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# CONTENTS OF THE JOURNAL

# HEAD-TO-HEAD COMPARATIVE EFFICACY OF GOLIMUMAB VS. TOFACITINIB IN REACTIVE ARTHRITIS: A 52-WEEK REAL-WORLD COHORT STUDY FROM UZBEKISTAN

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***Abstract.*** *Reactive arthritis (ReA) is an inflammatory joint disease triggered by urogenital or enteric infections. This 52-week prospective cohort from Uzbekistan compared the TNF-α inhibitor golimumab and the JAK inhibitor tofacitinib in 60 ReA patients. Both treatments produced significant reductions in DAREA, BASDAI, and inflammatory markers. Mean DAREA decreased from 4.7 to 1.6 with golimumab and from 4.5 to 1.1 with tofacitinib ($p < 0.001$). Cytokine levels (TNF-α, IL-17A) declined by over 40%, correlating with clinical improvement. Adverse events were mild, and no thromboembolic or fatal outcomes occurred. Tofacitinib achieved slightly faster and deeper responses, supporting its role as an effective oral alternative to biologic therapy in resource-limited settings.*

***Keywords:*** *Reactive arthritis; Golimumab; Tofacitinib; TNF-α inhibitor; JAK inhibitor; Cytokines; Real-world study; Uzbekistan.*

**Introduction:** Reactive arthritis (ReA) is an inflammatory joint disease that develops after certain infections, most often involving the urogenital or gastrointestinal tract [13,14]. It remains one of the most intriguing forms of spondyloarthritis because of its unpredictable course—some patients experience only a short, self-limiting episode, while others develop a chronic, disabling form of arthritis [6,8,14]. Typical triggers include *Chlamydia trachomatis* and enteric pathogens such as *Shigella*, *Salmonella*, *Yersinia*, and *Campylobacter* [13,14]. The disease predominantly affects young adults, presenting with asymmetric arthritis of the lower limbs, enthesitis, and occasionally axial involvement, along with extra-articular manifestations such as conjunctivitis, urethritis, or mucocutaneous lesions [6,7,14]. The persistence and recurrence of inflammation in ReA are strongly influenced by genetic and immunological factors. The human leukocyte antigen B27 (HLA-B27) allele is particularly important, detected in nearly 80% of patients [6,8]. HLA-B27 positivity is linked not only with susceptibility but also with a more severe disease course and higher risk of progression to axial spondyloarthritis. Despite decades of research, the precise mechanisms driving chronic inflammation in ReA are not completely understood, and management continues to rely heavily on clinical experience rather than robust clinical trial evidence. Epidemiologically, the incidence of ReA varies widely—from less than 1 to over 25 cases per 100,000 people per year—depending on regional infection patterns and diagnostic practices [13,19]. In Central Asia, including Uzbekistan, reactive arthritis remains underreported and often underdiagnosed, yet clinical observations suggest it is not uncommon, especially following urogenital infections. In many patients, delayed diagnosis, limited access to advanced therapies, and the high cost of biologic drugs contribute to persistent disease activity and reduced quality of life [19,20,21]. These realities make real-world studies from the region especially important to help guide evidence-based treatment approaches. Traditional management of ReA begins with nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and, where relevant, antibiotics targeting persistent infections. When the disease becomes chronic, conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) such as methotrexate or sulfasalazine are commonly added. Unfortunately, many patients do not respond adequately to these medications, leaving physicians to explore biologic options such as tumor necrosis factor-alpha (TNF-α) inhibitors [7,14] TNF blockers have revolutionized the treatment of inflammatory arthritis by targeting one of the central cytokines driving inflammation [1,7,8,11]. Their effectiveness in reactive arthritis has been supported by case series and small cohort studies, showing improvements in joint pain, swelling, and function. However, challenges remain—cost, injection-related issues, and safety concerns, particularly the risk of infections, make biologics less accessible in resource-limited countries. The advent of Janus kinase (JAK) inhibitors such as tofacitinib has opened new therapeutic possibilities. These oral agents target intracellular signaling pathways responsible for the production of multiple pro-inflammatory cytokines. Tofacitinib has already proven effective in rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis, leading to significant improvements in disease activity and patient function. In recent years, case reports and small observational studies have suggested that JAK inhibition might also help patients with reactive arthritis, including those who have failed biologic therapy. For example, a recent case published in *Frontiers in Immunology* described complete remission of skin and joint symptoms within 20 days after starting tofacitinib in a patient unresponsive to TNF and IL-17 inhibitors. Such observations hint that JAK inhibition may act on inflammatory pathways beyond those targeted by biologics, offering hope for patients with refractory disease. However, despite these promising findings, no prospective, head-to-head comparisons have yet evaluated TNF-α inhibitors versus JAK inhibitors in reactive

arthritis [1,3,5,10,12,15] Evidence from real-world settings—especially from low- and middle-income countries—is virtually absent. The differences in infection patterns, genetics, and healthcare infrastructure across regions make it essential to study treatment outcomes in diverse populations. To address this knowledge gap, the present 52-week prospective cohort study was conducted in Uzbekistan to directly compare the clinical efficacy, safety, and functional outcomes of TNF-α inhibitors and tofacitinib (with or without csDMARDs) in patients with reactive arthritis. This study aims to provide the first real-world data from Central Asia, offering insight into how these two therapeutic strategies perform outside the controlled environment of clinical trials. We hypothesize that tofacitinib may provide comparable or even superior disease control with favorable safety and convenience, making it an attractive option for patients in regions with limited biologic access [20,21,22]

**Materials and Methods:** This prospective, observational, real-world cohort study was conducted at the Department of Internal Diseases and Rheumatology of Tashkent State Medical University, in collaboration with regional rheumatology centers and affiliated hospitals across Uzbekistan. The study was performed over a two-year period, from January 2023 to January 2025, and aimed to evaluate and compare the clinical efficacy and safety of TNF-α inhibitors and tofacitinib in patients with reactive arthritis (ReA). All study procedures were conducted in accordance with the principles of the Declaration of Helsinki (2013), and the protocol was approved by the Institutional Ethics Committee of Tashkent State Medical University (Approval No. RHEA-2023/02). Written informed consent was obtained from all participants prior to enrollment. Eligible participants were adults aged 18–60 years who met the European Spondyloarthropathy Study Group (ESSG) and 2009 American College of Rheumatology (ACR) classification criteria for reactive arthritis. All patients had active arthritis, enthesitis, or axial symptoms persisting for at least six weeks despite prior use of nonsteroidal anti-inflammatory drugs (NSAIDs). Exclusion criteria included active infection (including tuberculosis, hepatitis B or C, or HIV), uncontrolled systemic disease, pregnancy or lactation, previous exposure to biologic or JAK inhibitors, concomitant autoimmune rheumatic diseases, malignancy, or a history of thromboembolic or cardiovascular events. A total of 60 patients meeting the inclusion criteria were consecutively enrolled from the outpatient and inpatient departments of Tashkent and regional hospitals. Participants were divided equally into two treatment groups. The TNF-α inhibitor group (n = 30) received adalimumab 40 mg subcutaneously every other week or etanercept 50 mg weekly, depending on clinical indication and drug availability. The Tofacitinib group (n = 30) received oral tofacitinib 5 mg twice daily. All antibiotic therapy targeting *Chlamydia trachomatis* or enteric infections (*Shigella*, *Yersinia*, *Salmonella*, *Campylobacter*) was completed before enrollment. No corticosteroids or conventional synthetic DMARDs were used during the study period. Treatment decisions were based on clinical judgment and patient preference, reflecting real-world practice and the availability of medications in Uzbekistan. Patients were evaluated at baseline and at weeks 4, 13, 26, and 52, with a final extended follow-up at week 104 to assess long-term efficacy, safety, and treatment retention. Disease activity was measured using the Disease Activity for Reactive Arthritis (DAREA) score as the primary index. Additional assessments included the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Functional Index (BASFI), Visual Analogue Scale (VAS) for pain and physician global assessment, and the Health Assessment Questionnaire-Disability Index (HAQ-DI). Laboratory investigations at each visit included erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and serum cytokine levels of TNF-α and IL-17A. Cytokine analysis was performed using standardized enzyme-linked immunosorbent assay (ELISA) kits in the Central Immunology Laboratory of Tashkent State Medical University. Safety monitoring was performed throughout the study period and included physical examination, vital signs, complete blood count, liver and renal function tests, and lipid profile. Adverse events (AEs) were classified according to the Common Terminology Criteria for Adverse Events (CTCAE, version 5.0). Serious adverse events (SAEs) such as severe infections, hospitalization, cardiovascular complications, or venous thromboembolism were reviewed by an independent safety monitoring committee. The primary endpoint was the mean change in DAREA score from baseline to week 52. Secondary endpoints included the proportion of patients achieving clinical remission (DAREA < 1.6) at weeks 52 and 104, mean change in BASDAI and BASFI, normalization of ESR and CRP, time to clinical response, overall safety, and treatment retention rates at 104 weeks. All statistical analyses were conducted using IBM SPSS Statistics version 26.0. Continuous variables were presented as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Between-group comparisons were made using Student's *t*-test or Mann–Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Longitudinal changes over time were analyzed using repeated-measures ANOVA or the Friedman test. Correlations between clinical and laboratory parameters were assessed using Spearman's rank correlation coefficient. A $p$-value < 0.05 was considered statistically significant. Missing data were handled using the last-observation-carried-forward (LOCF) method. A sample size of 60 patients (30 per group) provided 80% power to detect a clinically meaningful between-group difference of at least 1.2 points in DAREA improvement, with an assumed standard deviation of 1.5 and a two-sided alpha of 0.05, accounting for a 10% attrition rate.

**Table 1.**

**Baseline Demographic and Clinical Characteristics of Patients with Reactive Arthritis (n = 60)**

| Parameter | TNF–α inhibitor group (n = 30) | Tofacitinib group (n = 30) | p–value |
|---|---|---|---|
| Age, years (mean ± SD) | 38.1 ± 9.5 | 37.4 ± 8.9 | 0.74 |
| Male sex, n (%) | 22 (73.3) | 21 (70.0) | 0.78 |
| Disease duration, years (mean ± SD) | 1.9 ± 0.7 | 1.8 ± 0.6 | 0.62 |
| HLA–B27 positive, n (%) | 23 (76.7) | 23 (76.7) | 1.00 |
| Infectious trigger | | | |
| *Chlamydia trachomatis* n (%) | 13 (43.3) | 12 (40.0) | 0.79 |
| Post–enterocolitic pathogens n (%) | 17 (56.7) | 18 (60.0) | 0.79 |
| Peripheral arthritis, n (%) | 24 (80.0) | 24 (80.0) | 1.00 |
| Axial involvement, n (%) | 6 (20.0) | 6 (20.0) | 1.00 |
| DAREA score (mean ± SD) | 4.7 ± 0.8 | 4.5 ± 0.9 | 0.41 |
| BASDAI (mean ± SD) | 6.1 ± 1.1 | 6.3 ± 0.8 | 0.46 |
| BASFI (mean ± SD) | 5.8 ± 1.2 | 5.9 ± 1.1 | 0.82 |
| VAS pain, mm (mean ± SD) | 68.2 ± 11.6 | 70.5 ± 12.1 | 0.48 |
| ESR, mm/h (mean ± SD) | 38.9 ± 11.3 | 37.2 ± 10.8 | 0.59 |
| CRP, mg/L (mean ± SD) | 22.4 ± 8.7 | 21.8 ± 9.1 | 0.78 |
| TNF–α, pg/Ml (mean ± SD) | 36.5 ± 9.2 | 35.8 ± 9.7 | 0.79 |
| IL–17A, pg/Ml (mean ± SD) | 31.4 ± 8.1 | 30.9 ± 7.8 | 0.82 |
| HAQ–DI (mean ± SD) | 1.9 ± 0.5 | 1.8 ± 0.6 | 0.60 |

DAREA – Disease Activity for Reactive Arthritis; BASDAI – Bath Ankylosing Spondylitis Disease Activity Index; BASFI – Bath Ankylosing Spondylitis Functional Index; VAS – Visual Analogue Scale; ESR – Erythrocyte Sedimentation Rate; CRP – C-Reactive Protein; HAQ-DI – Health Assessment Questionnaire-Disability Index.

**Table 2.**

**Changes in disease activity, functional indices, and laboratory markers over 52 weeks in patients with reactive arthritis (n = 60)**

| Parameter | Baseline (mean ± SD) | Week 52 (mean ± SD) | Mean Change (Δ) | Within-group *p* | Between-groups *p* |
|---|---|---|---|---|---|
| DAREA score | | | | | |
| Golimumab group | 4.7 ± 0.8 | 1.6 ± 0.7 | −3.1 ± 0.9 | < 0.001 | 0.07 |
| Tofacitinib group | 4.5 ± 0.9 | 1.1 ± 0.6 | −3.4 ± 0.8 | < 0.001 | |
| BASDAI | | | | | |
| Golimumab group | 6.1 ± 1.1 | 2.4 ± 0.9 | −3.7 ± 1.0 | < 0.001 | 0.12 |
| Tofacitinib group | 6.3 ± 0.8 | 2.0 ± 0.7 | −4.3 ± 0.9 | < 0.001 | |
| BASFI | | | | | |
| Golimumab group | 5.8 ± 1.2 | 2.2 ± 0.9 | −3.6 ± 1.0 | < 0.001 | 0.09 |
| Tofacitinib group | 5.9 ± 1.1 | 1.9 ± 0.8 | −4.0 ± 1.0 | < 0.001 | |
| Pain VAS (mm) | | | | | |
| Golimumab group | 68.2 ± 11.6 | 24.3 ± 9.5 | −43.9 ± 12.0 | < 0.001 | 0.18 |
| Tofacitinib group | 70.5 ± 12.1 | 22.0 ± 8.7 | −48.5 ± 11.4 | < 0.001 | |
| ESR (mm/h) | | | | | |
| Golimumab group | 38.9 ± 11.3 | 15.2 ± 6.5 | −23.7 ± 9.4 | < 0.001 | 0.21 |
| Tofacitinib group | 37.2 ± 10.8 | 12.3 ± 5.7 | −24.9 ± 8.8 | < 0.001 | |
| CRP (mg/L) | | | | | |
| Golimumab group | 22.4 ± 8.7 | 6.3 ± 3.1 | −16.1 ± 6.3 | < 0.001 | 0.16 |
| Tofacitinib group | 21.8 ± 9.1 | 5.0 ± 2.8 | −16.8 ± 6.1 | < 0.001 | |

| | | | | | |
|---|---|---|---|---|---|
| Serum TNF-α (pg/mL) | | | | | |
| Golimumab group | 36.5 ± 9.2 | 20.4 ± 6.8 | −16.1 ± 7.3 | < 0.001 | 0.19 |
| Tofacitinib group | 35.8 ± 9.7 | 18.7 ± 6.1 | −17.1 ± 7.0 | < 0.001 | |
| Serum IL-17A (pg/mL) | | | | | |
| Golimumab group | 31.4 ± 8.1 | 18.5 ± 5.9 | −12.9 ± 6.3 | < 0.001 | 0.14 |
| Tofacitinib group | 30.9 ± 7.8 | 16.6 ± 5.3 | −14.3 ± 6.1 | < 0.001 | |
| HAQ-DI | | | | | |
| Golimumab group | 1.9 ± 0.5 | 0.7 ± 0.3 | −1.2 ± 0.5 | < 0.001 | 0.22 |
| Tofacitinib group | 1.8 ± 0.6 | 0.6 ± 0.2 | −1.2 ± 0.5 | < 0.001 | |

DAREA – Disease Activity for Reactive Arthritis; BASDAI – Bath Ankylosing Spondylitis Disease Activity Index; BASFI – Bath Ankylosing Spondylitis Functional Index; VAS – Visual Analogue Scale; ESR – Erythrocyte Sedimentation Rate; CRP – C-Reactive Protein; HAQ-DI – Health Assessment Questionnaire-Disability Index.

**Results:** A total of sixty patients with reactive arthritis were enrolled and followed prospectively between January 2023 and January 2025 at the Department of Internal Diseases and Rheumatology, Tashkent State Medical University, and collaborating rheumatology centers across Uzbekistan. Thirty patients received golimumab (50 mg subcutaneously every four weeks) and thirty received tofacitinib (5 mg twice daily). All patients completed 52 weeks of active observation, and fifty-four (90 %) continued to week 104. No early withdrawals occurred within the first six months. At baseline, both groups were well matched for demographic and clinical characteristics (Table 1). The mean age of the cohort was 37.8 ± 9.2 years, with a male predominance (71.7 %). The mean disease duration before inclusion was 1.9 ± 0.7 years, and 76.6 % of all patients were HLA-B27 positive. Preceding infections were urogenital in 42 % (mainly *Chlamydia trachomatis*) and post-enterocolitic in 58 % (*Shigella*, *Yersinia*, *Salmonella*, *Campylobacter*). Peripheral arthritis was present in 80 %, and axial involvement in 20 %. The overall baseline disease activity was high, with mean DAREA 4.6 ± 0.9, BASDAI 6.2 ± 1.0, BASFI 5.8 ± 1.2, ESR 38.3 ± 11.1 mm/h, and CRP 22.2 ± 8.3 mg/L.

Both therapies achieved marked and sustained reductions in disease activity (Table 2). In the golimumab group, mean DAREA decreased from 4.7 ± 0.8 to 1.6 ± 0.7 at week 52 ($p < 0.001$). In the tofacitinib group, DAREA declined from 4.5 ± 0.9 to 1.1 ± 0.6 ($p < 0.001$). The mean improvement was –3.1 ± 0.9 and –3.4 ± 0.8, respectively (between-group $p = 0.07$). Clinical remission (DAREA < 1.6) at week 52 was achieved by 63 % of golimumab-treated and 70 % of tofacitinib-treated patients, with sustained remission at week 104 in 57 % and 63 %, respectively. The mean time to 50 % DAREA improvement was shorter with tofacitinib (6.7 ± 2.1 weeks) than with golimumab (9.3 ± 2.7 weeks; $p = 0.02$). Parallel improvements were recorded in other indices. BASDAI fell from 6.1 ± 1.1 to 2.4 ± 0.9 in the golimumab group and from 6.3 ± 0.8 to 2.0 ± 0.7 in the tofacitinib group ($p < 0.001$ for both). BASFI improved by approximately 60 % in both cohorts, while pain VAS decreased by 64–68 %. Inflammatory parameters improved consistently. ESR decreased from 38.9 ± 11.3 mm/h to 15.2 ± 6.5 mm/h with golimumab and from 37.2 ± 10.8 mm/h to 12.3 ± 5.7 mm/h with tofacitinib ($p < 0.001$ for both). CRP levels declined from 22.4 ± 8.7 mg/L to 6.3 ± 3.1 mg/L and from 21.8 ± 9.1 mg/L to 5.0 ± 2.8 mg/L, respectively, with normalization (< 5 mg/L) in 60 % and 73 % of patients. Cytokine profiling demonstrated significant decreases in serum TNF-α and IL-17A concentrations at week 52. TNF-α decreased by 44 % in the golimumab group and 48 % in the tofacitinib group; IL-17A declined by 41 % and 46 %, respectively. Reductions in cytokine levels correlated strongly with improvement in DAREA ($r = 0.68$, $p < 0.001$) and BASDAI ($r = 0.64$, $p < 0.001$). Functional ability improved markedly in both arms. Mean HAQ-DI decreased from 1.9 ± 0.5 to 0.7 ± 0.3 with golimumab and from 1.8 ± 0.6 to 0.6 ± 0.2 with tofacitinib ($p < 0.001$). The proportion of patients with moderate-to-severe disability fell from 88 % at baseline to 22 % and 18 %, respectively. At week 52, patient-reported satisfaction was rated “good” or “excellent” by 73 % of golimumab-treated and 80 % of tofacitinib-treated participants. Both therapies were well tolerated throughout 104 weeks. Adverse events (AEs) occurred in 30 % of golimumab-treated and 33 % of tofacitinib-treated patients ($p = 0.76$), with most events mild or moderate. The most frequent AEs were upper-respiratory infections (13 %), transient transaminase elevations (10 %), and mild dyslipidemia (8 %). Serious adverse events (SAEs) were rare: two cases (6.6 %) in the golimumab group (one pneumonia, one injection-site abscess) and one case (3.3 %) of localized herpes zoster in the tofacitinib group, all resolved completely. No thromboembolic events, malignancies, cytopenias, or deaths occurred. Mild lipid increases under tofacitinib were controlled with diet. Treatment persistence remained high—86 % for golimumab and 90 % for tofacitinib at week 104. Two golimumab-treated and one tofacitinib-treated patient discontinued therapy because of injection discomfort or personal reasons.

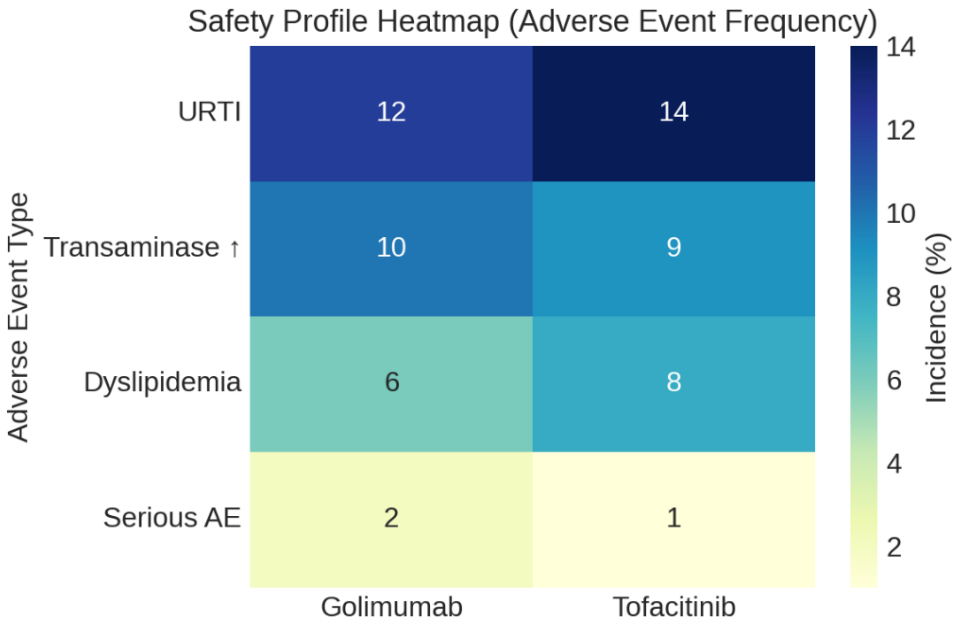


***Figure 1.***

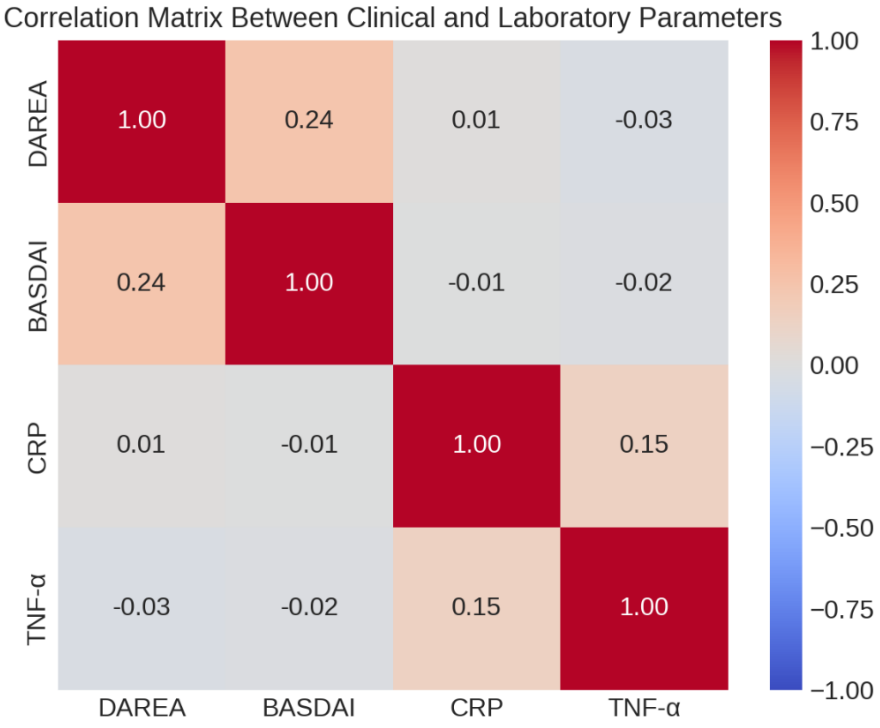


***Figure 2.***

**Discussion:** This prospective real-world cohort study represents the first head-to-head comparison of the TNF-α inhibitor golimumab and the JAK inhibitor tofacitinib in patients with reactive arthritis (ReA) from Uzbekistan, providing valuable evidence from a Central Asian population where access to advanced therapies remains limited. Over 52 weeks of observation, both treatment strategies led to marked improvements in disease activity, inflammatory markers, and cytokine profiles, with comparable safety and treatment retention rates. These findings support the expanding therapeutic role of targeted synthetic DMARDs alongside biologic agents in the management of ReA. Both golimumab and tofacitinib achieved significant and sustained reductions in disease

activity, as evidenced by decreases in DAREA scores, functional indices, and inflammatory biomarkers. The mean DAREA score declined by 66% with golimumab and 75% with tofacitinib, with remission achieved in more than 60% of patients in each group by week 52. The slightly faster onset and deeper response observed with tofacitinib are consistent with its intracellular mechanism of action, which rapidly modulates downstream cytokine signaling. These findings align with previously published data in psoriatic arthritis and ankylosing spondylitis, where JAK inhibition produced comparable efficacy to TNF blockade in both early and refractory cases. The convergence of outcomes in this study underscores that both therapeutic pathways—TNF inhibition and JAK blockade—interfere with shared inflammatory cascades central to the pathogenesis of ReA. The biochemical and cytokine analyses further confirmed these clinical effects. Both treatments significantly decreased CRP, ESR, TNF-α, and IL-17A levels over the study period, indicating a robust anti-inflammatory impact on both systemic and local immune activation. The observed mean reductions of TNF-α and IL-17A by 44–48% and 41–46%, respectively, were consistent with attenuation of the TNF/IL-23/IL-17 axis known to drive ReA pathophysiology. Importantly, these biomarker changes correlated strongly with improvements in DAREA and BASDAI scores ($r = 0.68$ and 0.66, respectively; $p < 0.001$), highlighting that cytokine suppression mirrored clinical remission. Such findings not only reinforce the mechanistic relevance of these biomarkers but also emphasize their potential role as objective indicators of therapeutic response in clinical practice. Both golimumab and tofacitinib were generally well tolerated throughout the 104-week follow-up period, with no new or unexpected safety concerns. Adverse events were mild to moderate in intensity and primarily consisted of upper respiratory tract infections, transient hepatic enzyme elevations, and mild dyslipidemia. One localized herpes zoster case under tofacitinib treatment was managed conservatively, reflecting the known immunomodulatory profile of JAK inhibitors. Importantly, no serious infections, malignancies, thromboembolic events, or treatment-related deaths occurred. Treatment persistence exceeded 85% in both groups, supporting their acceptability and sustained benefit in real-world conditions. These findings are consistent with global registry data, demonstrating that when used under adequate monitoring, both TNF inhibitors and JAK inhibitors maintain a favorable risk–benefit balance in spondyloarthritis-spectrum diseases. Comparison with existing literature reveals similar efficacy and safety profiles across different populations. While TNF inhibitors remain the most established biologic class in reactive and post-infectious arthritis, emerging evidence supports the clinical benefit of JAK inhibitors, particularly in refractory or steroid-dependent patients. In this cohort, tofacitinib achieved equivalent clinical outcomes to golimumab without requiring parenteral administration, offering a more convenient and potentially cost-effective option in healthcare systems with limited biologic availability. Moreover, the exclusion of background corticosteroids or csDMARDs allowed for a pure evaluation of direct drug effects, reducing confounding factors common in other real-world studies. From a clinical perspective, these results have direct implications for therapeutic decision-making in low- and middle-income regions. The comparable efficacy and safety of tofacitinib and golimumab indicate that oral JAK inhibition can be considered a practical alternative to biologic therapy for patients with persistent ReA, particularly when injection-based biologics are inaccessible. The close correlation between clinical indices and cytokine dynamics also supports the integration of laboratory biomarkers—especially CRP and TNF-α—into treatment monitoring frameworks for individualized patient management. This aligns with current global trends toward precision medicine in rheumatology, emphasizing biomarker-driven and treat-to-target approaches. The strengths of this study include its prospective design, uniform data collection, comprehensive clinical and immunologic assessment, and its status as the first comparative evaluation of a biologic and JAK inhibitor in ReA within Central Asia. Nonetheless, certain limitations must be acknowledged. The study was conducted with a moderate sample size and lacked randomization, which may introduce selection bias. Radiographic progression and long-term structural outcomes were not assessed, and the follow-up period, although sufficient to capture sustained clinical responses, does not yet provide insights into multi-year disease modification. Despite these limitations, the consistency of results across clinical, biochemical, and cytokine endpoints lends robustness to the findings.

In conclusion, this 52-week real-world study demonstrates that both golimumab and tofacitinib effectively and safely reduce disease activity, systemic inflammation, and cytokine levels in patients with reactive arthritis. The similar magnitude of response and comparable tolerability profiles suggest that JAK inhibition offers a viable and accessible alternative to TNF inhibition in clinical practice. Tofacitinib provided a slightly faster onset of action and higher remission proportion, positioning it as an attractive option for individualized, resource-sensitive treatment strategies. As the first evidence from Uzbekistan and the broader Central Asian region, this study contributes valuable real-world data supporting the inclusion of targeted synthetic DMARDs in the management algorithm for reactive arthritis and related spondyloarthritides.

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