

**Background:** Axial involvement in psoriatic arthritis (PsA) is quite common. Tofacitinib (TOFA) is an oral Janus kinase inhibitor. There is no data on the use of TOFA in PsA patients (pts) with axial involvement, nor is there any data on its effect on active MRI sacroiliitis (MRI-SI). The decrease in number of active MRI-SI pts is statistically significant (p = 0.017; Pearson-$\chi^2$). At baseline, inflammatory changes were detected in 23 of 80 (28.8%) SIJs, after 6 months of therapy they were found in 5 of 70 (7.1%) SIJs observed. Decrease in number of SIJs with active inflammation is statistically significant (p = 0.001; Pearson-$\chi^2$). At baseline, Me BASDAI 6.0 [4.2; 7.0], Me ASDAS 3.8 [2.8; 4.4]. After 6 months of treatment, Me BASDAI 1.4 [0.6; 3.2], Me ASDAS1.5 [1.0; 2.1] (p = 0.001 for both comparisons).

**Conclusion:** JAK inhibition using TOFA therapy shows high efficacy in reducing active MRI-SI and decreasing activity of axial involvement in PsA. More extensive studies are needed.

**References:**


**Disclosure of Interests:** ELENA GUBAR: None declared, Tatiana Korotaeva Grant/research support from: Pfizer, Consultant of: Abbvie, BIOCAD, Bristol-Myers Squibb, Celgene, Eli Lilly, Janssen, Merck Sharp & Dohme, Novartis, Novartis-Sandoz, Pfizer, UCB, Speakers bureau: Abbvie, BIOCAD, Bristol-Myers Squibb, Celgene, Eli Lilly, Janssen, Merck Sharp & Dohme, Novartis, Novartis-Sandoz, Pfizer, UCB, Yulia Korosakova: None declared, Elena Loginova

**Objectives:** To study the effect of TOFA therapy in PsA pts having active SI on MRI (MRI-SI) and dactylitis. Methods: 40 pts (M/F – 23/17) with active PsA fulfilling the CASPAR criteria were examined. Median (Me) age 41.0 [35.0; 50.0] yrs, Me PsA duration 6.0 [3.0; 10.0] yrs. Pts underwent a standard clinical examination of PsA activity: Me tender joint count 19 [12; 24], swollen joint count 11 [8; 16], patient’s global disease activity measured by Visual Analogue Scale (VAS) 70 [50; 80], patient’s pain VAS 65 [50; 75], Me activity indexes: DAPSA 44.2 [37.8; 55.3], BASDAI 6.0 [4.2; 7.0], ASDAS 3.8 [2.8; 4.4], CRP 21.3 [3.2; 72.3] mg/L, ESR 28 [12; 52] mm/h. Enthesitis was observed in 65.9% of pts with Me LEI index 1 [0; 1], dactylitis in 53.7% of pts, Me digits with dactylitis 1 [0; 2]. Apart from a standard clinical examination, MRI of sacroiliac joints (SIJs) was performed in all 40 pts using MRI scanner Siemens General Electric 1.5 TESLA. Bone marrow edema/ostitis on MRI (STIR) considered active MRI sacroiliitis (MRI-SI), was evaluated by 2 independent readers (radiologist and rheumatologist). TOFA was given in 5mg tablets bds over a period of 6 months, after which 35 patients underwent SI MRI. Me [2Q5; 2Q7], Pierson-$\chi^2$ tests were performed. All p < 0.05 were considered to indicate statistical significance.

**Results:** Prior to TOFA therapy, active MRI-SI was detected in 14 of 40 (35.0%) pts. At the end of 6 months therapy, MRI-SI was detected in 4 of 35 (11.4%) pts: in 1 pt with baseline bilateral MRI-SI and in 2 pts with unilateral MRI-SI. 1 pt showed negative dynamics, that is, development of active MRI-SI (absent at baseline). The decrease in number of active MRI-SI patients is statistically significant (p = 0.017; Pearson-$\chi^2$).
Dynamics of symptoms in TOFA treated pts with or without MRI-SI

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>MRT-SI (+) (n=14)</th>
<th>MRT-SI (-) (n=26)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>After 6 months</td>
</tr>
<tr>
<td>Number of digits with dactylitis</td>
<td>2 (0, 4)</td>
<td>0</td>
</tr>
<tr>
<td>ESR (mm/h)</td>
<td>47 (26, 76)</td>
<td>12 (6, 16)</td>
</tr>
<tr>
<td>WPAI [%]</td>
<td>80 (56; 84)</td>
<td>0</td>
</tr>
</tbody>
</table>

After 6 months of TOFA therapy, no differences were found between groups of pts with and without MRI-SI in the number of digits with dactylitis (p=0.47), in ESR (p=0.78) and in WPAI (p=0.93).

Conclusion: In PsA pts significant association of active MRI-SI was found with dactylitis, high ESR level and WPAI. Use of TOFA in pts with both active MRI-SI and dactylitis demonstrated its high efficacy in reduction of SI inflammation and dactylitis; it also significantly improved pts' work productivity. These findings are important for personalized approach to treatment of PsA.

References:

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Efficacy of Non-Tumour Necrosis Factor Biologics and Targeted Systemic Disease Modifying Anti-Rheumatic Drugs in the Treatment of Psoriatic Arthritis: A Systematic Review and Meta-Analysis

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Background: Psoriatic arthritis (PsA) is a systemic, inflammatory condition presenting in approximately 30% of patients with psoriasis and associated with functional impairment and a reduced health-related quality of life. Current treatment guidelines recommend non-steroidal anti-inflammatory drugs, conventional Disease Modifying Anti-Rheumatic Drugs (cDMARDs) and Tumour Necrosis Factor α inhibitors (TNFi). Recent research has focused on alternative biologic medications which target interleukin (IL) 6, 12/23, 17A, 23 and T Cell co-stimulation, as well as targeted synthetic DMARDs (tsDMARDs) including Janus Kinase inhibitors (JAKi) and Phosphodiesterase 4 inhibitors (PDE4i). Evidence of the safety and efficacy, measured using the American College of Rheumatology-20 (ACR20), has been demonstrated leading to the inclusion of several biologics and tsDMARDs in guidelines. However, it can be argued that ACR50, indicating a 50% improvement in disease, is a more clinically relevant outcome measure.

Objectives: To conduct a systematic review and meta-analysis of the efficacy of non-TNFi biologics and tsDMARDs in the treatment of PsA.

Methods: A systematic literature search of Embase, Medline and Web of Science was undertaken to identify randomised controlled trials (RCTs) investigating efficacy and safety of non-TNFi biologics and tsDMARDs published in English from the inception of the databases to September 2019. The Cochrane Risk of Bias tool was used to assess methodological rigour of included trials. A meta-analysis was performed using a random effects model to estimate odds ratios of ACR 50 response vs placebo. A subgroup analysis was performed using patients with previous TNFi exposure.

Results: 21 RCTs were eligible with 6389 participants. Evaluation periods ranged from 12 to 24 weeks. JAKi, PDE4i, IL6i, IL12/23i, IL17Ai and IL23i treatments were more efficacious than placebo for ACR50 response (p<0.0001) (Figure 1). Only tofacitinib (JAKi), secukinumab (IL17Ai) and ixekizumab (IL17Ai) were able to demonstrate efficacy at the ACR50 level in participants with prior TNFi exposure (p<0.0001) (Figure 2). All treatments demonstrated an adequate safety profile.

Conclusion: Non TNFi biologics and tsDMARDs are able to demonstrate 50% improvement with adequate safety profiles. These therapies are often used in patients who are inadequate responders to TNFi but there is less robust data in this specific patient group. Studies with clinically relevant primary endpoints should be considered in this patient population.