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RESEARCH PAPER

Therapeutic Outcomes of Tofacitinib with Dose Escalation Strategies in Patients with NSAID-Refractory Axial Spondyloarthritis

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Abstract

Background: Axial spondyloarthritis (axSpA) is often resistant to first-line non-steroidal anti-inflammatory drugs (NSAIDs), necessitating the use of advanced therapies. Janus kinase (JAK) inhibitors have become highly effective oral alternatives to biological agents. In resource-limited settings like Bangladesh, the availability of lower-cost tofacitinib provides a crucial therapeutic option, reducing financial and accessibility barriers associated with biologics. Effective patient management in these contexts requires tailored dosing strategies to carefully balance efficacy, safety, and affordability.

Objective: To assess the therapeutic efficacy and safety of tofacitinib in patients with NSAID-refractory axSpA, with a particular focus on the outcomes of a dose-escalation strategy for patients showing an inadequate response to standard dosing.

Methods: This prospective clinical trial (NCT03738956) enrolled 52 patients with NSAID-refractory axSpA. All participants initially received tofacitinib 5 mg twice daily (Phase A, months 0–3). At the end of month 3, treatment response was evaluated: patients achieving a clinically important improvement in ASDAS-CRP ("ASDAS-CRP e" 1.1) were maintained on 5 mg twice daily for the remainder of the study (Phase B, months 3–6). For those who failed to achieve this threshold, the tofacitinib dose was escalated to 10 mg twice daily. The primary efficacy endpoint was the ASAS20 response rate. Secondary assessments, conducted at baseline, 1, 3, and 6 months, included ASAS40/70, BASDAI, BASFI, ASDAS, MASES, CRP, ESR, spinal pain, and fatigue. Adverse events (AEs) and serious adverse events (SAEs) were actively monitored. Statistical significance was determined using two-sided tests, with $p < 0.05$ considered significant.

Results: In this study, 52 patients (mean age 32.9 years, 78.8% male) were included. Tofacitinib demonstrated rapid and sustained efficacy; by month 3, overall ASAS20, ASAS40, and ASAS70 response rates were 73.1%, 65.4%, and 30.8%, respectively. Patients maintained on 5 mg twice daily (n=42) showed continued significant improvement at month 6 (ASAS20: 95.2%; ASAS40: 88.1%; ASAS70: 50.0%; all p<0.05). Ten patients (19.2%) with inadequate response at month 3 underwent dose escalation to 10 mg twice daily, achieving modest subsequent improvements by month 6 (ASAS20: 30%; ASAS40: 30%; ASAS70: 20%). All composite disease measures (including BASDAI, BASFI, and ASDAS-CRP) improved significantly from baseline (p<0.05). Adverse events occurred in 63.5% of patients, with serious adverse events, including herpes zoster and tuberculosis, reported in 13.5%.

Conclusions: Standard dosing of tofacitinib showed rapid response, high efficacy, and sustained disease control in NSAID-refractory axial spondyloarthritis. Those with a poor response to the standard dose may benefit from dose escalation, but caution is required as serious adverse events are relatively common. Clinicians should prioritise vigilant safety monitoring and carefully weigh the risks and benefits before increasing the dose in non-responders.

Keywords: Axial Spondyloarthritis (axSpA); tofacitinib; ASAS20; ASAS40; ASAS70; JAK inhibitor.

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Introduction

Spondyloarthritis (SpA) comprises a group of common chronic inflammatory diseases that share genetic, immunopathological, clinical, laboratory, and radiological features. Among the SpA group, axial SpA

(axSpA) is characterised by inflammation at sacroiliac joints and the spine, with or without peripheral and extra-articular manifestations.[1] In 2009, the Assessment of SpondyloArthritis International Society (ASAS) proposed and validated classification criteria for axial spondyloarthritis (axSpA).[2]

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and exercises are the first-line treatment for axial SpA.[3] If a patient does not respond to two sequential NSAIDs at optimum dosage for at least two to four weeks, he is said to have refractory axial SpA.[4]

The Assessment of SpondyloArthritis International Society (ASAS) recommends anti-TNF therapy as first-line treatment for refractory axSpA[4]. As there is no convincing evidence for the efficacy of disease-modifying anti-rheumatic drugs (DMARDs), including sulfasalazine and methotrexate, for the treatment of axial disease, [5]and only one-third of patients achieve a partial response with NSAIDs alone.[6] To achieve low disease activity and remission, in addition to therapeutic exercise and other lifestyle measures, the pharmacological treatment of axSpA has undergone significant changes since the introduction of biological drugs and Janus Kinase (JAK) inhibitors. Anti-Tumour Necrosis Factor (TNF) drugs were the first biologics approved for AS and, more recently, for the whole group of axSpA, showing efficacy and effectiveness in reducing spinal inflammation and pain, and improving function and quality of life.[7-9]

Janus Kinase Inhibitors (JAKs) are a family of non-receptor protein tyrosine kinases having four members: JAK1, JAK2, JAK3, and Tyrosine Kinase 2 (Tyk2). Distinct combinations of homodimers of JAK proteins selectively associate with different cytokine receptors. After the interaction between cytokine and its receptor, activation of JAK leads to the phosphorylation of the signal transducer and activator of transcription (STAT) family proteins. Dimers of STATs translocate to the nucleus, where they regulate the expression of cytokine-responsive genes.[10] Inhibition of each JAK member may affect the signalling of different cytokines. Thus, JAK proteins are potential targets for the treatment of several inflammatory diseases. Different cytokines signal using pairings of individual JAKs. IL-2, IL-4, IL-7, IL-9, IL-15, and IL-21 signal through the JAK1/JAK3 combination and play a pivotal role in modulating adaptive immune functions, including Th cell differentiation.[11] In particular, in the pathogenesis of SpA, innate lymphoid cells appear to play a crucial role and depend on IL-7 signalling.[12] IFNã and IL-12 are also reliant on JAK1/JAK2 and JAK2/TYK2 combinations and are critical for Th1 cell response, which modulates the production of TNFá by macrophages.[13] Significantly, given the role of the IL-23/IL-17 axis in SpA, JAKs influence signalling for several key cytokines involved in this pathway. JAK2/TYK2 combination is essential for the signalling of IL-23 produced by activated dendritic cells. In addition to the direct blockade of IL-23 signalling, an indirect consequence of JAK inhibition is the downstream blockade of IL-17 production. Furthermore, IL-22 signalling is mediated by the JAK1/TYK2 pair.[10] Different phase II and III studies demonstrated the efficacy of JAK inhibitors, including tofacitinib, upadacitinib, and nilotinib, for treating active AxSpA despite treatment with NSAIDs.

Tofacitinib inhibits JAK1, JAK3, and, to a lesser extent, JAK2. Data from the phase 3 study of tofacitinib for the treatment of axial spondyloarthritis were published. Another study also shows its efficacy and safety in treating axSpA. [14]

Data on the effectiveness and safety of tofacitinib across diverse real-world populations remain limited, and evidence from Bangladesh is scarce. To our knowledge, this is the first Bangladeshi study evaluating tofacitinib in NSAID-refractory axSpA. Additionally, practical issues such as dose escalation for nonresponders and local considerations of cost and access (eg, lower monthly cost of tofacitinib compared with biologic agents) motivate investigation of tofacitinib as a potential therapeutic option in this setting. Therefore, we conducted an observational study to assess the effectiveness, multidomain outcomes, dose-response effects, and safety of tofacitinib in NSAID-refractory axSpA patients, including the impact of dose escalation in initial nonresponders.

Materials and Methods

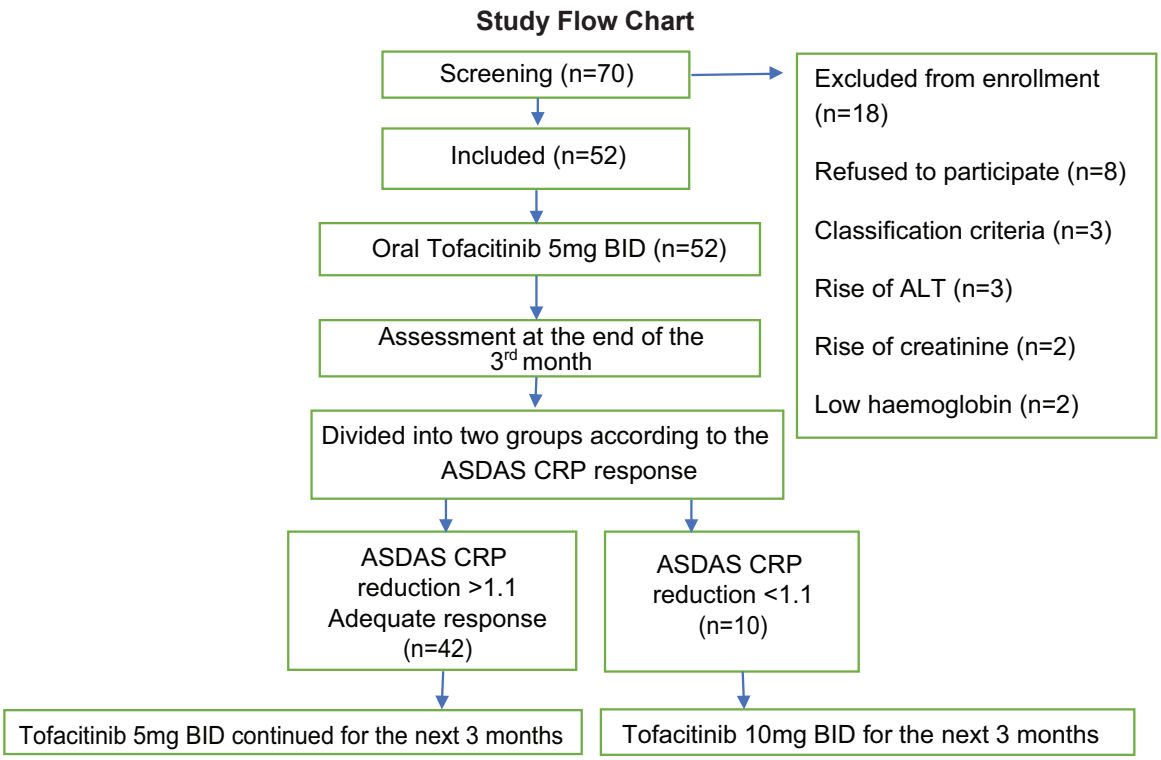
This prospective clinical trial was carried out at the Rheumatology Outpatient Clinic of Bangladesh

Medical University (former Bangabandhu Sheikh Mujib Medical University), Dhaka, Bangladesh, from January 2018 to October 2019. Adults aged 18 years or older were consecutively enrolled if they presented with chronic back pain lasting at least three months, had symptom onset before 45 years of age, and met the Assessment of SpondyloArthritis International Society (ASAS) classification criteria for axial spondyloarthritis. An inadequate response to conventional therapy was defined as either failing at least two adequate non- steroidal anti- inflammatory drug (NSAID) trials (each for a minimum of two weeks at an optimal dose) or showing only a partial response, evidenced by a Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) score ≥4 or an Ankylosing Spondylitis Disease Activity Score using C- reactive protein (ASDAS- CRP) > 2. 1.

The exclusion criteria included any history of systemic infections requiring hospital admission within the six months prior to enrolment, or active, recurrent, serious, and opportunistic infections. Pregnancy or breastfeeding, along with women of childbearing potential unwilling or unable to use highly effective contraception, were also exclusion factors. Other grounds for exclusion comprised of any history or current evidence of malignancy, New York Heart Association (NYHA) class III–IV heart failure, or significant laboratory abnormalities such as haemoglobin below 9 g/dL, and cytopenias (white blood cell count < 4, 000/mm³, neutrophil count < 1, 000/mm³, or platelet count <100, 100,000/mm³). Additionally, renal dysfunction (serum creatinine exceeding the laboratory's upper normal limit or an estimated glomerular filtration rate [eGFR] <50 mL/min), hepatic impairment (alanine aminotransferase [ALT] > 2 × the upper normal limit), and recent receipt of live vaccines (within three months prior to baseline) constituted further exclusion criteria. Of the 70 patients screened, 18 were excluded (8 declined, 3 did not meet ASAS criteria, and 7 had laboratory abnormalities), leaving 52 participants to be enrolled and analysed.

The study treatment began with tofacitinib 5 mg twice daily for all participants during Phase A (from baseline to month 3). Clinical and laboratory assessments, including complete blood count, erythrocyte sedimentation rate, serum creatinine, and ALT, were performed at baseline, 1 month, and 3 months to evaluate efficacy and adverse events. At the end of month 3, marking the start of Phase B, patients who achieved a clinically significant improvement—defined as a decrease of at least 1.1 units from baseline in ASDAS-CRP—continued on tofacitinib 5 mg twice daily. Those who did not reach this response had their tofacitinib dose escalated to 10 mg twice daily. All participants were followed up to month 6, with scheduled clinical and laboratory assessments to monitor disease activity and safety. The primary efficacy endpoint was the ASAS20 response, while secondary endpoints included ASAS40 and ASAS70 responses, along with changes in other composite measures of disease activity and function such as BASDAI, BASFI, ASDAS-ESR, MASES, CRP, ESR, spinal pain, and fatigue. Improvement in ASDAS-CRP and ASDAS-ESR was quantitatively measured by a significant absolute decrease from baseline (≥1.1 units indicating clinically important improvement). Safety evaluations at baseline included a comprehensive history, physical examination, urine analysis, CBC, serum creatinine, CRP, ALT, pelvic radiographs (modified Ferguson view), and rigorous screening for latent tuberculosis infection (LTBI) via Mantoux test or QuantiFERON-TB Gold Plus, as well as chest radiography to exclude active TB. Patients with LTBI received isoniazid and rifampicin for three months, with tofacitinib initiated one month after starting LTBI treatment. Routine laboratory monitoring (CBC, ESR, creatinine, ALT) was conducted at each follow-up visit, and adverse events were recorded throughout the study and managed according to departmental protocols.

Efficacy analyses used a modified intention-to-treat population that included all participants with at least one post-baseline assessment. Categorical outcomes (ASAS responses) were compared using chi-square or Fisher's exact tests as appropriate, while continuous changes in composite scores were analysed with paired t-tests. All statistical analyses were performed using SPSS version 26 (IBM Corp.), and two-sided p-values <0.05 were deemed statistically significant. The study was approved by the Ethical Review Committee of Bangabandhu Sheikh Mujib Medical University, and all participants provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki.



Results

In this study, we screened 70 patients, of whom 18 were excluded based on the exclusion criteria. The study subjects were assessed at the end of the 1st, 3rd and 6th month. Dose escalation occurred at the end of the 3rd month, depending on the ASDAS-CRP response.

Baseline demographic and disease characteristics are summarised in Table I. Mean age was 32.9 ± 9.2 years; 78.8% were male. Mean BMI was 21.9 ± 2.4 kg/m2.

Table I: Baseline demographic, disease activity, clinical and laboratory characteristics of the participant (n=52)

Variables	Mean ± SD / Median (IQR) / n(%)
Age (yrs)	32.94 ± 9.19
Gender	
Male	41 (78.8)
Female	11 (21.2)
Monthly family income (BDT)	12000 (9500 -17500)
BMI (kg/m2)	21.87±2.37
Tobacco use (smoking or chewable)	8 (15.4)
Disease duration (years)	3 (2-4)
BASDAI	5.00±1.07
BASFI	5.70 ±1.26
MASES	1.86±1.60
ASDAS ESR	4.39 ±0.82
ASDAS CRP	4.04±0.85
ESR	71.27 ± 35.82
CRP	45.21±44.85
Spinal pain	7.32±0.94
Fatigue	6.98±0.87

SD= Standard deviations, n= Number of patients, %= Percentages of patients;
ASDAS= Ankylosing spondylitis disease activity score, BASDAI= Bath ankylosing spondylitis disease activity index, BASFI= Bath ankylosing spondylitis functional index, ESR= Erythrocyte sedimentation rate, CRP= C-reactive protein; MASES=Maastrict ankylosing spondylitis enthesitis score

Median disease duration, monthly family income and baseline disease activity indices are presented in the table.

At month 1, a rapid clinical response was observed: ASAS20: 63.5%; ASAS40: 34.6%; and ASAS70: 5.8%. By month 3, overall response rates increased to ASAS20 73.1% (38/52), ASAS40 65.4% (34/52), and ASAS70 30.8% (16/52). Patients who failed to achieve ASAS20 by month 3 had significantly longer disease duration than responders (p = 0.003) (Figure 1).

Patients who remained on tofacitinib 5 mg twice daily through month 6 (n=42) showed particularly high and increasing response rates: ASAS20 88.1% at month 3 and 95.2% at month 6 (p < 0.001); ASAS40 81.0% at month 3 and 88.1% at month 6 (p < 0.001); ASAS70 38.1% at month 3 and 50.0% at month 6 (p< 0.001) (Table II).

Ten patients (19.2%) who did not meet the ASDAS CRP improvement threshold at 3rd month were escalated to 10 mg twice daily. At the end of 3rd month, only one of these ten patients (10%) achieved ASAS20; none achieved ASAS40 or ASAS70. After further escalation to a total daily dose of 20 mg in this subgroup, response rates rose modestly: ASAS20 - 30%, ASAS40-30% and ASAS70-20% (Figure 2).

All composite and patient reported measures improved significantly from baseline at 1st, 3rd and 6th month (Table III). Statistically significant reductions were observed in BASDAI, BASFI, MASES, ASDAS CRP, ASDAS ESR, CRP, ESR, spinal pain and fatigue at each follow up (all p < 0.05).

Adverse events occurred in 33 of 52 patients (63.5%). Seven patients (13.5%) experienced serious adverse events, including herpes zoster, disseminated tuberculosis, lower respiratory tract infection and urinary tract infection. Three patients (5.7%) were lost

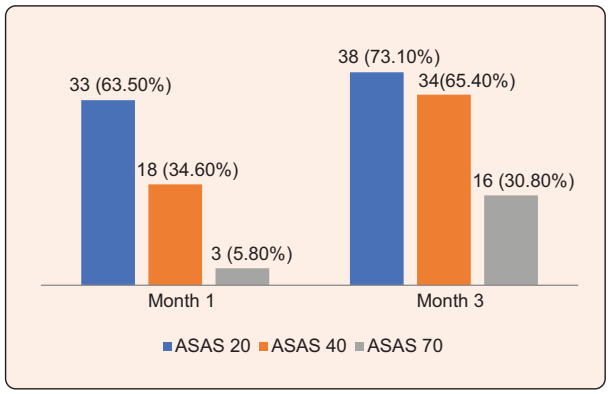


Figure 1: ASAS 20, 40, and 70 responses at 1st and 3rd month following treatment initiation in Phase A of the study (n=52).

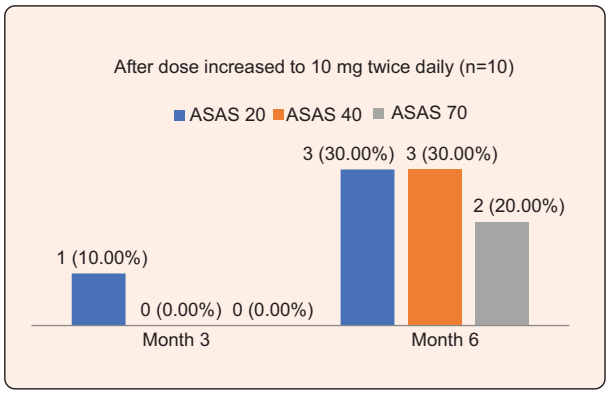


Figure 2: ASAS responses after increasing the tofacitinib dose to 10 mg twice daily in Phase B of the study.

Table II: ASAS 20, 40, 70 responses in the 5 mg and 10 mg twice daily groups at the end of the 3rd month (M3) and 6th month (M6) during Phase B.

ASAS responses	5 mg twice daily group (n=42)			10 mg twice daily group (n=10)		
	M3	M6	p-value	M3	M6	p-value
ASAS 20	37 (88.1%)	40 (95.2%)	0.00	1 (10.0%)	3 (30.0%)	0.11
ASAS 40	34 (81.0%)	37 (88.1%)	0.00	0 (0.0%)	3 (30.0%)	
ASAS 70	16 (38.1%)	21 (50.0%)	0.00	0 (0.0%)	2 (20.0%)	

ASAS: Assessment of Spondyloarthritis International Society, n = number, M3=After end of 3rd month, M6= After end of 6th month.

Table III: Disease activity measures at baseline, month 1, month 3, and month 6 (N=52)

	Baseline	Month 1		Month 3		Month 6	
	Mean± SD	Mean± SD	*p-value*[1]	Mean± SD	*p-value*[2]	Mean± SD	*p-value*[3]
BASDAI	5.00 ±1.07	3.50 ±1.42	<0.001	2.65 ±1.57	<0.001	1.93 ±1.24	<0.001
BASFI	5.70 ±1.26	3.98 ±1.70	<0.001	3.00 ±1.90	<0.001	2.26 ±1.75	<0.001
MASES	1.86 ±1.60	1.25 ±1.29	<0.001	0.90 ±1.03	<0.001	0.63 ±0.99	<0.001
ASDASESR	4.39 ±0.82	4.04 ±0.85	<0.001	2.46 ±1.10	<0.001	2.12 ±0.82	<0.001
ASDASCRP	4.04 ±0.85	2.49 ±1.05	<0.001	1.95 ±1.05	<0.001	1.80 ±0.82	<0.001
Spinal pain	7.32 ±0.94	6.17 ±1.50	<0.001	4.38 ±2.35	<0.001	3.17 ±2.20	<0.001
Fatigue	6.98 ±0.87	5.26 ±1.56	<0.001	3.67 ±2.05	<0.001	2.71 ±2.44	<0.001

n= Number of patients, SD= Standard deviation; ASDAS= Ankylosing spondylitis disease activity score, BASDAI= Bath ankylosing spondylitis disease activity index, BASFI= Bath ankylosing spondylitis functional index, ESR= Erythrocyte sedimentation rate, CRP= C- reactive protein, MASES= Maastricht's ankylosing spondylitis enthesitis index; p-values were determined by paired samples t-test at [1]month 1, [2]month 3, [3]month 6 compared to baseline.

Table IV: Different types of adverse effects (N=52).

Common		Others		Serious adverse effects	
Nasopharyngitis	12 (23.07%)	Anorexia	1 (1.9%)	Herpes zoster	2 (3.8%)
Diarrhoea	10 (19.23%)	Hypertension	1 (1.9%)	Disseminated TB	1 (1.9%)
Headache	10 (19.25%)	Abdominal	1 (1.9%)	Intractable itching	1 (1.9%)
Fever	9 (17.3%)	pain		Raised creatinine	1 (1.9%)
Itching	2 (3.8%)	Insomnia	1 (1.9%)	LRTI	1 (1.9%)
Nausea	2 (3.8%)			Recurrent UTI	1 (1.9%)

%= Percentages of patients; LRTI= Lower respiratory tract infection; UTI= Urinary tract infection

to follow up; one patient withdrew in month 4 due to intractable pruritus (Table IV).

Discussion

In this study, NSAID-refractory axial spondyloarthritis (axSpA) patients treated with tofacitinib, we observed rapid and progressively deepening clinical responses across standard ASAS endpoints and multiple disease activity measures, confirming the strong anti-inflammatory effects of JAK inhibition. By month 1, ASAS20, ASAS40, and ASAS70 rates were already notable at 63.5%, 34.6%, and 5.8%, respectively; these improvements continued to increase by month 3, reaching 73.1%, 65.4%, and 30.8%, respectively. Patients continuing on tofacitinib 5 mg twice daily until month 6 showed particularly high, sustained response rates: ASAS20 88.1% at month 3 and 95.2% at month 6 ($p < 0.001$); ASAS40 81.0% at month 3 and 88.1% at month 6 ($p < 0.001$); ASAS70 38.1% at month 3 and 50.0% at month 6 ($p = 0.012$). These quick and maintained improvements align well with findings from both randomised and open-label studies of JAK inhibitors and other advanced therapies in axSpA. The phase II randomised trial of tofacitinib in ankylosing spondylitis, for instance, showed significant reductions in ASDAS and BASDAI as early as week 12, confirming the rapid anti-inflammatory effects of JAK inhibition.[15] Likewise, the SELECT-AXIS 1 study of upadacitinib reported substantial improvements in ASAS responses, ASDAS, BASDAI, spinal pain, and CRP by week 14, providing a comparative view of JAK inhibitor efficacy in this population.[16] When compared with TNF and IL-17 inhibitor trials, such as adalimumab in ABILITY-1, certolizumab in C-axSpAnd, and secukinumab in the MEASURE trials, which generally report ASAS20/40 responses in the 50–70% range at 12–24 weeks and often lower ASAS70 rates early on the high ASAS40/70 rates observed in our 5 mg BID persistence subgroup appear notably impressive.[17-19] Variability in absolute response rates between our real-world cohort and established trial populations can be attributed to differences in study design (open-label versus randomised), baseline disease characteristics,

prior biologic exposure, and the natural selection of patients who tolerated and continued on therapy in a real-world setting.

Regarding dose escalation, our findings help improve the understanding of managing initial non-responders. Among 10 patients who did not meet the ASDAS-CRP improvement threshold at month 3 and subsequently underwent dose escalation, the immediate benefit was limited: only 1 (10%) achieved ASAS20 by month 3, and no ASAS40/70 responses were observed. Even after increasing the dose to a total of 20 mg daily (equivalent to 10 mg BID for a sustained period), only modest improvements were seen in this group, with 30%, 30%, and 20% achieving ASAS20, ASAS40, and ASAS70, respectively. These findings align with known dose–response patterns for tofacitinib and other JAK inhibitors, where higher doses may produce greater mean effects in some analyses but do not reliably turn non-responders into strong responders.[15,16] Such findings demonstrate that a subset of patients remains resistant despite higher therapeutic doses, suggesting that switching to a different mechanism of action, such as TNF or IL-17 inhibitors, could be more effective for many non-responders.[17-19] Healthcare professionals considering dose escalation should therefore set realistic expectations of response levels and carefully weigh potential safety concerns related to higher doses against possible incremental benefits.

Beyond objective response criteria, tofacitinib treatment also led to significant multidomain improvements. We observed concordant and statistically significant enhancements across patient reported outcomes (BASDAI, BASFI, spinal pain, fatigue), clinician assessed enthesitis (MASES), and objective inflammatory markers (CRP, ESR) at all assessment points (1, 3, and 6 months; all $p < 0.05$). This broad improvement supports a genuine anti inflammatory effect of tofacitinib, rather than mere symptomatic relief, and highlights its capacity to impact various facets of axSpA. Similar multidomain benefits, encompassing symptomatic, functional, and laboratory improvements, have been consistently documented in tofacitinib phase II data and in trials of other JAK inhibitors and biologic agents, where they typically occur in parallel with effective targeted therapy in axSpA .[15-20] The functional gains (BASFI) observed by months 3–6 are particularly crucial, translating into tangible improvements in patient quality of life and work capacity.

The safety profile in our cohort warrants careful examination. Adverse events were frequent, occurring in 33 of 52 patients (63.5%), including mild issues such as nasopharyngitis (23.07%), diarrhoea (19.23%), and headache (19.25%). More importantly, 7 of 52 patients (13.5%) experienced serious adverse events, including herpes zoster, disseminated tuberculosis, lower respiratory tract infection, and urinary tract infection. Three patients (5.7%) were lost to follow-up. This AE profile aligns with known safety signals for tofacitinib and the wider JAK inhibitor class. The phase II tofacitinib trial in ankylosing spondylitis reported similar common AEs and specifically noted opportunistic infections, including herpes zoster.[15] Broader JAK inhibitor safety data from rheumatoid arthritis trials and post-marketing surveillance have consistently emphasised increased risks of herpes zoster and serious infections, especially in older patients or those with comorbidities, leading regulatory bodies to issue guidance on vigilance and risk mitigation. The occurrence of disseminated tuberculosis in our series highlights the importance of thorough latent TB screening and a heightened awareness of regional TB prevalence before starting JAK inhibitor therapy. Similar infection risks are recognised with biologic agents, requiring robust pre-treatment screening and ongoing monitoring.[17,18] Given the known dose-related safety concerns reported across the JAK inhibitor class, decisions on dose escalation must carefully weigh the modest potential efficacy gains against the increased risk of adverse events.

This analysis is limited by its single-cohort, open-label design and relatively small sample size, which inherently limit causal inference and increase susceptibility to selection bias, particularly among patients who persisted on the 5 mg BID regimen. The absence of a randomised control arm further constrains direct comparisons and the precision of adverse event incidence estimates. Heterogeneity in baseline characteristics, such as radiographic status, disease duration, and prior biologic exposure, may also influence observed responses and complicate direct comparisons with more rigorously controlled randomised trials.

Conclusion

In summary, tofacitinib delivered rapid, broad, and increasingly profound clinical benefits in this cohort of NSAID-refractory axSpA patients, showing high ASAS20/40/70 rates among those maintained on the 5 mg BID regimen and notable improvements across various disease activity and functional measures.

While dose escalation provided only limited additional benefit for many initial non-responders, the safety findings aligned with known JAK inhibitor risks, including opportunistic and serious infections, highlighting the need for comprehensive screening, suitable vaccination, and close monitoring during treatment. These results support the role of tofacitinib as a practical therapeutic option for axSpA, especially in settings where biologics may be less accessible. However, they also emphasise the importance of larger, randomised, and longer-term studies to verify durability, identify predictors of response, and better assess the overall risk–benefit profile.

Author's contribution

All the authors were involved in the conception and design of the work and helped draft and revise the article. They have given the final approval and taken accountability.

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Disclosures

We used AI for drafting, editing, literature searching, formatting, and language polishing.

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