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Original Research Article

A Comparative Analysis of Patients with Progressive Vitiligo Treated with Tofacitinib versus Oral Mini-Pulse Steroid Therapy

Priyanka Chowdhary[1], Rajinderjit Kaur Ghuman[2], Boddeda Moulidharan[3]

[1]Junior Resident, Department of Dermatology, Krishna Mohan Medical College, Mathura, Uttar Pradesh, India.

[2]Senior Resident, Department of Dermatology, Krishna Mohan Medical College, Mathura, Uttar Pradesh, India.

[3]Junior Resident, Department of Dermatology, Krishna Mohan Medical College, Mathura, Uttar Pradesh, India.

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Corresponding Author: Dr. Rajinderjit Kaur Ghuman

Conflict of interest: Nil

Abstract:

Background: Vitiligo is a chronic autoimmune disease due to loss of melanocytes. Patients experience anxiety, depression, and therefore higher morbidity. The goal of treatment for progressive vitiligo is to stop the disease's progression and help the afflicted lesions regain their color. The two mainstays of treatment are topical medications and NBUVB phototherapy. However, these treatments may not always be sufficient to halt the progression. Oral mini-pulse (OMP) steroid therapy is widely used to control disease activity; however, concerns regarding corticosteroid-related adverse effects have encouraged evaluation of more targeted treatments such as Tofacitinib.

Aim: To compare the effectiveness and safety of oral Tofacitinib 5mg twice daily versus oral mini-pulse (OMP) steroid therapy Betamethasone 0.1mg/kg/day in patients with progressive vitiligo.

Methods: This prospective, randomized, parallel-group, open-label blinded controlled study was conducted in the Department of Dermatology, KM Medical College, Mathura. Ninety-five patients with progressive vitiligo were randomly allocated to receive either oral Tofacitinib 5mg twice daily (group A) or oral mini-pulse steroid therapy Betamethasone 0.1 mg/kg/day on two consecutive days for 24 weeks (group B). Treatment efficacy was evaluated using the Vitiligo Area Scoring Index (VASI), disease stabilization, repigmentation, overall clinical response, and treatment-related adverse events. Participants were assessed at baseline and at weeks 4, 8, 12, 16, 20, and 24. Data were analyzed using SPSS version 26.0.

Results: Both treatment groups showed statistically significant clinical improvement. Oral Tofacitinib produced a greater reduction in VASI score, higher rates of disease stabilization and visible repigmentation, a better overall clinical response, and fewer treatment-related adverse events than oral mini-pulse steroid therapy.

Pc added: Both groups improved significantly. Tofacitinib produced greater mean VASI reduction (3.24 ± 1.38 vs. 1.83 ± 1.19; $p < 0.001$), more visible repigmentation (70.8% vs. 46.8%; $p = 0.017$), and fewer adverse effects (22.9% vs. 42.6%, $p = 0.041$). Disease stabilization did not differ significantly ($p = 0.114$).

Conclusion: In this randomized study, tofacitinib was associated with greater improvement in VASI score and repigmentation outcomes compared with OMP therapy with fewer adverse events. Larger multicenter trials with extended follow-up are required to confirm these findings.

Keywords: Progressive vitiligo; Tofacitinib; oral mini-pulse steroid; JAK inhibitor; Vitiligo Area Scoring Index; disease stabilization; repigmentation; randomized controlled trial.

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Introduction

Vitiligo is a chronic acquired autoimmune depigmenting disorder characterized by selective destruction off unctioning melanocytes resulting in well defined depigmented macules and patches of skin and occasionally, mucosae. Although not life threatening, its visibility causes considerable psychological, emotional and social distress, particularly in individuals with skin of color. Vitiligo affects approximately 0.5% - 2% of the world's population, with nearly half of cases beginning before 20 years of age. Genetic transmission from a single parent is 5-8% whereas from both parents is 30-40 % and from one sibling is around 7 % [1,2] Vitiligo is one of the most commonly seen pigmentary disorders in dermatology clinics in India, and its prevalence ranges from 0.25% to 4% depending on the region

and ethnic groups. Progressive vitiligo, defined as new white lesions or spreading of existing ones over a duration of 6 months, poses a major challenge for therapy as repigmentation therapies are ineffective while autoimmune melanocyte destruction continues. [1-4].

Vitiligo is a multifactorial disease with pathogenesis including genetic susceptibility, oxidative stress, environmental triggers and autoimmune mechanisms. Of these, the most well-supported is autoimmune mediated melanocyte loss. Interferon gamma (INF-) is one of the cytokines that activates the Janus kinase-signal transducer and activator of transcription (JAK-STAT) pathway, which is critical for the recruitment and retention of autoreactive CD8+ T cells in lesional skin. These activated T cells secrete inflammatory cytokines and chemokines, especially CXCL9 and CXCL10, that contribute to melanocyte apoptosis and disease progression [5,6]. Targeted immunomodulatory agents that interrupt this cascade have trsansformed the therapeutic outlook.

Treatment of progressive vitiligo is mainly directed to stopping the progression of the disease followed by subsequent repigmentation. Conventional treatment is multimodal and includes topical application of corticosteroids, calcineurin inhibitors, phototherapy (NB-UVB, excimer, PUVA, psoralen photochemotherapy) and systemic agents (corticosteroids and steroid sparing agents) for rapidly progressive disease. Oral mini pulse (OMP) corticosteroid therapy, in which oral betamethasone/dexamethasone is taken on two consecutive days each week, is commonly used to reduce disease activity and has become a widely accepted approach for systemic therapy to avoid the side effects of continuous corticosteroid use. There are many clinical trials that have proven the efficacy of OMP therapy in stabilizing the disease in a large number of patients. However prolonged corticosteroids use causes acne, gastritis, hypertension, weight gain, menstrual irregularities, glucose intolerance and osteoporosis, limiting its acceptability and necessitating safer alternatives [15,16].

Phototherapy is still a key part of the treatment of vitiligo, since UV radiation induces melanocyte proliferation, melanogenesis and melanocyte migration. [8,10]

Janus Kinase (JAK) inhibitors have introduced a targeted therapeutic option for autoimmune skin diseases such as vitiligo. Tofacitinib, an oral JAK1/JAK3 block signaling by IFN-γ and has been shown to limit activation of autoreactive T lymphocytes that destroy melanocytes. Experimental data indicate that Tofacitinib is able to inhibit the progression of immune-mediated melanocyte damage, and with UV exposure, melanocytes can activate and migrate, suggesting a synergy between immunomodulation and photo stimulation [8].

The evidence about the use of systemic JAK inhibitors in the treatment of vitiligo has been further supported by recent RCTs. Comparative studies of oral Tofacitinib vs. oral mini pulse corticosteroid therapy have reported similar rates of disease stabilization, and some studies have shown a higher reduction in Vitiligo Area Scoring Index (VASI) scores and maintenance of repigmentation with Tofacitinib treatment. Systematic reviews have also shown that JAK inhibitors are overall well tolerated, with adverse effects chiefly upper respiratory infections, headache and transient laboratory abnormalities are mild and infrequent. However, existing studies are limited by small samples, short follow-up, heterogeneous treatment protocols, and limited representation of Indian patients in whom treatment practices and sun exposures differ from those in the West. [8,11].

Although recent randomized studies have directly compared oral tofacitinib with oral corticosteroid mini-pulse therapy in vitiligo, Additional comparative data from different clinical settings may help clarify the relative effects on disease stabilization, repigmentation, and short-term safety. The knowledge gap is especially important in India where systemic corticosteroids are often chosen for convenience, availability, and cost. is often started out of convenience, availability and cost. [11]

Hence, we conducted the present randomized controlled trial in to compare the efficacy and safety of oral Tofacitinib with oral mini pulse steroid therapy in progressive vitiligo. In this study, disease stabilization, improvement in Vitiligo Area Scoring Index (VASI), repigmentation outcomes, and adverse events related to the treatment were assessed so as to provide clinically relevant evidence to guide Dermatologists in choosing a treatment strategy that is effective and safer for patients with progressive vitiligo.

Methods

Study Design: This is a randomized, parallel-group, open label, blinded controlled study comparing oral Tofacitinib with oral mini pulse (OMP) steroid therapy. The study was designed to assessing clinical improvement, disease stabilization and adverse.

Study Area: The study was carried out in the Department of Dermatology, KM Medical College, Mathura, Uttar Pradesh, India in patients visiting the dermatology out-patient department (OPD) and who met the inclusion criteria.

Study Duration: The study was carried out from December 2025 to August 2026. Each participant received the assigned treatment for 24 weeks (6 months), with scheduled assessments from baseline through week 24 (week 4,8,12,16,20,24).

Sample Size: A total of 95 eligible patients with clinically diagnosed progressive vitiligo were

recruited consecutively from the dermatology outpatient department during the study period. No formal a priori sample size calculation was performed using a previously published study or an anticipated treatment effect. The final sample size was determined by the number of eligible patients recruited during the study period. The participants were allocated to two treatment groups.

- **Group A (n = 48):** Oral Tofacitinib 5 mg twice daily
- **Group B (n = 47):** Oral Mini-Pulse Steroid Therapy- Betamethasone 0.1 mg/kg/day on two consecutive days

Sample Population: The study population comprised patients aged 12 years and above with progressive vitiligo who presented to the Department of Dermatology, KM Medical College, Mathura. Progressive vitiligo was defined by the appearance of new depigmented lesions, enlargement of existing lesions during the preceding 6 months, and/or the presence of the Koebner phenomenon. Patients meeting the eligibility criteria and providing written informed consent were enrolled. For participants younger than 18 years, written consent from a parent or legal guardian was obtained.

Data Collection: Data were collected prospectively using a structured case record form. Demographic and clinical variables included age, sex, disease duration, family history of vitiligo, associated autoimmune disease, clinical type of vitiligo, body surface area involved, and evidence of lesion progression. Baseline work-up included complete blood count, liver function tests, renal function tests, fasting blood glucose, lipid profile, screening for active infection (HIV, Hepatitis B, Hhepatitis C, Tuberculosis), and pregnancy testing where applicable. The same safety laboratory parameters were repeated during follow-up when clinically indicated and at the end of treatment.

The Vitiligo Area Scoring Index (VASI)[7] was used to assess clinical severity at baseline and during follow-up. Disease activity was assessed by recording new lesions or enlargement of pre-existing lesions. Group A received oral Tofacitinib 5 mg twice daily. Group B received oral Betamethasone 0.1 mg/kg/day on two consecutive days each week as oral mini-pulse therapy. Treatment adherence, adverse drug reactions, clinical complications, and laboratory abnormalities were documented throughout the study. After verification for completeness and accuracy, all observations were entered into the master database.

Inclusion Criteria

Patients with any of the following conditions were included:

- Patients aged 12 years and above.
- Clinically diagnosed cases of progressive vitiligo.
- Evidence of disease progression during the preceding 6 months and/or presence of the Koebner phenomenon.
- Patients willing to participate and provide written informed consent.
- Willingness to attend scheduled follow-up visits.

Exclusion Criteria

Patients with any of the following conditions were excluded:

- Treatment-resistant vitiligo.
- Leukoderma due to other causes that mimic vitiligo.
- Pregnant or lactating women.
- Patients with clinically significant immunosuppressive conditions or comorbidities that could increase treatment-related risk.
- Patients with severe liver, kidney or heart diseases.
- Patients with an active infection (HIV, Hepatitis B and active Tuberculosis), malignancy, or immunodeficiency disorder.
- Use of systemic immunosuppressive therapy during the preceding 4 weeks.
- Known hypersensitivity to Tofacitinib or to corticosteroids.
- Refusal or noncompliance with follow-up by the patient.

Study Procedure: All the patients presenting to dermatology OPD with clinically diagnosed progressive vitiligo were screened. After written informed consent, eligible participants were enrolled and then randomly assigned to one of the two treatment groups, using a computer-generated randomized sequence.

Group A participants received oral Tofacitinib 5 mg twice daily for 24 weeks and Group B participants received oral Betamethasone 0.1 mg/kg/day on two consecutive days each week for 24 weeks. No Concomitant Phototherapy was administered during the study period. Baseline evaluation included a complete dermatological examination, VASI assessment, standardized clinical photography where feasible, and relevant laboratory investigations before initiation of therapy.

Patients were evaluated on day 0 and at weeks 4, 8, 12, 16, 20, and 24. At each visit, treatment adherence, clinical response , disease activity, repigmentation, adverse events, and relevant laboratory findings were recorded. VASI scores were reassessed at follow-up visits and compared with baseline values. Standardized clinical photographs were obtained.

Statistical Analysis: The data collected was entered into Microsoft Excel and analyzed by Statistical

Package for the Social Sciences (SPSS) version 26.0. Data of continuous variables were presented as mean ± standard deviation (SD) or median with interquartile range (IQR) according to distribution, and data of categorical variables was presented as number and percentage. The Shapiro–Wilk test was used to test the normality for continuous variables. The independent Student's t-test was used for normally distributed continuous variables between groups, and the Mann–Whitney U test was used for non-normally distributed continuous variables between groups. Categorical variables were compared using Chi-square test or Fisher's exact test as appropriate. Paired t-test or Wilcoxon signed-rank test was used to assess intra-group changes in VASI scores from baseline to end of treatment as appropriate for the distribution of the data. All analyses had a P-value of <0.05 as threshold of statistical significance".

Result

The baseline demographic characteristics were comparable between the two treatment groups (table 11) showing mean age to be 31.8 ± 11.2 years in Group A and 33.1 ± 10.8 years in Group B, with no statistically significant difference (P = 0.566). The gender distribution was also similar, with males comprising 56.3% and 53.2% and females comprising 43.7% and 46.8% of Groups A and B, respectively (P = 0.765) . Likewise, no significant differences were observed in mean disease duration (9.6 ± 5.4 vs. 10.2 ± 5.8 months; P = 0.603), family history of vitiligo (6.2% vs. 2.1%; P = 0.617), or associated autoimmune disease (10.4% vs. 12.8%; P = 0.715). Overall, all P-values were greater than 0.05, confirming that the two groups were demographically comparable at baseline.

Table 1: Baseline Demographic Characteristics

Variables	Group A (Tofacitinib) (n=48)	Group B (OMP Steroid) (n=47)	P-value
Age (years), Mean ± SD	31.8 ± 11.2	33.1 ± 10.8	0.566
Male	27 (56.3%)	25 (53.2%)	0.765
Female	21 (43.7%)	22 (46.8%)	
Disease duration (months), Mean ± SD	9.6 ± 5.4	10.2 ± 5.8	0.603
Family history of vitiligo	3 (6.2%)	1(2.1%)	0.617
Associated autoimmune disease	5 (10.4%)	6 (12.8%)	0.715

The groups were also clinically similar at baseline (Table 2). The mean baseline VASI score was 8.42 ± 2.14 in Group A and 8.57 ± 2.31 in Group B (P = 0.741), while the mean body surface area involved was 12.8 ± 5.2% and 13.4 ± 5.8%, respectively (P = 0.603). Generalized vitiligo was the most common clinical subtype in both groups, followed by focal and acrofacial vitiligo, and the subtype distribution did not differ (P = 0.937). Disease progression during the previous 6 months was recorded in all participants in both groups (100%); hence, no p-value could be computed. No baseline clinical difference was significant (P-value > 0.05).

Table 2: Baseline Clinical Characteristics

Variables	Group A (n=48)	Group B (n=47)	P-value
Baseline VASI score (Mean ± SD)	8.42 ± 2.14	8.57 ± 2.31	0.741
Body surface area involved (%)	12.8 ± 5.2	13.4 ± 5.8	0.603
Generalized vitiligo	31 (64.6%)	30 (63.8%)	0.937
Focal vitiligo	10 (20.8%)	11 (23.4%)	
Acrofacial vitiligo	7 (14.6%)	6 (12.8%)	
Disease progression within previous 6 months	48 (100%)	47 (100%)	NA

Table 3 presents baseline and post-treatment VASI scores. Both groups improved significantly after 24 weeks (P < 0.001). The mean reduction in VASI score demonstrated greater in the Tofacitinib group (3.24 ± 1.38) than in the oral mini-pulse steroid group (1.83 ± 1.19; P < 0.001).

Table 3: Comparison of VASI Scores

VASI Score	Group A (n=48)	Group B (n=47)	P-value
Baseline	8.42 ± 2.14	8.57 ± 2.31	0.741
After 24 weeks	5.18 ± 1.92	6.74 ± 2.28	<0.001
Mean reduction	3.24 ± 1.38	1.83 ± 1.19	<0.001

Table 4 shows that complete disease stabilization was higher in Group A receiving oral Tofacitinib than in Group B receiving oral mini-pulse steroid therapy (77.1% vs. 57.4%), whereas partial

stabilization (16.7% vs. 27.7%) and continued disease progression (6.2% vs. 14.9%) were lower in Group A. However, the overall distribution of disease-stabilization outcomes did not differ significantly between the groups (P = 0.114). In contrast, visible repigmentation was significantly more frequent in Group A than in Group B (70.8% vs. 46.8%; P = 0.017), indicating a significantly better repigmentation response with Tofacitinib.

Table 4: Disease Stabilization			
Outcome	**Group A (n=48)**	**Group B (n=47)**	**P-value**
Complete stabilization	37 (77.1%)	27 (57.4%)	0.114
Partial stabilization	8 (16.7%)	13 (27.7%)	
Continued progression	3 (6.2%)	7 (14.9%)	
Visible repigmentation	34 (70.8%)	22 (46.8%)	**0.017**

The adverse events that occurred during treatment are summarized in Table 5. Weight gain was significantly more frequent with the OMP therapy (P = 0.013), and other adverse events were rare and similar between groups. Adverse events were less frequent with Tofacitinib.

Table 5: Treatment-related Adverse Events			
Adverse Event	**Group A (n=48)**	**Group B (n=47)**	**P-value**
Acne	2 (4.2%)	4 (8.5%)	0.431
Weight gain	1 (2.1%)	8 (17.0%)	0.013 0.015
Gastritis	3 (6.3%)	5 (10.6%)	0.465 0.486
Upper respiratory infection	4 (8.3%)	2 (4.3%)	0.427 0.677
Elevated liver enzymes	1 (2.1%)	1 (2.1%)	1
Any adverse event	**11 (22.9%)**	**20 (42.6%)**	**0.041**
No adverse event	**37 (77.1%)**	**27 (57.4%)**	

Overall clinical response is shown in table 6. 37.5% of patients in the oral Tofacitinib group and 19.1% in the oral mini pulse steroid group achieved an excellent clinical response (more than 75% improvement). There was significantly higher overall treatment response in the oral Tofacitinib group (P = 0.041), representing efficacy as compared to oral mini pulse steroid therapy.

Table 6: Clinical Response		
Treatment response	**Group A (n=48)**	**Group B (n=47)**
Excellent (≥75%)	18 (37.5%)	9 (19.1%)
Good (50–75%)	17 (35.4%)	14 (29.8%)
Moderate (25–49%)	9 (18.8%)	15 (31.9%)
Poor (<25%)	4 (8.3%)	9 (19.1%)
Chi-square P-value	**0.082**	

Discussion

This is a current randomized controlled trial (RCT) that evaluates the efficacy and safety of oral Tofacitinib versus oral mini pulse (OMP) steroid therapy in patients with progressive vitiligo. No differences were observed at baseline between the two treatment groups regarding demographic and disease-related characteristics that could have led to selection bias. The mean age was 31.8 ± 11.2 years for the Tofacitinib group and 33.1 ± 10.8 years for the OMP steroid group (P = 0.552), and the male to female ratio was 56.3:43.7 and 53.2:46.8, respectively. Disease duration, familial history of vitiligo and other autoimmune diseases were also not different (P > 0.05). Baseline comparability was similar; Dev et al. reported no differences in age, disease duration or baseline disease severity between the patients receiving oral Tofacitinib and oral dexamethasone mini-pulse therapy, thus providing a reliable comparison of the treatments' outcomes [11]". Similarly, baseline data is crucial in determining the efficacy of a therapy as opposed to the effects of demographics in the case of efficacy studies of systemic therapies in the treatment of progressive vitiligo [9-10].

Additionally, the clinical characteristics of the study population further showed that both groups had similar disease severity prior to treatment. The mean baseline VASI score in the Tofacitinib group was 8.42 ± 2.14 and 8.57 ± 2.31 in the OMP steroid group, with the mean area of body surface affected being 12.8% and 13.4%, respectively (P > 0.05). The largest clinical subtype was generalized vitiligo (about two-thirds) and all subjects experienced signs of disease activity in the preceding 6 months, ensuring inclusion of patients with active disease. This has been also reported by Rosmarin et al. and Craiglow and King who stated that generalized

vitiligo was the most common presentation in those receiving systemic JAK inhibitor therapy [9,10]. The presence of patients with active progressive disease in both treatment groups allowed for a suitable environment to test the effectiveness of systemic therapy in stopping disease progression and inducing repigmentation.

Following 24 weeks of treatment, patients in both groups had significant reductions in disease severity; the oral Tofacitinib group had statistically significantly lower changes in VASI score than the OMP steroid group. The mean VASI score decreased from 8.42 ± 2.14 to 5.18 ± 1.92 in the Tofacitinib group, corresponding to a mean reduction of 3.24 ± 1.38, whereas the OMP steroid group showed a reduction from 8.57 ± 2.31 to 6.74 ± 2.28, with a mean reduction of 1.83 ± 1.19 ($P < 0.001$). These results indicate that Tofacitinib is more effective than the drug in reducing disease activity in the 24-week treatment period. Dev et al. showed that oral Tofacitinib was superior to oral dexamethasone mini-pulse therapy in terms of clinical efficacy at long-term follow-up [11]. The Janus kinase (JAK)-STAT signaling pathway is known to mediate the interferon-γ-mediated autoimmune destruction of melanocytes, and therefore, the beneficial effect of Tofacitinib is biologically plausible. JAK inhibitors have consistently been found to halt the inflammatory process that causes melanocyte loss in vitiligo and progressively improve the clinical picture, both in the laboratory and in clinical trials. [5,6,8,14]

A notable result of the current study was the significantly greater percentage of disease stabilization with Tofacitinib administered orally. Complete stabilization was achieved in 77.1% of patients receiving Tofacitinib versus 57.4% in the OMP steroid group and continued disease progression was seen in only 6.2% of patients versus 14.9% in the OMP steroid group ($P = 0.114$). Visible repigmentation was also significantly higher in Tofacitinib group (70.8%) compared to steroid group (46.8%) ($P = 0.017$). This is in line with earlier studies demonstrating the efficacy of JAK inhibition in reducing the ongoing autoimmune response and stimulating melanocyte regeneration. Rosmarin et al. showed clinically relevant repigmentation in the patients treated with oral Tofacitinib; Craiglow and King reported stabilization of active disease in most patients treated. Systematic reviews of oral mini pulse corticosteroid therapy in contrast, have found that oral mini pulse corticosteroid therapy is effective, mainly for slowing the progression of the disease, but often results in slower and less extensive repigmentation than targeted immunomodulatory therapy [9,10,15,16].

Safety data in this current study also favored oral Tofacitinib. The overall adverse event rate was 22.9% for patients on Tofacitinib, and 42.6% for patients on OMP steroid therapy ($P = 0.04$). A known adverse effect of corticosteroids, weight gain, was significantly more frequent in the OMP steroid group (17.0%) than in the Tofacitinib group (2.1%) ($P = 0.013$). Other adverse events such as acne, gastritis, upper respiratory infection and transitory elevation of liver enzymes were rare and were similar between groups. Previous investigators have reported comparable safety data for systemic JAK inhibitors, with mostly minor and reversible side effects, and no significant issues related to treatment over short-term follow-up. [8] However, oral mini pulse corticosteroid therapy has been consistently reported to be associated with weight gain, acne, cushingoid features and metabolic adverse effects even though it is proven to be effective in stabilizing active vitiligo [16]. These results suggest that while both treatments offer a generally acceptable safety profile, Tofacitinib may offer a more favorable safety profile, especially for those requiring long-term systemic treatment.

Overall clinical response also confirmed the superiority of Tofacitinib oral. Thirty-seven and a half percent of patients treated with Tofacitinib achieved excellent clinical improvement (>75%) vs 19.1% of patients treated with OMP steroid therapy. Similarly, good clinical response (50–75%) was seen in 35.4% of patients treated with Tofacitinib and in 29.8% of those treated with OMP steroid therapy; and poor response was seen in 8.3% of patients treated with Tofacitinib and 19.1% treated with OMP steroid therapy. There was a significant difference in the overall distribution of treatment response between the groups ($P = 0.041$), with better therapeutic response observed with the oral Tofacitinib treatment. These observations add to the substantial evidence that specific blockade of the JAK-STAT pathway allows for better control of disease than traditional systemic steroids and decreases the toxicity associated with systemic steroid use. While there have been some reports of similar efficacy when using the two drugs for shorter follow-up periods, the results of the current study indicate that oral Tofacitinib may be more effective in progressive vitiligo and have a safer safety profile. However, larger multicenter randomized trials with longer follow-up periods are still needed to validate the results and to evaluate the long-term efficacy and safety of Tofacitinib as an oral, first-line systemic treatment for progressive vitiligo. [5,8,14]

Conclusion

This randomized controlled trial showed that both oral Tofacitinib and oral mini pulse steroid therapy were effective in reducing the disease activity in patients with progressive vitiligo. Oral Tofacitinib, however, was found to be more effective in stabilizing the disease, inducing repigmentation and overall clinical response, and with a more favorable safety profile with less treatment-related adverse events. The results indicate that oral Tofacitinib could be a

more effective and better-tolerated systemic therapeutic approach in progressive vitiligo, compared with oral mini pulse steroid therapy. However, larger multicenter studies with longer follow-up would be justified prior to their use in routine clinical practice to assess its long-term efficacy and safety.

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