Efficacy and Safety of Oral Tofacitinib in Lichen Planus and Its Variants: A Prospective Interventional Study from a Tertiary Care Centre in Western Uttar Pradesh

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Article Info

E-ISSN: 2582-8940
Volume: 07
Issue: 02
Received: 11-04-2026
Accepted: 10-05-2026
Published: 09-06-2026
Page No: 269-273

Abstract

Background: Lichen planus (LP) is a chronic, T-cell-mediated inflammatory dermatosis in which the Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathway is increasingly implicated. Conventional therapies frequently yield incomplete or non-durable responses, creating a need for targeted options in progressive and refractory disease.

Objective: To evaluate the efficacy and safety of oral tofacitinib in patients with LP and its clinical variants.

Methods: A prospective interventional study enrolled 50 patients (aged 18–60 years) with clinically or histopathologically confirmed LP. Patients received oral tofacitinib 5 mg twice daily, escalated to 5 mg thrice daily for inadequate response, and were followed at 15-day intervals. Outcomes were assessed using the Physician Global Assessment (PGA), Visual Analog Scale (VAS) for pruritus, standardized photographic grading, and serial laboratory monitoring.

Results: Complete cutaneous response rose from 40% at 8 weeks to 72% at 18 weeks. Mean PGA fell from 3.5 to 0.8 and mean VAS from 7.6 to 1.2 (both $p < 0.001$). Classical and hypertrophic variants showed the highest excellent-response rates; the variant–response association was significant ($p < 0.05$). No serious adverse events occurred; a statistically significant but clinically manageable rise in total cholesterol was the only significant laboratory change ($p = 0.01$).

Conclusion: Oral tofacitinib was an effective and well-tolerated treatment for LP and its variants, supporting JAK–STAT inhibition as a rational therapeutic strategy in progressive or treatment-resistant disease.

DOI: https://doi.org/10.54660/IJMBHR.2026.7.2.269-273

Keywords: Lichen planus, Tofacitinib, JAK inhibitors, Pruritus, Lichen planopilaris, Treatment outcome

Introduction

Lichen planus is a chronic, immune-mediated inflammatory disorder that affects the skin, mucous membranes, hair follicles, and nails, classically presenting with pruritic, polygonal, violaceous papules and plaques. The disease is histologically defined by a dense band-like lymphocytic infiltrate at the dermo-epidermal junction, basal cell degeneration, and colloid body formation, and it most commonly affects adults in the third to fifth decades of life [1, 2]. Beyond the classical cutaneous form, several variants—hypertrophic, follicular (lichen planopilaris), oral, and nail LP—carry distinct morbidity, with follicular and nail disease in

particular capable of causing irreversible scarring and dystrophy.

Current understanding implicates an interferon-driven, cytotoxic T-cell response in LP pathogenesis, with downstream signaling routed through the JAK–STAT pathway [3]. This mechanistic insight has positioned Janus kinase inhibitors as a biologically rational therapeutic class in dermatology [5, 6].

Conventional treatments—topical and systemic corticosteroids, retinoids, and immunosuppressants—are often limited by incomplete response, relapse on withdrawal, and cumulative toxicity, particularly in chronic or refractory presentations [4].

Tofacitinib, an oral JAK1/JAK3 inhibitor, has shown promising results across multiple off-label dermatological indications, and a growing body of case reports and small series describes its benefit in difficult-to-treat LP variants [9,10]. However, prospective interventional data assessing both efficacy and safety across the spectrum of LP variants in Indian patients remain limited. The present study was undertaken to address this gap by systematically evaluating oral tofacitinib in a real-world tertiary-care cohort.

Materials and Methods

Study design and setting

This prospective interventional study was conducted in the Department of Dermatology, Venereology and Leprology at a tertiary-care teaching hospital in Western Uttar Pradesh, India, over the period March 2024 to February 2026. Institutional Ethics Committee approval was obtained and the study was conducted in accordance with ICMR and Good Clinical Practice guidelines, with written informed consent from all participants.

Participants and sample size

Patients aged 18–60 years with clinically or histopathologically diagnosed LP or its variants were enrolled. Pregnant or lactating women and patients with cardiovascular disease, active acute or chronic infection, malignancy, hematological abnormalities, or known tofacitinib hypersensitivity were excluded. Using a prevalence-based formula with 95% confidence and 3% allowable error, a base sample of 40 was inflated for a 20% anticipated attrition to a final sample of 50 patients.

Intervention and follow-up

All patients received oral tofacitinib 5 mg twice daily, with escalation to 5 mg thrice daily permitted for inadequate response. No other systemic or topical agents except emollients were allowed. Patients were reviewed every 15 days for clinical response and adverse drug reactions, with laboratory re-evaluation at 4 weeks and 3 months and continued monitoring thereafter. Baseline workup included complete blood count, comprehensive metabolic and liver panels, lipid profile, viral markers, and screening for latent tuberculosis.

Outcome assessment and statistics

Efficacy was assessed by objective cutaneous response (complete/partial/none), the 0–5 Physician Global Assessment, the 0–10 Visual Analog Scale for pruritus, and standardized photographic grading (mild ≤25%, moderate >25–<75%, excellent ≥75% improvement). Temporal trends were analyzed using repeated-measures ANOVA and the Friedman test; baseline-versus-endpoint comparisons used the paired t-test; categorical associations used the chi-square test. A p-value < 0.05 was considered statistically significant.

Results

Of the 50 patients, the largest group was aged 41–50 years (38%), with a slight male predominance (male-to-female ratio 1.08:1). Most patients (40%) presented within 7–12 months of onset, although 32% had disease exceeding one year. A progressive disease course predominated (76%), and pruritus or burning was the leading presenting symptom (88%). Classical cutaneous LP was the commonest variant (36%), followed by lichen planopilaris (24%) and hypertrophic LP (20%) (Tables 1 and 2).

Table 1: Baseline demographic and clinical characteristics (n = 50)

Characteristic	Categories — n (%)
Age (years)	18–30: 8 (16); 31–40: 13 (26); 41–50: 19 (38); 51–60: 10 (20)
Sex	Male: 26 (52); Female: 24 (48)
Duration at presentation	≤6 mo: 14 (28); 7–12 mo: 20 (40); 13–24 mo: 11 (22); >24 mo: 5 (10)
Disease course	Progressive: 38 (76); Static: 8 (16); Recurrent: 4 (8)
Symptoms / history	Pruritus or burning present: 44 (88); Positive family history: 6 (12)

Table 2: Distribution and final treatment response by LP variant (n = 50)

LP variant	n (%)	Excellent	Moderate	Poor
Classical cutaneous LP	18 (36)	14	3	1
Hypertrophic LP	10 (20)	7	2	1
Lichen planopilaris	12 (24)	6	4	2
Oral LP	6 (12)	4	1	1
Nail LP	4 (8)	1	2	1

Clinical response was progressive over the 18-week assessment window. Complete cutaneous clearance increased from 40% at 8 weeks to 60% at 12 weeks and 72% at 18 weeks. Mean PGA declined from 3.5 at baseline to 0.8, and mean VAS pruritus fell from 7.6 to 1.2, both reductions reaching high statistical significance ($p < 0.001$). By 18 weeks, 60% of patients achieved excellent (≥75%) photographic improvement (Table 3, Figures 1 and 2). Classical and hypertrophic variants achieved the highest excellent-response rates, whereas lichen planopilaris and nail LP responded more slowly; the variant–response association was statistically significant ($p < 0.05$). Among the 12 lichen planopilaris patients, complete scalp response rose from 8.3% at 4 weeks to 58.3% at 18 weeks (Friedman test, $p < 0.05$).

Patients requiring dose escalation had higher proportions of moderate and poor responses, reflecting more refractory disease (chi-square, $p < 0.05$).

Table 3: Clinical, symptomatic, and photographic response over time (n = 50)

Outcome	2 wk	4 wk	8 wk	12 wk	18 wk
Complete cutaneous response	0%	0%	40%	60%	72%
Partial cutaneous response	0%	12%	44%	28%	20%
Complete pruritus relief	4%	12%	28%	52%	68%
Excellent photographic (≥75%)	0%	10%	28%	52%	60%
Mean PGA score	3.1	2.9	2.1	1.3	0.8
Mean VAS pruritus	6.7	5.8	4.0	2.3	1.2

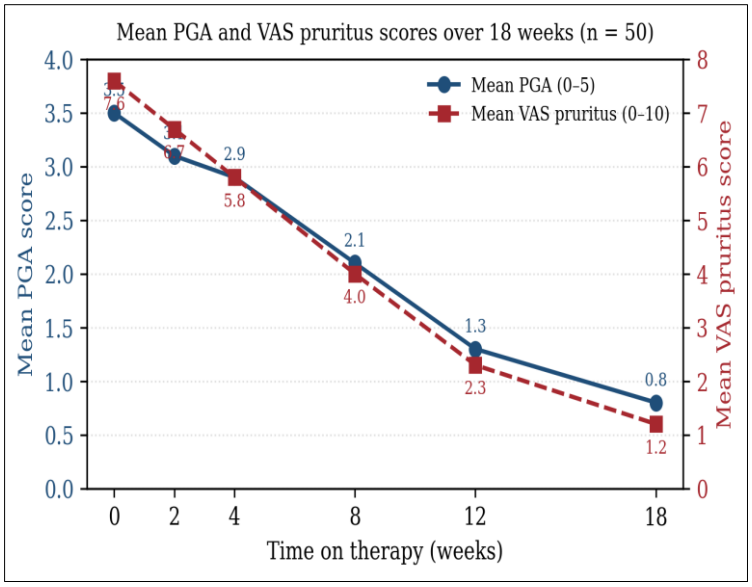

Fig 1: Mean PGA and VAS pruritus scores over 18 weeks.

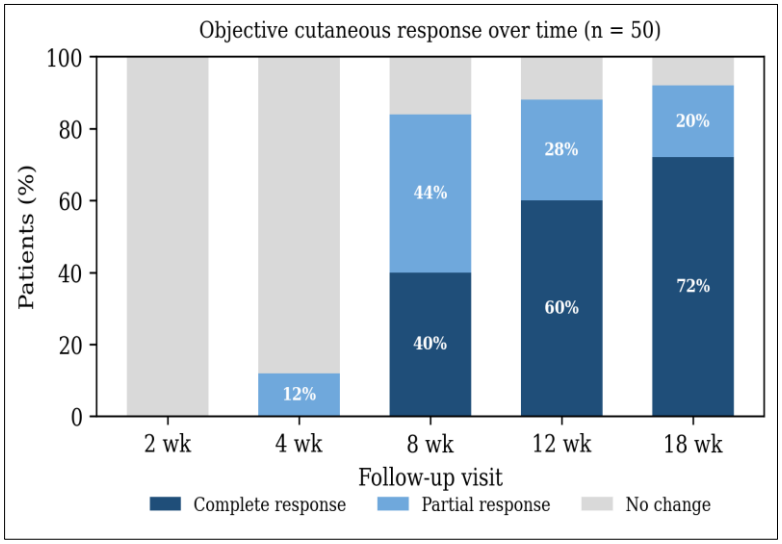

Fig 2: Objective cutaneous response at each follow-up visit.

Laboratory parameters remained largely stable. Hemoglobin, total leukocyte count, platelets, and transaminases showed only minor, non-significant drifts and stayed within reference ranges throughout follow-up. The only statistically significant change was a gradual rise in total cholesterol, from 171.6 ± 18.4 mg/dL at baseline to 186.7 ± 22.0 mg/dL at 9 months (paired t-test $p = 0.01$; repeated-measures ANOVA $p = 0.003$), which remained clinically manageable (Table 4). No serious adverse events or treatment discontinuations occurred; 74% of patients reported no adverse events, with mild hyperlipidemia the most frequent finding (Table 5).

Table 4: Baseline versus 9-month laboratory parameters (paired t-test)

Parameter	Baseline	9 months	p-value
Hemoglobin (g/dL)	12.6 ± 1.2	12.3 ± 1.0	0.08
TLC (/mm³)	6820 ± 840	6590	0.12
AST (IU/L)	26.4 ± 4.2	31.1	0.06
ALT (IU/L)	27.1 ± 4.6	32.3	0.07
Total cholesterol (mg/dL)	171.6 ± 18.4	186.7 ± 22.0	0.01*

*Statistically significant ($p < 0.05$).

Table 5: Adverse events observed during the study (n = 50)

Adverse event	n (%)
None	37 (74)
Hyperlipidemia (mild)	8 (14)
Upper respiratory tract infection	2 (4)
Headache	2 (4)
Transaminitis (transient)	1 (2)
Gastrointestinal upset	1 (2)

Discussion

This prospective study demonstrates that oral tofacitinib produces substantial, progressive, and well-tolerated improvement across the clinical spectrum of LP. The demographic profile—peak involvement in the fourth and fifth decades with a near-equal sex ratio—is consistent with established epidemiology of cutaneous LP and reflects the inclusion of multiple variants rather than a single subtype [1,2]. The marked reductions in PGA and VAS confirm that the drug improves both lesion morphology and the symptomatic burden of pruritus, which dominated the presenting complaints.

The 72% complete-response rate at 18 weeks aligns with and extends earlier observations. Damsky and colleagues reported rapid clinical control of severe LP with tofacitinib [11], while variant-focused reports have documented benefit in hypertrophic [14,15], follicular [12,13], oral erosive [17], and nail disease [16]. The higher excellent-response rates seen here in classical and hypertrophic LP, contrasted with the slower but meaningful response in lichen planopilaris and nail disease, mirror the variable kinetics described in larger variant series [18,19]. The statistically significant scalp response in lichen planopilaris is clinically notable, suggesting disease-modifying potential even in cicatricial forms, consistent with recent retrospective and randomized data [18,20].

The safety profile was reassuring. Hematological and hepatic parameters remained within normal limits, and the absence of serious infections or discontinuations is concordant with dermatological safety reviews of the JAK-inhibitor class, which identify lipid elevation among the more consistent laboratory signals requiring monitoring [7,8]. The significant but clinically manageable rise in total cholesterol observed here reinforces the recommendation for periodic lipid surveillance, and the association between dose escalation and poorer response likely reflects intrinsically more refractory disease rather than treatment failure. The principal limitations—single-centre design, modest sample size, absence of a comparator arm, and follow-up that does not capture relapse after discontinuation—temper causal inference and generalizability. Nonetheless, the consistency of response across variants and outcome measures, combined with objective photographic documentation, strengthens internal validity and supports JAK–STAT inhibition as a rational target in LP.

Conclusion

Oral tofacitinib was an effective and well-tolerated therapeutic option for lichen planus and its variants, producing significant and sustained clinical and symptomatic improvement—including 72% complete response at 18 weeks—with an acceptable safety profile dominated by mild, manageable lipid elevation. These results support the therapeutic potential of targeting the JAK–STAT pathway, particularly in progressive or treatment-resistant disease. Larger, multicentre randomized controlled trials with longer follow-up are warranted to standardize dosing, define long-term safety, and assess relapse after discontinuation.

Declarations

Ethics: Institutional Ethics Committee approval and written informed consent were obtained.
Conflict of interest: None declared.
Funding: None.

References

1. Boch K, Langan EA, Kridin K, Zillikens D, Ludwig RJ, Bieber K. Lichen planus. Front Med. 2021;8:737813.
2. Solimani F, Forchhammer S, Schloegl A, Ghoreschi K, Meier K. Lichen planus – a clinical guide. J Dtsch Dermatol Ges. 2021;19(6):864-82.
3. Vičić M, Hlača N, Kaštelan M, Brajac I, Sotošek V, Prpić Massari L. Comprehensive insight into lichen planus immunopathogenesis. Int J Mol Sci. 2023;24(3):3038.
4. Husein-ElAhmed H, Gieler U, Steinhoff M. Lichen planus: a comprehensive evidence-based analysis of medical treatment. J Eur Acad Dermatol Venereol. 2019;33(10):1847-62.
5. Damsky W, King BA. JAK inhibitors in dermatology: the promise of a new drug class. J Am Acad Dermatol. 2017;76(4):736-44.
6. Solimani F, Meier K, Ghoreschi K. Emerging topical and systemic JAK inhibitors in dermatology. Front Immunol. 2019;10:2847.
7. Samuel C, Cornman H, Kambala A, Kwatra SG. A review on the safety of using JAK inhibitors in dermatology: clinical and laboratory monitoring. Dermatol Ther. 2023;13(3):729-49.
8. Muddebihal A, Khurana A, Sardana K. JAK inhibitors in dermatology: the road travelled and path ahead, a

narrative review. Expert Rev Clin Pharmacol. 2023;16(4):279-95.
9. Motamed-Sanaye A, Khazaee YF, Shokrgozar M, Alishahi M, Ahramiyanpour N, Amani M. JAK inhibitors in lichen planus: a review of pathogenesis and treatments. J Dermatolog Treat. 2022;33(8):3098-103.
10. Abduelmula A, Bagit A, Mufti A, Yeung KC, Yeung J. The use of Janus kinase inhibitors for lichen planus: an evidence-based review. J Cutan Med Surg. 2023;27(3):271-6.
11. Damsky W, Wang A, Olamiju B, Peterson D, Galan A, King B. Treatment of severe lichen planus with the JAK inhibitor tofacitinib. J Allergy Clin Immunol. 2020;145(6):1708-10.e2.
12. Yang CC, Khanna T, Sallee B, Christiano AM, Bordone LA. Tofacitinib for the treatment of lichen planopilaris: a case series. Dermatol Ther. 2018;31(6):e12656.
13. Plante J, Eason C, Snyder A, Elston D. Tofacitinib in the treatment of lichen planopilaris: a retrospective review. J Am Acad Dermatol. 2020;83(5):1487-9.
14. Seiringer P, Lauffer F, Pilz AC, Boehmer D, Biedermann T, Eyerich K. Tofacitinib in hypertrophic lichen planus. Acta Derm Venereol. 2020;100(14):adv00220.
15. Youssef S, Bordone LA. Oral tofacitinib effectively treating eruptive and hypertrophic cutaneous lichen planus. JAAD Case Rep. 2023;37:16-20.
16. Iorizzo M, Haneke E. Tofacitinib as treatment for nail lichen planus associated with alopecia universalis. JAMA Dermatol. 2021;157(3):352-3.
17. Gowda S, Thakur V, Behera B, Garg S, Sahu D, Sethy M, *et al*. The efficacy and safety of oral tofacitinib in oral erosive lichen planus: a case series. Int J Dermatol. 2024;63(11):1561-3.
18. Goodarzi M, Dadkhahfar S, Yahyaei F, Razzaghi Z, Moravvej H. Efficacy and safety of tofacitinib in lichen planopilaris: a retrospective series of 74 patients. Arch Iran Med. 2025;28(1):24-8.
19. Sharath S, Sardana K, Khurana A, Yadav A, Singh A. Therapy-resistant hypertrophic lichen planus and its response to oral tofacitinib with a priori tissue cytokine expression: a real-world hospital-based study. Arch Dermatol Res. 2025;317(1):590.
20. Mahmoudi H, Daneshpajooh M, Dadkhahfar S, Robati R, Tehranchinia Z, Farokh P, *et al*. Efficacy and safety of tofacitinib in the treatment of adults with lichen planopilaris: a randomized placebo-controlled trial. Int Immunopharmacol. 2025;162:115129.

How to Cite This Article
Widhwani D, Saxena D, Kashyap S, Noor J, Kainam. Efficacy and safety of oral tofacitinib in lichen planus and its variants: a prospective interventional study from a tertiary care centre in Western Uttar Pradesh. International Journal of Medical and All Body Health Research. 2026;7(2):269-273. doi: https://doi.org/10.54660/IJMBHR.2026.7.2.269-273