NR patients; CCL20 was downregulated similarly in ADA R and NR patients; and IL17C, IL22RA1, TIMP4, and CCL11 were not modulated by ADA. Finally, taken together, among the 184 inflammation-related pBM tested, none were associated with clinical response for both ADA and UPA.

Conclusion: We detected common but also discrete alterations in pBMs upon exposure to ADA and UPA overall and further distinctions when we examined pathway changes associated with achievement of a low disease clinical status achieved by each patient population. Whereas both drugs exhibit inhibition of macrophages and granulocyte associated pathways, ADA appears to preferentially affect M1 macrophages and UPA appears to preferentially affect T cells. This modulatory pattern by UPA is consistent with its broad cytokine receptor inhibition profile compared with highly specific TNF inhibition and could account at least in part, for the greater efficacy of UPA over ADA in the SELECT-COM-PARE study.

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FRI0027

INTENSIVE THERAPY IMPROVES SUBCLINICAL SYNOVITIS AND REDUCE FLARE IN RHEUMATOID ARTHRITIS PATIENTS ACHIEVED TREATMENT TARGET

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Background: Subclinical synovitis has been confirmed to be associated with the subsequent bone erosion and flare in rheumatoid arthritis (RA) patients who have already achieved clinical remission. But whether intensive therapy can improve the subclinical synovitis and reduce flare remains controversial.

Objectives: We designed an 1-year open-labelled, randomized and controlled clinical trial to elucidate if intensive therapy can improve the subclinical synovitis and reduce clinical relapse.

Methods: RA patients in clinical remission/low disease activity (defined by DAS28-CRP<3.2), however with subclinical synovitis [power Doppler (PD)≥1 and/or gray scale (GS)≥2 on ultrasound] were randomized into either maintenance or intensive treatment group. The rate of relapse [defined by DAS28(CRP) increased by at least 0.6 and >3.2] in 12 months was taken as the primary outcome. The improvement of PD and GS synovitis at 3, 6, 9 and 12 months were taken as secondary outcomes.

Results: 108 patients with 54 in each group were enrolled. Patients in maintenance group were older [59.5(19.4-77.5) vs. 51.8 (22.1-73.0), p=0.009] with lower baseline PD score [0(0-4) vs. 1(0-11), p=0.021] than intensive treatment group. The relapse rate was significantly higher in maintenance group than in intensive group [24.1% (13/54) vs. 9.1% (5/54), p=0.039] in one year. The intensive therapy is the single independent protective factor for relapse [OR=0.284 (0.081-0.998), p=0.049]. For 26 patients in maintenance group and 38 in intensive group with PD score≥1, their age and baseline PD were comparable. The relapse rate was also higher in maintenance group than in intensive group, although the difference was statistically insignificant [23.1% (6/26) vs. 7.9% (3/38), p=0.164].

Both maintain and intensive therapy could improve GS and PD scores (Table 1). However, at 12 month, both GS and PD score improvement were significantly better in intensive group than in maintenance group (Table 2).

Table 1. The improvement of GS and PD scores at each follow up time point in the maintenance and intensive therapy group.

Patients with GS≥2					Patients with PD≥1				
GS	maintenance group(n=54)	P (against baseline)	Intensive group(n=54)	P (against baseline)	PD	maintenance group (n=26)	P (against baseline)	Intensive group(n=38)	P (against baseline)
Baseline	5(1-24)	—	5.5(0-28)	—	Baseline	2(1-4)	—	1.5(1-11)	—
3m	4(0-16)	0.031	5(0-28)	0.233	3m	0(0-6)	0.006	1(0-10)	0.001
6m	4(0-17)	0.025	4(0-17)	0.014	6m	1(0-9)	0.006	0(0-4)	<0.001
9m	2(0-21)	<0.001	2(0-12)	<0.001	9m	0(0-2)	0.001	0(0-3)	<0.001
12m	3(0-23)	0.002	2(0-11)	<0.001	12m	0(0-6)	0.001	0(0-4)	<0.001

Table 2. The improvement of GS and PD scores at each follow up time point between the maintenance and intensive therapy group.

Patients with GS≥2				Patients with PD≥1			
Change of GS against baseline	maintenance group(n=54)	Intensive group(n=54)	P	Change of PD against baseline	maintenance group (n=26)	Intensive group(n=38)	P
3m	1(-10-12)	1(-11-13)	0.815	3m	1(-4-3)	1(-3-7)	0.499
6m	1(-14-21)	2(-14-19)	0.330	6m	1(-7-4)	1(-1-8)	0.099
9m	3(-3-16)	4(-2-26)	0.266	9m	1(0-3)	1.5(1-11)	0.256
12m	2(-7-19)	3.5(-4-26)	0.048	12m	1(-2-2)	1(-1-11)	0.020

Conclusion: For RA patients who have already achieved treatment target, intensive therapy can alleviate the subclinical synovitis better than maintenance therapy and reduce the relapse.

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FRI0028

JAK2 MUTATION MAY PREDICT RESPONSE AND GUIDE FIRST LINE TREATMENT IN RHEUMATOID ARTHRITIS

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Background: JAK2 mutation found to be associated with the myeloproliferative disorders [1] with possibility of measurement in blood by PCR [2]. There are many interesting parallels between the CTDs and MPDs [3]. STAT4 is activated by the JAK members as JAK2 in response to IL and TNF, with subsequent down-stream signaling for autoimmunity [4]. JAK inhibitors are medications function by inhibiting activity of Janus kinase enzymes and have therapeutic application in treatment of cancer and rheumatoid arthritis [5].

Objectives: Impact of pretreatment JAK2 mutation on response to first line csDMARDs in RA.

Methods: 76 newly diagnosed RA patients and 50 matched controls were included. Pretreatment JAK2 mutation was measured in serum by PCR with TNF-α and IL 6 were assessed by ELISA in patients and controls. All patients started treatment by conventional synthetic DMARDs (including methotrexate). Response assessment at 3rd month was evaluated by DAS28 and ACR response criteria. JAK2 mutation was correlated with different clinical and laboratory parameters of patients.

Results: 62 females (81.6%) and 14 males (18.4%) with age mean ± SD; (48.8 ± 7.3). Pretreatment JAK2 mutation, TNF-α and IL 6 were significantly high in patients. JAK2 mutation was detected in 39 (51.3%) patients while 37 (48.7%) patients were JAK2 non mutant. Mutant JAK2 was significantly associated with severity of disease assessed by DAS28; 14 (70%) of patients with DAS28 (≤2.6) were non mutant JAK2 versus sex (30%)

patients mutant JAK2 while 13 (76.5%) of patients with DAS28 (>5.1) were mutant JAK2 versus four (23.5%) patients non mutant JAK2 (P 0.03), table1. JAK2 mutation found to be significantly correlated with ACR 20, 50 and 70 response criteria; 40% of patients with non mutant JAK2 showed ACR 70 versus 17.9% in mutant group, 35.1% of patients with non mutant JAK2 showed ACR 50 versus 30.8% in mutant group while 24.3% of patients with non mutant JAK2 showed ACR 20 versus 51.3% in mutant group (P 0.02), table1. JAK2 mutation was associated with high pretreatment TNF α (mean $\pm$ SD; 41.7 $\pm$ 39.5 in mutant versus 24.3 $\pm$ 23.04 pg/ml in non mutant group) with P (0.04), while no significant relation between JAK2 mutation and pretreatment IL6 level.

Conclusion: Adult SLE with pretreatment JAK2 mutation significantly showed high disease activity, high pretreatment TNF α level and poor response to 1st line csDMARDs including MTX so they could get benefit with introduction of JAK inhibitors as first line mono or in combination with csDMARDs especially those with moderate to severe active RA.

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Table 1. JAK2 mutation with disease activity and response criteria.

DAS28	Non Mutant JAK2	Mutant JAK2	X	P
Remission ($\leq$ 2.6)	14 (70%)	6 (30%)	8.6	0.03
Low disease activity ($\leq$ 3.2)	13 (54.2%)	11 (45.8%)		
Moderate disease activity (3.2 - 5.1)	6 (40%)	9 (60%)		
Sever disease activity (>5.1)	4 (23.5%)	13 (76.5%)		
ACR	Non Mutant JAK2	Mutant JAK2		
ACR 20	9 (24.3%)	20 (51.3%)	7.0	0.02
ACR 50	13 (35.1%)	12 (30.8%)		
ACR 70	15 (40.5%)	7 (17.9%)		

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FRI0029

EFFECTIVENESS OF A NURSE LED TREAT TO TARGET (T2T) MODEL IN ACHIEVING REMISSION FOR EARLY RHEUMATOID ARTHRITIS PATIENTS IN A REAL-WORLD SETTING

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Background: Rheumatoid Arthritis (RA) is a chronic, inflammatory and systemic autoimmune disease, affecting multiple joints particularly small joints in hands and feet.¹ For the past two decades, there has been significant change in approach for the diagnosis and treatment of RA. Now early diagnosis and aggressive management of RA is considered key role for preventing permanent damage to joints.² Treat to Target (T2T) strategy has been proven to be very effective in achieving pre-decided outcome either clinical remission (CR) or low disease activity(LDA).³

Objectives: To assess the outcome of nurse led T2T strategy in Early RA patients within first 2 years of treatment.

Methods: This was Observational Prospective Study, based on data collected in our ANP led Early Inflammatory Arthritis clinic. All patients diagnosed with inflammatory arthritis were managed following a T2T protocol. Every patient had predefined target set according to co-morbidities, preexisting osteoarthritis and duration of disease. Patients were followed up at varying intervals depending on clinical need until the steroid free target was achieved or for up to 2 years. Clinical Disease Activity Index (CDAI) tool was used to assess disease activity.

Data collected included age, gender, diagnosis, Rheumatoid factor and/or Anti CCP positivity, erosions on x ray at the time of diagnosis and Health Assessment Questionnaire (HAQ-DI). For this analysis, only patients meeting the 2010 ACR/EULAR criteria⁴ for the diagnosis of RA were included.

The primary analysis in this study was the proportion of patients who achieved CR or LDA using different DMARDs used alone or in combination and the clinical determinants of this outcome.

Results: Out of 417 patients with Early Inflammatory Arthritis, 300 met criteria for RA. 27 were lost to follow up (3 deaths) and 44 remained on the T2T pathway. For the 229 patients completing the pathway, mean age was 43.25 years (SD 9.405), Female gender 146 (63.75%), Anti CCP positive in 160 (69.86%), Rheumatoid Factor (RF) positive in 154 (67.24%), Both RF and Anti CCP positive 141 (61.57%), CDAI at baseline was 22.65 (SD 22.19) and Mean HAQ-DI at presentation 1.19 (SD 0.629). There were 18.34% (42) of patients who had erosive changes on x-ray at presentation. Outcome of T2T strategy showed that 151 (65.93%) achieved clinical remission and 78(34.06%) achieved low disease activity

Conclusion: Remission in patients who were less than 40 years of age was 100% as opposed to 49% above the age of 40 years. This difference was found to be statistically significant with a p value of <0.05 (Table 1).

Table 1. Treat to target outcomes with analysed variables.

Group Characters	Remission n (%)	Low disease activity n (%)	Total	P value
Female	92 (63.01)	54 (36.99)	146	0.215
Male	59 (71.08)	24 (28.92)	83	
RF +	103 (66.88)	51 (33.12)	154	0.666
RF -	48 (64)	27 (36)	75	
CCP +	105 (65.63)	55 (34.37)	160	0.760
CCP -	44 (67.79)	21 (32.31)	65	
< 40 years	76 (100)	0 (0)	76	0.000
>40 years	75 (49.02)	78 (50.98)	153	

Further to this, Methotrexate monotherapy was the dominant DMARD used to achieve remission and/or low disease activity in more than 50 % of the RA patients (Figure 1).

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