

Original Article

Tofacitinib demonstrates favorable efficacy but an increased risk of herpes zoster in elderly patients with rheumatoid arthritis: a retrospective cohort analysis

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Received March 16, 2026; Accepted May 22, 2026; Epub July 15, 2026; Published July 30, 2026

Abstract: Objective: To evaluate the efficacy of tofacitinib in elderly patients with rheumatoid arthritis (RA) and analyze herpes zoster risk factors. Methods: A retrospective analysis included elderly RA patients from January 2022 to June 2024. Patients were assigned to a tofacitinib group (n=62, tofacitinib + methotrexate) or a conventional disease-modifying antirheumatic drugs (DMARDs) group (n=58, methotrexate + leflunomide). Clinical efficacy, inflammatory markers, American College of Rheumatology (ACR) response rates, joint function, herpes zoster incidence, and other adverse reactions were assessed. Results: After treatment, the tofacitinib group had lower Disease Activity Score 28-erythrocyte sedimentation rate (ESR) scores than the conventional DMARDs group ($P<0.05$). ESR, C-reactive protein, and rheumatoid factor levels were significantly lower ($P<0.05$), while ACR20/50/70 response rates were significantly higher in the tofacitinib group ($P<0.05$). Health Assessment Questionnaire Disability Index scores, morning stiffness duration, and pain scores were also lower ($P<0.05$). Herpes zoster incidence was higher in the tofacitinib group (12.90%) than that in the conventional DMARDs group (3.45%; $P<0.05$). Multivariable logistic regression identified tofacitinib use, age ≥ 75 years, glucocorticoid use, reduced lymphocyte count, and prior herpes zoster as independent risk factors ($P<0.05$). Conclusions: Tofacitinib is efficacious in elderly RA patients, improving disease activity and joint function, but increases herpes zoster risk. High-risk patients require monitoring; prophylactic vaccination may be considered.

Keywords: Tofacitinib, rheumatoid arthritis, elderly, herpes zoster, adverse reaction

Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune disorder mainly characterized by symmetrical polyarthritis, resulting in joint destruction, deformity, and loss of function. Elderly patients with RA face greater challenges in treatment and management owing to declining immune function, multiple comorbidities, and reduced drug tolerance [1, 2]. In recent years, with increasing understanding of the pathogenesis of RA, Janus kinase (JAK) inhibitors, a novel class of oral small-molecule targeted drugs, have been increasingly used in clinical practice [3].

As the first approved JAK inhibitor for RA, tofacitinib selectively inhibits JAK1 and JAK3 and blocks inflammatory cytokine signaling, thereby

exerting anti-inflammatory and immunomodulatory effects [4]. Numerous studies have demonstrated the efficacy of tofacitinib in reducing disease activity in RA and alleviating joint symptoms [5, 6]. However, because the JAK pathway plays a key role in immune responses, the use of JAK inhibitors may increase the risk of infection, particularly herpes zoster [7]. Herpes zoster is caused by reactivation of varicella-zoster virus (VZV), and its risk is significantly increased in elderly individuals due to age-related decline in cellular immunity [8]. Previous research has indicated that the incidence of herpes zoster in patients with RA treated with tofacitinib ranges from 3.9% to 8.0%, significantly higher than that in those treated with conventional disease-modifying antirheumatic drugs (DMARDs) [9]. However, relatively few studies in China have explored the risk of herpes zoster in elderly

patients with RA receiving tofacitinib, and the associated risk factors remain to be clarified.

In the present retrospective cohort study, we compared the clinical efficacy of tofacitinib with that of conventional DMARDs in elderly patients with RA, with a focus on the occurrence of herpes zoster and its associated risk factors. This study may provide evidence to support rational drug use and optimize treatment strategies in this population.

Materials and methods

Research design

This retrospective cohort study was permitted by the Ethics Committee of Hangzhou Fuyang District Hospital of Traditional Chinese Medicine. It was carried out following the Declaration of Helsinki. Using the hospital information system, we included elderly patients with RA who were admitted as inpatients or outpatients to our institution between January 2022 and June 2024.

Patients were included if they met the following criteria: (1) age ≥ 65 years; (2) fulfillment of the 2010 American College of Rheumatology (ACR)/European League Against Rheumatism criteria for RA [10]; (3) poor treatment response or intolerance of conventional DMARDs; (4) receipt of tofacitinib or conventional DMARDs for ≥ 24 weeks; (5) complete clinical records.

Patients who met the following criteria were excluded: (1) concurrent autoimmune diseases; (2) active infections; (3) malignancies; (4) severe dysfunctions of the liver or kidneys; (5) use of biological agents within 6 months prior to enrollment; (6) interruption of medication for more than 4 weeks during treatment.

After applying the above criteria, 120 patients were included. Among them, 62 patients who received tofacitinib plus methotrexate were classified into the tofacitinib group, while 58 patients who received methotrexate plus leflunomide were classified into the conventional DMARDs group.

Sample size was calculated based on the primary outcome measure [Disease Activity Score 28 (DAS28)-erythrocyte sedimentation rate (ESR) score]. According to previous literature, the expected difference between the two gr-

oups was approximately 0.8, with a standard deviation of 1.2. With a two-sided significance level of $\alpha=0.05$ and a statistical power of 80% ($1-\beta=0.80$), a minimum of 36 patients per group was required. After considering a 10% dropout rate, at least 40 patients were required in each group. Although sample size calculation was performed a priori, the final sample size in this retrospective study was ultimately determined by the number of eligible patients available. A total of 62 patients were included in the tofacitinib group and 58 in the conventional DMARDs group, both exceeding the minimum required sample size and ensuring adequate statistical power.

Treatment regimens

In the tofacitinib group, patients received tofacitinib 5 mg twice daily in combination with methotrexate 10-15 mg once weekly. In the conventional DMARDs group, patients received methotrexate 10-15 mg once weekly combined with leflunomide 20 mg once daily. In both groups, adjunctive treatments, including folic acid and nonsteroidal anti-inflammatory drugs, were administered as clinically indicated. Some patients also received low-dose glucocorticoids (prednisone ≤ 10 mg/day).

Observation indicators

Primary outcomes: (1) Disease activity assessment: The level of disease activity was determined based on the DAS28-ESR score [11] before treatment and at 4, 12, and 24 weeks of treatment. (2) Inflammatory markers: ESR, C-reactive protein (CRP), and rheumatoid factor (RF) were detected before treatment and at 12 and 24 weeks of treatment. (3) ACR response rate: The response rates of ACR20, ACR50, and ACR70 were evaluated at 12 and 24 weeks of treatment.

Secondary outcomes: (1) Baseline characteristics: The baseline characteristics, including age, sex, body mass index, course of disease, and comorbidities, were collected. (2) Joint function and quality of life assessment: The Health Assessment Questionnaire Disability Index (HAQ-DI) [12] was used to evaluate joint function. The duration of morning stiffness was recorded. The visual analog scale (VAS) was used to evaluate pain intensity. (3) Occurrence of herpes zoster: The number of cases, onset

Table 1. Comparison of baseline information between the two groups

Characteristic	Tofacitinib group (n=62)	Conventional DMARDs group (n=58)	t/ χ^2	P
Average age (years)	71.85±5.62	72.14±5.89	0.278	0.782
Sex (male/female, cases)	15/47	13/45	0.065	0.799
BMI (kg/m ²)	23.52±3.18	23.86±3.42	0.568	0.571
Disease duration (years)	8.65±4.32	8.42±4.18	0.298	0.766
Hypertension	28 (45.16)	25 (43.10)	0.051	0.821
Diabetes mellitus	18 (29.03)	16 (27.59)	0.032	0.858
History of herpes zoster	4 (6.45)	3 (5.17)	0.089	0.765
Baseline DAS28-ESR score	5.68±0.85	5.72±0.92	0.248	0.805
Concomitant glucocorticoid use	25 (40.32)	22 (37.93)	0.068	0.795

DMARDs: disease-modifying antirheumatic drugs; BMI: body mass index; DAS28-ESR: Disease Activity Score 28-erythrocyte sedimentation rate.

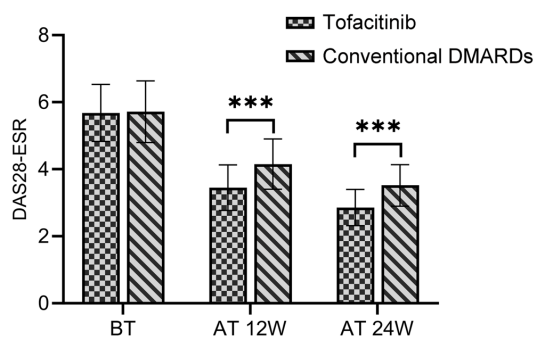


Figure 1. Comparison of pre- and post-treatment DAS28-ESR scores between the tofacitinib and conventional DMARDs groups. The pre-treatment DAS28-ESR scores did not differ significantly between the groups ($P>0.05$). At 12 and 24 weeks, the tofacitinib group exhibited significantly lower DAS28-ESR scores compared with the conventional DMARDs group ($P<0.05$). Note: DAS28-ESR: Disease Activity Score 28-erythrocyte sedimentation rate; DMARDs: disease-modifying antirheumatic drugs; BT: before treatment; AT12W: At 12 weeks after treatment; AT24W: At 24 weeks after treatment. *** $P<0.001$. Repeated measures analysis of variance was used for intergroup comparisons at multiple time points, and Bonferroni correction was used for pairwise comparisons within groups.

time, affected sites, severity, and occurrence of postherpetic neuralgia were recorded. (4) Other adverse reactions: The occurrence of adverse reactions, including upper respiratory tract infection, gastrointestinal discomfort, abnormal liver function, elevated blood lipids, urinary tract infection, headache, and cardiovascular events, was documented.

Statistical methods

Data analyses were conducted using Statistical Package for the Social Sciences version 26.0.

Continuous data were tested to conform to normal distribution and were described by mean $\pm$ standard deviation. Differences between groups were compared using the independent samples t-tests. Categorical data were presented as number of cases and percentages. Comparisons between groups were analyzed using χ^2 test or Fisher's exact test. Repeated measures analysis of variance was carried out to analyze repeated measurements, and pairwise comparisons between time points were performed using the Bonferroni correction. Risk factors for herpes zoster were determined using multivariable logistic regression analysis. Baseline lymphocyte counts prior to treatment were used to assess immune status. Statistical significance was set at $P<0.05$.

Results

Comparison of baseline characteristics between the two groups

The two groups did not differ significantly in baseline characteristics, including sex, age, body mass index, course of disease, comorbidities, history of herpes zoster, concomitant glucocorticoid use, and baseline DAS28-ESR score ($P>0.05$, **Table 1**).

Comparison of pre- and post-treatment DAS28-ESR scores between the two groups

The pre-treatment DAS28-ESR scores did not differ significantly between the groups ($P>0.05$). At 12 and 24 weeks, the tofacitinib group exhibited significantly lower DAS28-ESR scores than the conventional DMARDs group ($P<0.05$, **Figure 1**).

Efficacy of tofacitinib and risk of herpes zoster

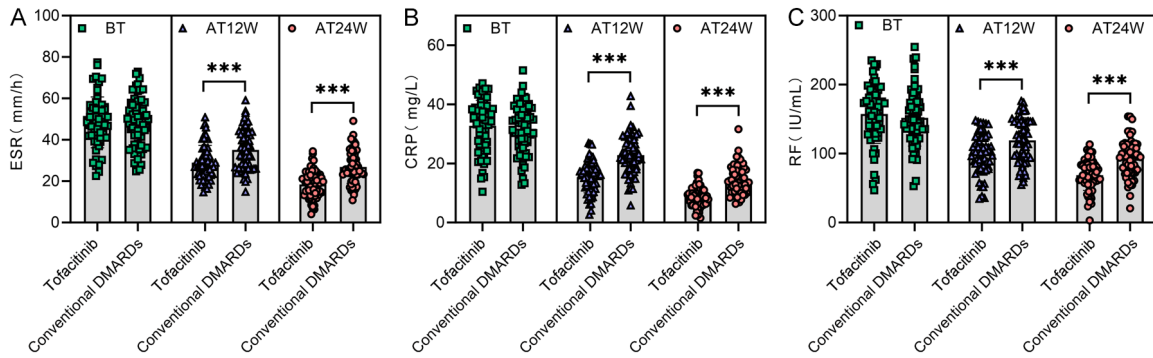


Figure 2. Comparison of inflammatory markers between the two groups. Before treatment, both groups had no significant difference in ESR (A), CRP (B), and RF (C) levels ($P>0.05$). At 12 and 24 weeks, the tofacitinib group exhibited significantly lower ESR, CRP, and RF levels compared with the conventional DMARDs group ($P<0.05$). Note: ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; RF: rheumatoid factor; DMARDs: disease-modifying antirheumatic drugs; BT: before treatment; AT12W: at 12 weeks after treatment; AT24W: at 24 weeks after treatment. *** $P<0.001$. Repeated measures analysis of variance was used for comparisons across multiple time points, and Bonferroni correction was applied for pairwise comparisons within groups.

Table 2. Comparison of ACR response rates between the two groups [n (%)]

ACR response	Tofacitinib group (n=62)	Conventional DMARDs group (n=58)	χ^2	P
ACR20 (12 weeks)	52 (83.87)	38 (65.52)	5.248	0.022
ACR50 (12 weeks)	38 (61.29)	24 (41.38)	4.756	0.029
ACR70 (12 weeks)	22 (35.48)	12 (20.69)	3.156	0.076
ACR20 (24 weeks)	56 (90.32)	42 (72.41)	6.382	0.012
ACR50 (24 weeks)	46 (74.19)	32 (55.17)	4.826	0.028
ACR70 (24 weeks)	32 (51.61)	18 (31.03)	5.186	0.023

ACR: American College of Rheumatology; DMARDs: disease-modifying antirheumatic drugs; ACR20/50/70: 20%/50%/70% improvement in American College of Rheumatology response criteria.

Comparison of inflammatory markers between the two groups

Before treatment, both groups exhibited no significant difference in ESR, CRP, and RF levels ($P>0.05$). At 12 and 24 weeks, the tofacitinib group exhibited significantly lower ESR, CRP, and RF compared with the conventional DMARDs group ($P<0.05$, **Figure 2**).

Comparison of ACR response rates between the two groups

At 12 weeks of treatment, the tofacitinib group exhibited significantly higher response rates of ACR20 and ACR50 compared with the conventional DMARDs group ($P<0.05$), while both groups did not differ significantly in the ACR70 response rate ($P>0.05$). At 24 weeks, higher response rates of ACR20, ACR50, and ACR70 were observed in the tofacitinib group ($P<0.05$, **Table 2**).

Comparison of joint function and quality of life between the two groups

Before treatment, there were no statistically significant differences between the two groups in HAQ-DI scores, duration of morning stiffness, and VAS pain score ($P>0.05$). At 12 and 24 weeks, the tofacitinib group had significantly lower values of the aforementioned indicators ($P<0.05$, **Figure 3**).

Comparison of herpes zoster occurrence between the two groups

The tofacitinib group exhibited a higher incidence of herpes zoster than the conventional DMARDs group (12.90% vs. 3.45%, $P<0.05$). Patients in the tofacitinib group developed herpes zoster at a median onset time of 14.50 weeks. The thoracic and dorsal regions were the most frequently affected sites (62.50%). Postherpetic neuralgia was observed in 3 patients (37.50%, **Table 3**).

Efficacy of tofacitinib and risk of herpes zoster

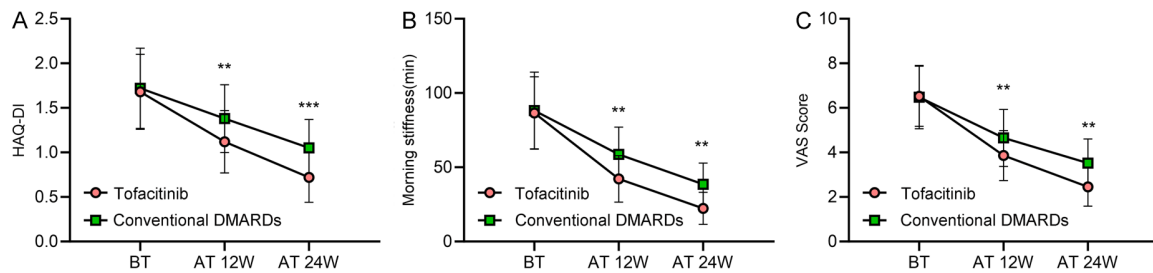


Figure 3. Comparison of joint function and quality of life scores between the two groups. Before treatment, there were no statistically significant differences between the two groups in HAQ-DI scores (A), duration of morning stiffness (B), and VAS pain score (C) ($P>0.05$). At 12 and 24 weeks, the aforementioned indicators were significantly lower in the tofacitinib group ($P<0.05$). Note: HAQ-DI: Health Assessment Questionnaire Disability Index; VAS: visual analog scale; BT: before treatment; AT12W: at 12 weeks after treatment; AT24W: at 24 weeks after treatment. ** $P<0.01$; *** $P<0.001$. Repeated measures analysis of variance was used for comparisons across multiple time points, and Bonferroni correction was applied for pairwise comparisons within groups.

Table 3. Comparison of herpes zoster occurrence between the two groups [n (%)]

Herpes zoster-associated indicators	Tofacitinib group (n=62)	Conventional DMARDs group (n=58)	χ^2/t	P
Herpes zoster occurrence	8 (12.90)	2 (3.45)	3.562 ^a	0.049
Median onset time (weeks)	14.50 (8.25-18.75)	16.00 (12.00-20.00)	-	0.524
Affected areas				
Thoracic and dorsal regions	5 (62.50)	1 (50.00)		
Lumbar and abdominal regions	2 (25.00)	1 (50.00)		
Facial region	1 (12.50)	0 (0.00)		
Postherpetic neuralgia	3 (37.50)	0 (0.00)		

DMARDs: disease-modifying antirheumatic drugs. ^aindicates Fisher's exact test.

Table 4. Evaluation of risk factors associated with herpes zoster

Risk factor	β	SE	Wald χ^2	OR (95% CI)	P
Tofacitinib use	1.452	0.685	4.492	4.27 (1.12-16.32)	0.034
Age ≥ 75 years	1.286	0.612	4.412	3.62 (1.09-12.05)	0.036
Diabetes	1.125	0.586	3.685	3.08 (0.98-9.72)	0.055
Concomitant glucocorticoid use	1.358	0.628	4.676	3.89 (1.14-13.32)	0.031
Baseline lymphocyte count $<1.0 \times 10^9/L$	1.568	0.702	4.986	4.80 (1.21-19.02)	0.026
History of herpes zoster	1.856	0.824	5.074	6.40 (1.27-32.18)	0.024

SE: standard error; OR: odds ratio; 95% CI: 95% confidence interval. The lymphocyte count at baseline (prior to treatment) was used for the analysis.

Analysis of risk factors associated with herpes zoster

Multivariable logistic regression analysis was performed with herpes zoster occurrence as the dependent variable and tofacitinib use, age, sex, diabetes, concomitant glucocorticoid use, baseline lymphocyte count, and history of herpes zoster as independent variables. The analysis showed that tofacitinib use (odds ratio [OR]=4.27), age ≥ 75 years (OR=3.62), concomitant glucocorticoid use (OR=3.89), lym-

phocyte count $<1.0 \times 10^9/L$ (OR=4.80), and history of herpes zoster (OR=6.40) were identified as independent risk factors for herpes zoster ($P<0.05$ for all, **Table 4**).

Comparison of other adverse events between the two groups

A higher incidence of elevated blood lipids was observed in the tofacitinib group ($P<0.05$). There was no significant difference between the two groups in other adverse events and overall incidence ($P>0.05$, **Table 5**).

Table 5. Comparison of other adverse events between the two groups [n (%)]

Adverse reactions	Tofacitinib group (n=62)	Conventional DMARDs group (n=58)	χ^2	P
Upper respiratory tract infection	6 (9.68)	4 (6.90)	0.312	0.576
Gastrointestinal discomfort	5 (8.06)	8 (13.79)	0.992	0.319
Abnormal liver function	4 (6.45)	5 (8.62)	0.207	0.649
Elevated blood lipid	8 (12.90)	2 (3.45)	3.562	0.049
Urinary tract infection	3 (4.84)	2 (3.45)	0.148	0.701
Headache	4 (6.45)	2 (3.45)	0.556	0.456
Cardiovascular events	1 (1.61)	1 (1.72)	0.002	0.962
Overall incidence	31 (50.00)	24 (41.38)	0.894	0.344

DMARDs: disease-modifying antirheumatic drugs.

Discussion

Clinical effects of tofacitinib in elderly patients with RA

In this study, the tofacitinib group exhibited significantly reduced DAS28-ESR scores at all treatment time points, greater improvements in ESR, CRP, and RF, and higher ACR response rates compared with the conventional DMARDs group. This suggests that tofacitinib has a significant advantage in controlling disease activity in elderly patients with RA. Evidence suggests that tofacitinib rapidly and effectively suppresses inflammatory responses by inhibiting the JAK1/JAK3-mediated cytokine signaling pathway, with a typical onset of action within 2-4 weeks, which is earlier than that of conventional DMARDs [13, 14]. In the present study, the tofacitinib group showed a significant difference in efficacy at 4 weeks of treatment, which is consistent with previous findings. In terms of joint function assessment, the tofacitinib group showed significantly better HAQ-DI scores, shorter duration of morning stiffness, and lower VAS score compared with the conventional DMARDs group. The findings demonstrate that tofacitinib more effectively improves joint function and quality of life in elderly patients with RA. These findings may be attributed to tofacitinib's ability to rapidly control inflammation and alleviate joint swelling and pain. Effective symptom relief may facilitate early functional exercise, thereby promoting recovery of joint function [15, 16].

Tofacitinib and the risk of herpes zoster

This study investigated the risk of herpes zoster associated with tofacitinib therapy. The find-

ings revealed a higher incidence of herpes zoster in the tofacitinib group. Studies have indicated that the occurrence of herpes zoster is closely associated with the decline in VZV-specific T-cell immunity. Tofacitinib may impact T-cell differentiation and function by inhibiting the JAK signaling pathway, thereby increasing the risk of VZV reactivation [17, 18]. To identify patients at higher risk and guide preventive strategies, this study further analyzed the risk factors for herpes zoster. The results showed that tofacitinib use, age ≥ 75 years, concomitant glucocorticoid use, reduced lymphocyte count, and history of herpes zoster were independent risk factors. These findings suggest that clinicians should carefully assess patients' risk of infection when prescribing tofacitinib. Enhanced monitoring or preventive measures should be considered for elderly patients, immunocompromised patients, those receiving concomitant glucocorticoids, and those with a history of herpes zoster. The recombinant herpes zoster vaccine is currently recommended for prophylactic use in individuals over 50 years of age. For high-risk patients scheduled to receive tofacitinib, vaccination may be considered prior to treatment initiation [19, 20]. The recombinant zoster vaccine was approved in China in 2023 and is now available as a preventive option against herpes zoster. Eligible high-risk patients with RA should consult their physicians prior to initiating tofacitinib therapy to assess the feasibility of vaccination.

Other safety issues of tofacitinib

In addition to herpes zoster, this study also analyzed other safety indicators of tofacitinib. The results showed a significantly higher incidence of elevated blood lipids in the tofacitinib group

compared with the conventional DMARDs group, which is in line with the known metabolic effects of tofacitinib. Clinical practice has shown that JAK inhibitors may increase lipid levels by affecting lipid metabolism pathways, particularly those involving low-density lipoprotein cholesterol and total cholesterol. Regular monitoring of lipid levels is recommended for patients receiving tofacitinib, with lipid-lowering therapy administered when necessary [21, 22]. Several studies have suggested that JAK inhibitors may increase the likelihood of cardiovascular events and malignancies, particularly among individuals aged >65 years [23, 24]. In our research, the incidence of cardiovascular events was low during the follow-up period, with no significant difference between the groups. However, due to the relatively short follow-up period and limited number of cases, extended follow-up observation is required. In view of this risk, clinicians should carefully assess the cardiovascular risk in patients when prescribing tofacitinib, and use the drug with caution in patients with cardiovascular risk factors.

Research implications and limitations

Our results indicate that tofacitinib demonstrates favorable clinical efficacy in treating elderly patients with RA, but attention should be paid to the elevated risk of herpes zoster. Tofacitinib may serve as an effective treatment option for elderly patients with RA responding poorly to conventional DMARDs, but treatment should be individualized based on a thorough assessment of the risk of infection. For patients at high risk of herpes zoster, prophylactic vaccination should be considered prior to treatment, monitoring should be enhanced during treatment, and any prodromal symptoms should be managed promptly.

Some limitations should be acknowledged in our study. First, it is a retrospective single-center study with limited sample size, and the generalizability of the findings needs to be validated. Second, patients were only followed up for 24 weeks. Long-term effects and safety require further investigation. Third, the study did not compare the efficacy and safety of various doses of tofacitinib. Fourth, the number of herpes zoster cases was limited (n=10). Six predictor variables were included in the multivariable logistic regression model, which may have introduced a risk of model overfitting, as reflected by wide confidence intervals. Therefore,

the estimated effects of the identified risk factors should be interpreted with caution, and the findings require validation in larger cohorts. Future research should consider conducting multicenter, large-scale, and prospective investigation with prolonged follow-up periods to further illustrate the benefit-risk ratio of tofacitinib in elderly patients with RA.

Conclusions

Tofacitinib demonstrates favorable clinical efficacy in elderly patients with RA, including reductions in disease activity and inflammatory markers, increases in ACR response rates, and improvements in joint function and quality of life. However, tofacitinib treatment may increase the risk of herpes zoster. Tofacitinib use, advanced age, concomitant glucocorticoid use, reduced lymphocyte count, and history of herpes zoster are independent risk factors for herpes zoster. It is important to thoroughly assess patients' risk of infection, closely monitor high-risk patients, and consider prophylactic vaccination when necessary to achieve a balance between efficacy and safety.

Disclosure of conflict of interest

None.

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References

- [1] Noto G, Salvetti F, Demaj A, Altieri P, Chericoni E, Ferrigno E, Alunno A and Puxeddu I. Pathogenesis of rheumatoid arthritis: one year in review 2025. *Clin Exp Rheumatol* 2025; 43: 1533-1540.
- [2] Peterson E, Gallagher MK and Wilbur J. Rheumatoid arthritis: diagnosis and management for the family physician. *Am Fam Physician* 2024; 110: 515-526.
- [3] Cush JJ. Rheumatoid arthritis: early diagnosis and treatment. *Med Clin North Am* 2021; 105: 355-365.
- [4] Mahajan A, Sharma G, Thakur A, Singh B, Mehta H, Mittal N, Dogra S and Katare OP. Tofacitinib in dermatology: a potential opportunity for topical applicability through novel drug-delivery systems. *Nanomedicine (Lond)* 2024; 19: 79-101.

- [5] Di Benedetto P, Ruscitti P, Berardicurti O, Panzera N, Grazia N, Di Vito Nolfi M, Di Francesco B, Navarini L, Maurizi A, Rucci N, Teti AM, Zazzeroni F, Guggino G, Ciccio F, Dolo V, Alesse E, Cipriani P and Giacomelli R. Blocking Jak/STAT signalling using tofacitinib inhibits angiogenesis in experimental arthritis. *Arthritis Res Ther* 2021; 23: 213.
- [6] Kubo S, Nakayamada S and Tanaka Y. JAK inhibitors for rheumatoid arthritis. *Expert Opin Investig Drugs* 2023; 32: 333-344.
- [7] Ireland PA, Verheyden M, Jansson N, Sebaratnam D and Sullivan J. Infection risk with JAK inhibitors in dermatoses: a meta-analysis. *Int J Dermatol* 2025; 64: 24-36.
- [8] Adas MA, Alvey E, Cook E, Dey M, Galloway JB and Bechman K. The infection risks of JAK inhibition. *Expert Rev Clin Immunol* 2022; 18: 253-261.
- [9] Jeong S, Choi S, Park SM, Kim J, Ghang B and Lee EY. Incident and recurrent herpes zoster for first-line bDMARD and tsDMARD users in seropositive rheumatoid arthritis patients: a nationwide cohort study. *Arthritis Res Ther* 2022; 24: 180.
- [10] Aletaha D, Neogi T, Silman AJ, Funovits J, Felson DT, Bingham CO 3rd, Birnbaum NS, Burmester GR, Bykerk VP, Cohen MD, Combe B, Costenbader KH, Dougados M, Emery P, Ferraccioli G, Hazes JM, Hobbs K, Huizinga TW, Kavanaugh A, Kay J, Kvien TK, Laing T, Mease P, Ménard HA, Moreland LW, Naden RL, Pincus T, Smolen JS, Stanislawski-Biernat E, Symmons D, Tak PP, Upchurch KS, Vencovsky J, Wolfe F and Hawker G. 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Ann Rheum Dis* 2010; 69: 1580-1588.
- [11] Bitirgen G, Kucuk A, Ergun MC, Satirtav G and Malik RA. Corneal nerve loss and increased Langerhans cells are associated with disease severity in patients with rheumatoid arthritis. *Eye (Lond)* 2023; 37: 2950-2955.
- [12] Cordeiro RA, Fischer FM and Shinjo SK. Work situation, work ability and expectation of returning to work in patients with systemic autoimmune myopathies. *Rheumatology (Oxford)* 2023; 62: 785-793.
- [13] Ma X, Yang F, Wu J, Xu B, Jiang M, Sun Y, Sun C, Yu Y, Xu D, Xiao L, Ren C, Chen C, Ye Z, Liang J, Lin J and Chen W. Efficacy and safety of tofacitinib in patients with polymyalgia rheumatica (EAST PMR): an open-label randomized controlled trial. *PLoS Med* 2023; 20: e1004249.
- [14] Ueki Y, Takimoto-Ito R, Saito MK, Tanizaki H and Kambe N. Tofacitinib, a suppressor of NOD2 expression, is a potential treatment for Blau syndrome. *Front Immunol* 2023; 14: 1211240.
- [15] Shi Y, Xie Y, Zhang G and Feng Y. Tofacitinib for the treatment of rheumatoid arthritis: a real-world study in China. *Intern Emerg Med* 2022; 17: 703-714.
- [16] Navarro-Compán V, Deodhar A, Bahiri R, Bushmakina AG, Cappelleri JC and Rammaoui J. Time to improvement of pain, morning stiffness, fatigue, and disease activity in patients with ankylosing spondylitis treated with tofacitinib: a post hoc analysis. *Arthritis Res Ther* 2024; 26: 105.
- [17] Hawerkamp HC, Domdey A, Radau L, Sewerin P, Oláh P, Homey B and Meller S. Tofacitinib downregulates antiviral immune defence in keratinocytes and reduces T cell activation. *Arthritis Res Ther* 2021; 23: 144.
- [18] Sieiro Santos C, Herrero JG, Ordas Martínez J, Álvarez Castro C, López Robles A, Colindres R, Sierra Ausin M, Martín ER, Sahagun AM and Ruiz de Morales JG. Differences in immunogenicity to zoster recombinant vaccine in IMiD patients undergoing treatment with non-selective or selective JAK-i. *Rheumatology (Oxford)* 2026; 65: keaf533.
- [19] Lewis CY, Mishra K, Sun Y, Sechrist SJ, Arnold BF and Acharya NR. Recombinant zoster vaccine coverage in the United States: an analysis of claims-based data. *Vaccine* 2023; 41: 3493-3496.
- [20] Sun Y, Kim E, Kong CL, Arnold BF, Porco TC and Acharya NR. Effectiveness of the recombinant zoster vaccine in adults aged 50 and older in the United States: a claims-based cohort study. *Clin Infect Dis* 2021; 73: 949-956.
- [21] Czókolyová M, Hamar A, Pusztai A, Tajti G, Végh E, Pethő Z, Bodnár N, Horváth Á, Soós B, Szamosi S, Szentpéteri A, Seres I, Harangi M, Paragh G, Kerekes G, Bodoki L, Domján A, Hodosi K, Seres T, Panyi G, Szekanecz Z and Szűcs G. Effects of one-year tofacitinib therapy on lipids and adipokines in association with vascular pathophysiology in rheumatoid arthritis. *Biomolecules* 2022; 12: 1483.
- [22] Koh JH, Lee BW and Kim WU. Changes in the cholesterol profile of patients with rheumatoid arthritis treated with biologics or Janus kinase inhibitors. *J Rheum Dis* 2023; 30: 234-242.
- [23] Duan H, Jiang C, Zhang B, Meng H, Zhou W, Yan W and Jiang T. Risk of malignancies and major adverse cardiovascular events related to JAK inhibitors in rheumatoid arthritis: a meta-analysis. *Clin Pharmacol Ther* 2026; 119: 598-607.
- [24] Yang V, Kragstrup TW, McMaster C, Reid P, Singh N, Haysen SR, Robinson PC and Liew DFL. Managing cardiovascular and cancer risk associated with JAK inhibitors. *Drug Saf* 2023; 46: 1049-1071.