Tabla 2. Treatment results during the follow-up period.

	Baseline (n 28)	3 m (n 28)	6 m (n 25)	12 m (n 21)
TJC	10,8±5,4	3,8±3,3	4,23±2,5	1,9±1,5
SJC	7,2±4,6	1,8±1,7	1,7±2	0,7±1
CPR mg/dL	1±0,6	0,54±0,48	0,64±0,9	0,33±0,24
DAS28CPR	5,4±0,91	3,29±0,97	3,15±1,2	2,15±0,6
Prednisone mg	7,2±4,2	6,8±3,5	5,3±2,5	3,1±2,1

Conclusion: Our data show that therapy with a second JAKi is a safe and efficacious option after discontinuation of the first JAKi due to either inefficacy or side effects. The response rate to the second JAKi is similar in patients with inefficacy or side effects which suggests that failure to the first does not reduce the chance of response to the second.

Acknowledgments: M. Retuerto was recipient of a training grant from Sociedad Española de Reumatología (SER).

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.6284

FRI0133 RELATIONSHIP BETWEEN MELATONIN SERUM LEVELS AND THE EFFICACY OF SELECTIVE SEROTONIN REUPTAKE INHIBITOR PAROXETINE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Numerous clinical and epidemiological studies have established that there is a close relationship between inflammation, chronic pain and psycho-emotional disorders in rheumatoid arthritis [1, 2]. The common pathogenetic mechanism is manifested in the defect of melatonin mediation and cytokine stimulation [3]. Therefore, features of the use of selective serotonin reuptake inhibitors is relevant.

Objectives: To study the relationship between serum melatonin level and the efficacy of selective serotonin reuptake inhibitor paroxetine in patients with RA.

Methods: A total of 127 RA patients and 71 healthy volunteers were examined. The following information was collected for each patient: medical history data, physical examination results, serum melatonin levels. RA patients were randomly categorized into two treatment groups – 63 and 64 patients. The basic treatment for patients of both groups included nonsteroidal anti-inflammatory drugs, glucocorticoids (equivalent to 10 mg of prednisolone), and disease-modifying antirheumatic drugs (methotrexate, leflunomide or sulfasalazine). To evaluate the effectiveness of treatment, patients of both groups were further divided into three subgroups depending on the serum melatonin level (low level corresponds to 25 percentile, medium - 25-75, high - 75 percentile). First group received paroxetine 20 mg once a day for 12 weeks in addition to the basic treatment. Effectiveness of the treatment was evaluated according to the ACR/EULAR criteria.

Results: The mean baseline plasma melatonin levels in RA patients were significantly higher than in the healthy volunteers (26.1±32.7 vs 13.6±4.6 pg/mL at 8 am and 11.5±15.5 vs 3.6±4.6 pg/mL at 20 pm (p<0,001), respectively). A good response to basic treatment was observed in groups with medium and high serum melatonin levels, who received paroxetine (p<0.05). However, patients who did not receive paroxetine gave best response to treatment in group with low serum melatonin levels (p<0.05).

Conclusion: Obtained data suggest that the high level of serum melatonin is one of the predictors of resistance for basic RA treatment. The proposed scheme of treatment with addition of paroxetine demonstrated high efficacy concerning the main manifestations of the disease in RA patients with high melatonin serum level. This study demonstrates the possible influence of serotoninergic interactions on the melatoninergic system and their contribution to the pathogenesis of RA.

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Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.5483

FRI0134 EFFECT OF JAK INHIBITORS ON PAIN AND QUALITY OF LIFE IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Pain control is considered a treatment priority from most patients with Rheumatoid Arthritis (RA). Despite the treat to target approach, residual pain is commonly reported by patients with RA. Treatment with JAK inhibitors (JAKi) has been associated to a rapid control of pain.

Objectives: To investigate the effect of JAKi on pain and quality of life in a mono-centric real-life clinical setting.

Methods: Patients candidate to baricitinib or tofacitinib were evaluated at baseline and after 12 and 24 weeks of treatment. Disease activity was assessed by Disease Activity Score (DAS)28 with C reactive protein (CRP). A reduction of ≥ 50% of pain visual-analogue scale (VAS) 0-100 mm was recorded as "very much improved, substantially improved" (1). Pain VAS score ≤ 10 mm was considered "no/limited pain" (2). Patients' satisfaction was assessed by the Patient Acceptable Symptom State question (3). Data were expressed as mean (SD) or median (interquartile range) according to the variables' distribution. Mann Witney test was use and p values <0.05 were considered statistically significant.

Results: Overall 108 patients started a JAK inhibitor (baricitinib n=67, tofacitinib n=41). Eighty-four patients (baricitinib n=51; tofacitinib n=33) were followed-up for at least 3 months and were included in the analysis. Table 1 summarizes demographic and clinical characteristic of the cohort. After 12 and 24 weeks of treatment we detected a significant reduction of DAS28 compared with baseline [from 4.7 (1.5) to 3.2 (1.7) 2.9 (1.5) and 2.7 (1.1), respectively; p<0.001; p<0.00001 and p<0.00001). At week 4, 27% and 51.8% of patients achieved remission and low disease activity, respectively; the percentages increase to 32.1% and 60.7% at week 12 and 42.2% and 70.3% at week 24. When evaluating the extent of reduction of the single items included in the DAS28 composite index we found that number of tender (TJ) and swollen joints (SJ) decreased from 9 (7.8) to 5 (3.5) to 4 (5) and 1 (3) at week 4, 2 (4) and 1 (3) at week 12, and 2 (4) and 1 (3) at week 24, respectively (p<0.00001 for all); the median reduction of TJC and SJC at week 4, 12 and 24 was 60%, 77% and 88%, and 81%, 86% and 100%, respectively. GH decreased from 70(30) to 40(40) at week 4, 40(30) at week 12 and 37(40) at week 24 (p<0.00001) with a median reduction of 37.5%, 44% and 46%. C reactive protein decreased by 54.5% at week 4, 47% at week 12 and 55% at week 24. VAS pain was significantly reduced at week 4, 12 and 24 [from 70(25) to 40(40,)30(40) at the three timepoints, p<0.00001] decreasing by 37.5%, 50% and 54%, respectively. A substantial reduction (≥50%) in VAS pain was reported by 41.3%, 54.4% and 53.9% of patients after 4, 12 and 24 weeks, respectively. Limited/no pain was reported by 21.3%, 24.7% and 36.5% at weeks 4, 12 and 24, respectively. Overall, 81.8% of patients achieved the PASS after a median time of 10 (7-15) days.

Conclusion: JAK inhibitors baricitinib and tofacitinib induce a rapid improvement of disease activity driven both by pain and inflammation control. Even if no/limited pain was described only by one third of the patients, most of them reported a rapid and sustained reduction of pain accounting for the achievement of a satisfactory health condition.

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	Baricitinib (n=51)	Tofacitinib (n=33)	P
F:M	43: 8	26:7	ns
Age, mean (SD)	59±12	60±12	ns
Disease duration, mean (SD)	163±101	170±112	ns
Baseline DAS28(PCR), median (IQR)	4.7 (4-5.6)	4.7 (4.3-5.4)	ns
Concomitant methotrexate, n (%)	27 (52.9)	8 (24.2)	<0.001
Daily prednisone dose, median (IQR)	5 (2.5-9.5)	5 (1.88-9.9)	ns
N° of previous csDMRADs, median (IQR)	3 (1-4)	2.5 (2-3)	ns
N° of previous bDMRADs, median (IQR)	2 (1-4)	1 (0-2.5)	ns

Disclosure of Interests: Francesca Romana Spinelli Grant/research support from: Pfizer, Speakers bureau: Lilly, BMS, Celgene, Cristina Garufi: None declared, Fulvia Ceccarelli: None declared, Silvia Mancuso: None declared, Ilaria Duca: None declared, cristiano alessandri Grant/research support from: Pfizer, Manuela Di Franco: None declared, Roberta Priori: None declared, Valeria Riccieri: None declared, Rossana Scrivo: None declared, Guido Valesini: None declared, fabrizio conti Speakers bureau: BMS, Lilly, Abbvie, Pfizer, Sanofi

DOI: 10.1136/annrheumdis-2020-eular.4903

Ann Rheum Dis: first published as 10.1136/annrheumdis-2020-eular.5483 on 13 June 2020. Downloaded from http://ard.bmj.com/ on October 12, 2020 by guest. Protected by copyright.