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Original Article

Efficacy and Baseline Predictors of Tofacitinib Response in Patients with Rheumatoid Arthritis at a Private Tertiary Care Hospital in Pakistan

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ABSTRACT

Objective: To analyze the efficacy of Tofacitinib and predictors of response in Rheumatoid Arthritis patients

Study Design: Prospective longitudinal study

Place and Duration of Study: Rheumatology Department, Fauji Foundation Hospital, Rawalpindi, Pakistan, from Mar to Sep 2024.

Methodology: The study included 76 adult RA patients (75 females and 1 male), who were advised to take Tofacitinib. We recorded the DAS-28 ESR at baseline and after 3 months and noted patient demographics, BMI, serology, and concomitant DMARD use. The primary outcome was remission/low disease activity (DAS-28≤3.2) at 3 months. DAS-28 levels were compared by Wilcoxon test. Treatment outcome was categorized as a good response and poor response. Predictors of treatment response were analyzed using univariate logistic regression analysis.

Results: The median age of patients was 50.50(IQR 57.00 - 45.25) years. Median RA duration was 9.00(IQR 15.00 – 6.00) years with 60(78.95%) patients being seropositive. Conventional DMARD was continued in combination with Tofacitinib in 55(72.4%) patients. Only 11(14.5%) patients achieved the treatment target. Tofacitinib therapy significantly reduced DAS-28 from baseline of 5.64 (IQR 5.99 - 5.08), to 3.99±0.76 at 3 months (*p*-value <0.001). Lower baseline DAS-28 predicted an effective treatment response (OR 4.13, 95%CI 1.07-16.03, *p*-value 0.03). Tofacitinib monotherapy predicted a good response in comparison to Tofacitinib combination with conventional DMARDs (OR 4.00, 95% CI1.06-14.96, *p*-value 0.03). Baseline BMI, inflammatory markers, and serology did not have an impact on treatment response.

Conclusion: Tofacitinib therapy significantly reduced DAS-28 ESR at 3 months with baseline predictive factors such as BMI and serology having no impact on treatment response.

Keywords: Tofacitinib, Efficacy, BMI, Serology, Inflammatory, Rheumatoid Arthritis

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INTRODUCTION

Rheumatoid Arthritis is one of the most common chronic inflammatory conditions in the world, which, if left untreated, can lead to significant deformities and a decline in overall quality of life. Over time, we have come to understand the profound impact of this disease on not only the joints but also bone health and cardiovascular disease.[1] The optimum management of this disease is the prime objective of physicians to limit the disability and misery of the patient.

The use of conventional synthetic Disease Modifying Anti-Rheumatic Drugs has proved to be ground-breaking, with a majority of the patients achieving good disease control within a matter of months. Methotrexate is the recommended first-line agent prescribed to treatment naïve patients, but a substantial proportion of patients, around 30%.[2] have inadequate response to these agents or cannot tolerate them.

Biologic agents, such as TNF-inhibitors and IL-6 inhibitors, have long been approved for treatment of Rheumatoid Arthritis, but their frequent dosing, subcutaneous administration, and non-availability due to prohibitive cost make them an unattractive treatment option. The advent of Janus kinase inhibitors has ushered in a new era of options to treat Rheumatoid arthritis since their approval by the FDA[3] and subsequent inclusion in treatment guidelines.[4] Tofacitinib , a second-generation JAK inhibitor, acts by blocking signal transduction and activation of transcription enzymes that initiate the inflammatory cascade responsible for the pathogenesis of Rheumatoid Arthritis.[5]

The use of Tofacitinib is reserved for patients who have failed to respond to at least one conventional synthetic DMARD, with moderate to severe disease, either as monotherapy or

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concomitantly with conventional synthetic DMARDs.6 Efficacy has been demonstrated with trials documenting adequate disease control while also halting the progression of radiological damage[7], and its combination with methotrexate has assessed to be non-inferior to TNF inhibitor and methotrexate.[8]

While the effectiveness of Tofacitinib in Rheumatoid Arthritis is well-established, there is still a lack of research on patient characteristics that make it effective.[9] Positive serology, high inflammatory markers, and radiological damage have earlier been documented to be indicators of poor response to treatment.[10]

Most of the data available to us is from studies conducted internationally. The Asian population has distinct genetic and phenotypic characteristics that can have a profound impact on Tofacitinib response. Since the use of Tofacitinib has increased exponentially in this part of the world, there is a greater need to conduct population-specific research, especially in Pakistan. This will help to recognize patients likely to respond to Tofacitinib and help in making effective treatment plans for these patients with better safety profiles.

METHODOLOGY

A prospective longitudinal study design was opted for this study. It was conducted at the Rheumatology department of Fauji Foundation Hospital, Rawalpindi, Pakistan, from Mar to Sept 2024. Prior ethical approval was obtained from the ethical review committee of the hospital (Letter: 810/RC/FFH/RWP dated 20/03/2024). Informed written consent was taken from the patients before inclusion in the study. The sample size required for this study was 76 with 95% confidence interval, 5% margin of error, and a Tofacitinib response of 94.78%.[11] The sample size was calculated using online free software from www.raosoft.com/samplesize.html

Inclusion Criteria: Adult patients, aged 18 to 65 years, of Rheumatoid Arthritis with moderate to high disease activity (baseline DAS-28>3.2), who have been recommended Tofacitinib therapy for the first time after failure of conventional DMARDs.

Exclusion Criteria: Age younger than 18 years or ≥ 65years. Active infection, previous history of cardiovascular or thromboembolic events. Any contraindication to Tofacitinib. Previously received Tofacitinib or other JAK-inhibitor. Patients with baseline low disease activity with DAS-28 of ≤3.2. Patients naïve to conventional DMARDs. Co-existing liver or renal disease, pregnant females, and those not willing to participate were excluded. Patients who failed to complete at least 3 months of Tofacitinib therapy were also not part of the study.

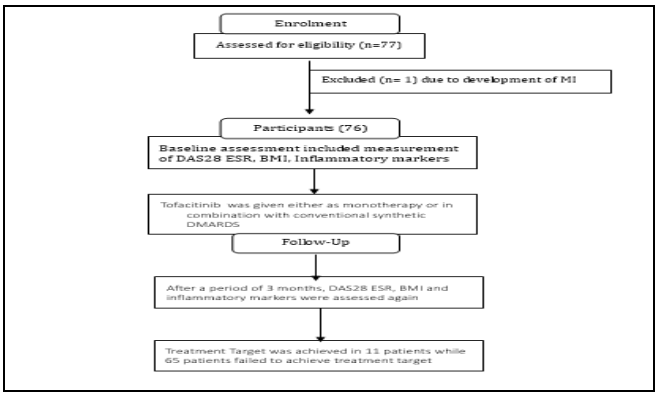

Figure: Patient Flow Diagram

Patients were recruited from the outpatient department using a consecutive convenience sampling technique. A structured proforma was designed for the data collection. Demographic data were collected from the patient after signing the consent form. A comprehensive physical examination was performed by the study investigator, including joint examination, disease activity score calculation using DAS-28 ESR, height, weight, BMI, and examination for extra-articular features of RA. Baseline chemistry was sent.

Serology profile was documented from patients' electronic health records; in case it was not available in the records, serology was sent to the lab along with baseline chemistry as well. The decision to start Tofacitinib was made by the physician treating the patient. The study investigator did not influence the decision to start Tofacitinib. Patients were followed up monthly for 3 months after receiving the Tofacitinib therapy. Assessment at each follow-up visit included repetition of the history, physical examination, and disease activity assessment.

Additionally, a detailed assessment for the side effects of Tofacitinib was made using a checklist of the known side effects documented in literature by detailed history, examination, and laboratory investigations as needed on each visit, and any positive findings were documented. Side effects screened for at follow-up visits included upper respiratory tract infections (nasopharyngitis), diarrhea, nausea, headache, development of rash or skin infections such as herpes zoster. Patients who had

major side effects were excluded from the study. At completion of three months, treatment outcome was documented using DAS-28 ESR. Tablet Tofacitinib was advised at 5mg BID.

The primary outcome of the study was the response of DAS-28 after 3 months of Tofacitinib treatment. The treatment target was defined as the achievement of remission/ low disease activity at 3 months (DAS-28 ≤3.2). The secondary outcome was the assessment of factors predicting the treatment response to Tofacitinib at 3 months. Patients were divided into two groups based on the treatment response at 3 months (response achieved vs not achieved). Patient age, RA duration, serology status, baseline disease activity score, Tofacitinib mono vs combination therapy with conventional synthetic DMARDs, BMI, ESR, and CRP were the predictive factors assessed.

DAS28-ESR is the measurement of disease activity in patients of Rheumatoid Arthritis calculated from total tender joints, swollen joints, pain on a visual analogue scale, and Erythrocyte sedimentation rate via a validated score. It is interpreted as <2.6 indicates remission, 2.6- 3.2 indicates low disease activity, >3.2 indicates active disease with moderate activity, >5.1 indicates high disease activity. ESR and CRP were monitored at baseline and then again at three months to assess disease outcome.

Statistical Package for Social Sciences (SPSS) version 27 was used to analyze the study data. Summary statistics were calculated as frequencies and percentages for the categorical variables. For quantitative variables, distribution was checked first by Shapiro-Wilk's test, the variables with a *p*-Value of >0.05 were considered normally distributed. Age, RA duration, body weight, BMI, and baseline DAS-28 were not normally distributed and were described as median and IQR. Whereas DAS-28 at 3 months and change in DAS-28 from baseline were normally distributed and were described as Mean±SD. DAS-28 levels at baseline were compared with those at 3 months by Wilcoxon test. Patients were divided into groups on the basis of achievement of treatment target at 3 months (good response vs poor response). Predictors of treatment response were analyzed using univariate binary logistic regression analysis. For all the analyses, the level of significance was considered at a *p*-value of less than 0.05.

RESULTS

A total of 77 patients were included in the study. One patient experienced a major cardiovascular event at 2 months, leading to discontinuation of Tofacitinib therapy. Consequently, that patient was excluded from the study. The aforementioned patient was included in the side effect analysis but not in the rest of the analyses because he did not complete three months of study treatment. The study population consisted of 75 females and 1 male, with a median age of 50.5(57.00-45.25) years. All the patients had established rheumatoid arthritis with a median disease duration of 9.00(15.00 – 6.00) years. 60(78.95%) were seropositive. All the patients had used conventional DMARDs as monotherapy or a combination of dual or triple DMARDs in varied combinations. 7(9.2%) patients had received biologic therapy in the past. The biologic used in all these patients was Rituximab. Conventional DMARD was continued in combination with Tofacitinib in 55(72.4%) patients. Table-I summarizes the baseline characteristics of the study population.

Table-I: Baseline characteristics of RA patients

Characteristics	Values
Gender	
Female	75(98.7%)
Male	1(1.3%)
Median Age (years)	50.50(57.00 - 45.25)
Median Ra duration (years)	9.00(15.00 - 6.00)
Median DAS-28 ESR	5.64 (5.99 - 5.08)
Use of conventional synthetic DMARDs	
Methotrexate	5(6.6%)
Leflunomide	1(1.3%)
Methotrexate and Leflunomide	34(44.7%)
Methotrexate, Leflunomide and Sulfasalazine	26(34.2%)
Methotrexate and Sulfasalazine	10(13.2%)
Smoking status	
Non-smoker	70(92.1%)
Smoker	2(2.6%)
Ex-smoker	2(2.6%)
Naswar-user	2(2.6%)
Previous biologic use	
Yes	7(9.2%)
No	69(90.2%)
Serology status	
RF positive	49(64.5%)
RF and anti-CCP positive	7(9.2%)
Anti-CCP positive	4(5.3%)
Seronegative	16(21.05%)
Combination therapy	
Methotrexate	28(36.8%)
Leflunomide	26(34.2%)
Sulfasalazine	1(1.3%)
None	21(27.6%)

At baseline median DAS-28 of the patients was 5.63(5.99 - 5.08). Change in DAS-28 score was calculated for each study participant by subtracting the DAS-28 score 3 months from baseline DAS-28 score. The change in the DAS-28 was statistically significant with a *p*-value of <0.001. Treatment target was achieved in 11(14.5%) patients at the end of 3 months, whereas 65(85.5%) failed to achieve the treatment target.

At the end of three months, 11 patients achieved the treatment target (good response), whereas 65 patients did not achieve the treatment target defined for the study (poor response). Patients with lower DAS28 at baseline were 4 times more likely to achieve good response at 3 months as compared to the patients with higher baseline DAS-28 score (95%CI 1.07-16.03, p-value 0.03). In comparison to the patients using Tofacitinib in combination with csDMARDs, patients using Tofacitinib monotherapy had significantly higher odds for a good outcome at 3 months (OR 4, 95% CI1.06-14.96, *p*-value 0.03). Baseline ESR and CRP values did not significantly affect the outcome at three months. Similarly, serology status was not associated with the disease outcome. Table-II show the results of regression analysis for predictors of response to Tofacitinib treatment.

Adverse reactions were noted in 6 (7.9%) patients; abdominal pain in 3(3.9%), skin lesions in 2(2.6%), Myocardial infarction in 1(1.3%). Treatment was stopped in the patient who experienced MI, and she was excluded from the study.

DISCUSSION

The study participants showed that Tofacitinib significantly reduced the disease activity scores after 3 months, regardless of whether used alone or in combination with conventional synthetic DMARDs. However, despite the statistically significant reductions, the treatment target of remission or low disease activity was achieved in only 14.5% of the patients. Comparable results were observed in a placebo-controlled trial of Tofacitinib monotherapy in patients with Rheumatoid Arthritis, with 12.5% of patients achieving DAS-28 ESR of 3.2 or less, and changes in disease scores were also statistically significant. Over the years, the treatment of Rheumatoid Arthritis has undergone many advancements, one of which is the advent of JAK inhibitors.[12] The goal of our study was to determine the effectiveness of Tofacitinib in a tertiary care setting and also to gauge whether baseline predictive factors had an impact on disease response.

Significant change in mean DAS-28 -2.3 was also observed in retrospective analysis of patients at different doses of Tofacitinib compared to placebo, as reported by Bykerk *et al*.[13]

Although the whole patient cohort showed a significant reduction in their disease activity score, the

Table-II: Factors predicting the response to 3 months of Tofacitinib treatment in RA patients (n=76)

Baseline characteristics	Treatment target achieved(good response) n=11	Treatment target not achieved (poor response) n=65	*p*-value	OR	95% CI
Age;Median (IQR)	49.00(57.00 – 40.00)	51.00(57.00 – 46.00)	0.13	1.04	0.98-1.11
RA duration;Median (IQR)	7.00(16.00–5.00)	9.00(15.00 – 6.00)	0.94	1.00	0.90-1.11
Baseline DAS-28 ESR;Median (IQR)	5.04(5.88-4.89)	5.73(6.04-5.22)	0.03	4.14	1.07-16.03
Serology;n(%) Sero-positive Sero-negative(ref)	 9(81.8) 2(18.2)	 51(78.5) 14(21.5)	0.80	1.24	0.23-6.38
csDMARDs status; n(%) No Yes (ref)	 6(54.5) 5(45.5)	 15(23.1) 50(76.9)	0.03	4.00	1.06-14.96
BMI;Median (IQR)	29.30(32.40-24.20)	25.90(28.05-23.5)	0.19	1.09	0.96-1.23
ESR;Median (IQR)	32.00(38.00-25.00)	30.00(35.00-28.00)	0.99	1.00	0.91-1.09
CRP;Median (IQR)	55.60(100.00-39.20)	85.50(100.00-54.56)	0.09	0.98	0.99-1.00

**csDMARD - conventional synthetic disease-modifying antirheumatic drug*
RA – Rheumatoid Arthritis
ESR – Erythrocyte Sedimentation Rate
BMI – Basic Metabolic Index
CRP – C-Reactive Protein

poor responders showed a lower mean reduction in their disease activity score compared to the good responders. Tofacitinib begins to function as soon as two weeks, as reported by D Alessandor *et al.*,[14], but the above findings may indicate that while some patients may respond well to Tofacitinib early on, patients with aggressive disease may need a longer duration of therapy to show a meaningful response as explained by Muller *et al.*[15] This is in line with analysis from a prior post HOC study that indicates that lower baseline DAS-ESR are predictors of better response in the short term, while those patients with higher disease activity at baseline were more likely to have greater radiological damage and longer duration of disease, and thus require a longer time to respond.

On the other hand, poor responders may have unique patient or disease-related factors, making these patients resistant to Tofacitinib. One of the patient-specific factors identified was prior exposure to biologic DMARDs that would result in a longer time to achieve remission. Retrospective analysis of patients in Latin America assessed patients with higher disease activity at baseline, longer duration of disease, and having received prior biologic treatment to be likely to respond poorly to Tofacitinib. The biologic naïve patients were also seen to be better responders to Tofacitinib in a retrospective study by Poudel *et al.*[16]

It was analyzed whether the continuation of the cs DMARD has any effect on the response. The study highlighted that usage of cs DMARD in combination with Tofacitinib was associated with 4 times higher odds of poor response. Since the treatment decisions were made by the treating physicians, the higher proportion of combination therapy in poor responders may be due to the physician's preference for continuing the cs DMARD along with Tofacitinib in patients with aggressive disease, rather than a causative factor for the poor response. Oral strategy trial of Tofacitinib monotherapy was found to be inferior when compared to Adalimumab and Methotrexate combination therapy, and Tofacitinib and Methotrexate combination therapy.[17]

Baseline BMI was not found to be a factor in patients who had a better response to Tofacitinib. Previously, obesity was thought to complicate the assessment of disease activity in patients with RA, as it is associated with chronic pain, disability, and depression. but the effectiveness of Tofacitinib was not statistically significant across different classes of BMI.[18]

The seropositivity status of the patients was also analyzed. Data have shown that dual seropositive patients have traditionally responded better to Tofacitinib compared to seronegative patients, but it was not a significant factor in our study. Baseline ESR and CRP levels did not have an impact on response, although it was found in a study by Desai *et al.*, after collecting data from multiple studies, that higher baseline CRP levels yield a better response to Tofacitinib.[19,20]

Apart from a few minor side effects, one patient developed ischemic heart disease and had to undergo Angioplasty. It has been noted that Tofacitinib can cause increased levels of low-density lipoproteins that can potentially cause an increased risk of cardiovascular disease.

LIMITATION OF STUDY

The obvious limitations of the study are the small sample size, with only one of the patients being male. This was because our hospital serves the families of retired government officials, and patient pool mostly consisted of females. Thus, the findings of our study cannot be generalized to the entire population. The effects of smoking and prior use of biologics could not be assessed as there were not enough patients with these characteristics to make an effective comparison. Furthermore, steroid usage was not quantified, which could impact the treatment response. The highlighted areas and deficiencies in this study can be exciting prospects for research in the future.

CONCLUSION

Tofacitinib significantly reduces the disease activity in RA patients with inadequate response to previous therapies. Prompt response to Tofacitinib is influenced by the disease activity scores at the time of treatment initiation. Tofacitinib's impact on disease activity is independent of the acute phase reactants and serology.

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Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

MA & SP: Data acquisition, data analysis, critical review, approval of the final version to be published.

SY & MF: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

SS & HG & BS & AN : Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity

of any part of the work are appropriately investigated and resolved.

REFERENCES

1. Dijkshoorn B, Raadsen R, Nurmohamed MT. Cardiovascular Disease Risk in Rheumatoid Arthritis: Anno 2022. J Clin Med 2022; 11(10): 2704. https://doi:10.3390/jcm11102704.
2. Bayrak ED, Aktas I. Efficacy and gastrointestinal tolerability of methotrexate in late-onset rheumatoid arthritis patients: a prospective cohort study. Egypt Rheumatol Rehabil 2023; 50:8. https://doi:10.1186/s43166-023-00178-w
3. Chatzidionysiou K, Emamikia S, Nam J, Ramiro S, Smolen J, van der Heijde D, et al. Efficacy of glucocorticoids, conventional and targeted synthetic disease-modifying antirheumatic drugs: a systematic literature review informing the 2016 update of the EULAR recommendations for the management of rheumatoid arthritis. Ann Rheum Dis 2017; 76(6): 1102–1107. https://doi:10.1136/annrheumdis-2016-210711
4. Van der Heijde D, Tanaka Y, Fleischmann R, Keystone E, Kremer J, Zerbini C, et al. Tofacitinib (CP-690,550) in patients with rheumatoid arthritis receiving methotrexate: Twelve-month data from a twenty-four-month phase III randomized radiographic study. Arthritis Rheum 2013; 65(3): 559–570. https://doi:10.1002/art.37816
5. Fleischmann R, Mysler E, Hall S, Kivitz AJ, Moots RJ, Luo Z, et al. Efficacy and safety of tofacitinib monotherapy, tofacitinib with methotrexate, and adalimumab with methotrexate in patients with rheumatoid arthritis (ORAL Strategy): a phase 3b/4, double-blind, head-to-head, randomised controlled trial. Lancet 2017; 390(10093): 457-468. https://doi:10.1016/S0140-6736(17)31618-5.
6. Fautrel B, Nab HW, Brault Y, Gallo G. Identifying patients with rheumatoid arthritis with moderate disease activity at risk of significant radiographic progression despite methotrexate treatment. RMD Open 2015; 1(1): e000018. https://doi:10.1136/rmdopen-2014-000018
7. Naseem S, Wani R, Yousaf J, Amjad A, Abbas Raja A, Qandeel M, et al. Efficacy and Safety of Tofacitinib in Patients With Rheumatoid Arthritis and Inadequate Response to Methotrexate: A Real-World Study. Cureus 2025; 17(10): e94745. https://doi:10.7759/cureus.94745.
8. Singh JA, Hossain A, Tanjong Ghogomu E, Mudano AS, Maxwell LJ, Buchbinder R, et al. Biologics or tofacitinib for people with rheumatoid arthritis unsuccessfully treated with biologics: a systematic review and network meta-analysis. Cochrane Database Syst Rev 2017; 3(3): CD012591. https://doi:10.1002/14651858.CD012591.
9. Lee S, Kang S, Eun Y, Won HH, Kim H, Lee J, Koh EM, Cha HS. Machine learning-based prediction model for responses of bDMARDs in patients with rheumatoid arthritis and ankylosing spondylitis. Arthritis Res Ther 2021; 23(1): 254. https://doi:10.1186/s13075-021-02635-3.
10. Sebastião Cézar Radominski, Cardiel MH, Citera G, Goecke A, Juan José Jaller, Barranjard A, et al. Tofacitinib , an oral Janus kinase inhibitor, for the treatment of Latin American patients with rheumatoid arthritis: Pooled efficacy and safety analyses of Phase 3 and long-term extension studies. Reumatología Clínica 2017; 13(4): 201–209. https://doi:10.1016/j.reuma.2016.04.010
11. Angelini J, Talotta R, Roncato R, Fornasier G, Barbiero G, Dal Cin L, et al. JAK-Inhibitors for the Treatment of Rheumatoid Arthritis: A Focus on the Present and an Outlook on the Future. Biomolecules 2020; 10(7): 1002. https://doi:10.3390/biom10071002.
12. Kucharz EJ, Stajszczyk M, Kotulska-Kucharz A, Batko B, Brzosko M, Jeka S, et al. Tofacitinib in the treatment of patients with rheumatoid arthritis: position statement of experts of the Polish Society for Rheumatology. Reumatologia 2018; 56(4): 203-211. https://doi:10.5114/reum.2018.77971.
13. Bykerk VP, Lee EB, van Vollenhoven R, Gruben DC, Fallon L, Woolcott JC, et al. Identification of Distinct Disease Activity Trajectories in Methotrexate-Naive Patients With Rheumatoid Arthritis Receiving Tofacitinib Over Twenty-Four Months. Arthritis Care Res 2022; 74(1): 131-141. https://doi:10.1002/acr.24709.
14. D'Alessandro F, Cazzato M, Laurino E, Morganti R, Bardelli M, Frediani B, et al. ToRaRI (Tofacitinib in Rheumatoid Arthritis a Real-Life experience in Italy): Effectiveness, safety profile of tofacitinib and concordance between patient-reported outcomes and physician's global assessment of disease activity in a retrospective study in Central-Italy. Clin Rheumatol 2024; 43(2): 657-665. https://doi:10.1007/s10067-023-06836-w
15. Mueller RB, Hasler C, Popp F, Mattow F, Durmisi M, Souza A, Hasler P, et al. Effectiveness, Tolerability, and Safety of Tofacitinib in Rheumatoid Arthritis: A Retrospective Analysis of Real-World Data from the St. Gallen and Aarau Cohorts. J Clin Med 2019; 8(10): 1548. https://doi:10.3390/jcm8101548.
16. Poudel D, George MD, Baker JF. The Impact of Obesity on Disease Activity and Treatment Response in Rheumatoid Arthritis. Curr Rheumatol Rep 2020; 22(9): 56 https://doi:10.1007/s11926-020-00933-4
17. Dikranian AH, Gonzalez-Gay MA, Wellborne F, Álvaro-Gracia JM, Takiya L, Stockert L, et al. Efficacy of Tofacitinib in patients with rheumatoid arthritis stratified by baseline body mass index: an analysis of pooled data from phase 3 studies. RMD open 2022; 8(1): e002103. https://doi:10.1136/rmdopen-2021-002103
18. Bird P, Hall S, Nash P, Connell CA, Kwok K, Witcombe D, et al. Treatment outcomes in patients with seropositive versus seronegative rheumatoid arthritis in Phase III randomized clinical trials of Tofacitinib . RMD Open 2019; 5(1): e000742. https://doi:10.1136/rmdopen-2018-000742
19. Desai RJ, Pawar A, Khosrow-Khavar F, Weinblatt ME, Kim SC. Risk of venous thromboembolism associated with tofacitinib in patients with rheumatoid arthritis: a population-based cohort study. Rheumatol 2021; 61(1): 121-130. https://doi:10.1093/rheumatology/keab294.
20. Khosrow-Khavar F, Desai RJ, Lee H, Lee SB, Kim SC. Tofacitinib and Risk of Malignancy: Results From the Safety of Tofacitinib in Routine Care Patients With Rheumatoid Arthritis (STAR-RA) Study. Arthritis Rheumatol 2022; 74(10): 1648-1659. https://doi:10.1002/art.42250.

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