (p<0.001 for ESR, tender joint count, swollen joint count and GH). Clinical activity in flaring subjects was paralleled by average changes in synovial US, with increased PD scores in hands joints (median [IQR]: 3.5 [0.5–7] vs 1 [0–2], p<0.001), feet (1 [0–4] vs 0 [0–0.5], p=0.002) and tendons (0 [0–2] vs 0 [0–0], p=0.002), determining *ex-novo* PD positivity in 85.7% of PD negative patients (p<0.001). Despite stringent remission achieved at the time of discontinuation, no significant differences were observed between disease onset and drug-free flare in DAS28 (p=0.26), patient global assessment of disease activity (p=0.53) and synovial US scores (p=0.61 for grey scale, p=0.31 for PD) with recurrence of similar patterns of clinical joint involvement.

Conclusions: Drug-free clinical flare can occur over a wide temporal window, in the absence of detectable signs of inflammation at the time of treatment discontinuation. It can associate with *ex-novo* recurrence of US pathologic changes at joint and tendon level, reproducing some of the quantitative/qualitative features of disease onset.

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AB0204

RADIOGRAPHIC PROGRESSION OF LARGE JOINT DAMAGE IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH BIOLOGICAL DISEASE-MODIFYING ANTIRHEUMATIC DRUGS (bDMARDs) AND ITS PREDICTIVE FACTORS: RESULTS OF 3 TO 4 YEARS FOLLOW-UP

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Background: Although damage to large joints such as the shoulder, elbow, hip, knee, and ankle has a substantially larger impact on functional ability than

damage to the small joints of the hand and foot,^{1 2} large joints are not routinely monitored for progressive damage in patients with rheumatoid arthritis (RA). Furthermore, a little information is available regarding long-term follow-up results of radiographic progression of damage (RPD) to the large joints during treatment with biological disease-modifying antirheumatic drugs (bDMARDs).

Objectives: We investigated ratios of RPD to the large joints in RA patients treated with bDMARDs for 3 to 4 years and analysed association between RPD and patient backgrounds or Larsen grades of individual large joint.

Methods: Sixty-eight patients (naïve: 42, switch: 26) receiving bDMARDs (IFX: 5, ETN: 9, ADA: 7, GLM: 3, CZP: 5, TCZ: 28, ABT: 11) for 3 to 4 years or achieving bDMARDs-free status were included in this study. The mean age and disease duration at the start of bDMARDs was 62.7 year-old and 10.7 years, respectively, and baseline DAS28-ESR and HAQ was 4.61 and 1.014, respectively. A total of 311 joints including shoulder, elbow, hip, knee, and ankle were evaluated whether there was RPD during the observation period by comparing radiographs before and after the treatment.

Results: RPD was found in 22 patients (31.4%) and 32 joints (10.0%), and it occurred during the former half period in 17 joints and during the latter half period in 15 joints. Joints with Larsen grade (LG) III or more had significantly higher ratios of RPD than those with LG II or less (p<0.01). An ROC analysis was performed to calculate the cut-off value for the progressive damage, which was 2.5 (sensitivity: 50.0%, specificity: 79.6%), suggesting that exceeding LG III would be a risk factor for the RPD to the large joints. A multivariate logistic regression analysis revealed that stage and baseline HAQ were independent risk factors for RPD (cut-off value: 2.5, odds ratio: 8.864 for stage; cut-off value: 1.4375, odds ratio: 6.316 for baseline HAQ).

Conclusions: The stage and baseline HAQ were associated with RPD to the large joints, and progressive damage is expected to increase when the stage exceeds III and/or functional disability exceeds an HAQ score of 1.5. Progressive damage also increases when LG exceeds III. Treatment with bDMARDs should be started before stage, HAQ, and LG exceed III, 1.5, and III, respectively.

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AB0205

COMPARISON OF THORACIC HRCT AND SELF-REPORTED QUESTIONNAIRES IN THE ASSESSMENT OF PULMONARY INVOLVEMENT IN RHEUMATOID ARTHRITIS PATIENTS: PRELIMINARY RESULTS

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Background: Pulmonary involvement in rheumatoid arthritis (RA) is one of the extra-articular manifestations affecting morbidity and mortality during the course of the disease. Pulmonary function tests (PFTs) and thoracic high-resolution computerised tomography (HRCT) are the standard of care in the assessment of pulmonary involvement in RA. In this study, we aimed to compare the findings between self-reported questionnaires and HRCT to detect pulmonary abnormalities in RA patients.

Methods: Forty-two RA patients fulfilling ACR/EULAR classification criteria (2010) who had thoracic HRCT within 6 months of any symptom and/or any pathology on radiography of chest were included in the study. The patients were also assessed by modified Borg Scale, SF-36 Quality of Life Scale and Leicester Cough Questionnaire for the evaluation of respiratory symptoms.

Results: Demographics and clinical characteristics were summarised in table 1. Warrick score, assessing the severity and extent of alveolitis and fibrosis on thoracic HRCT, was evaluated in 15 patients with ILD (score range:4–28). DLCO values were lower in patients with Warrick score ≥1 (73±22% vs. 88±12%, p=0.019) while FVC were not found to be different. The findings of HRCT and self-reported questionnaires were summarised in table 2. Any relationship between self-reported questionnaires and Warrick scores was not detected. Presence of any parenchymal lesions was found to be associated with SF-36 total score (p=0.048). DLCO levels were found to be negatively correlated with SF-36 total scores (r=-0.470, p=0.006).