

Case Report

Fading the Pigment: Oral Tofacitinib in Lichen Planus Pigmentosus—a Case Report

Dr. Koyana Anil Kardekar^{1*}, Dr. Vishwajeet Kalantri², Dr. Sharangdhar Kadam³

^{1*}Senior Resident, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India.

^{2,3}Junior Resident, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India.

Corresponding Author: Dr. Koyana Anil Kardekar

Senior Resident, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India.

Email: koyanaanilkardekar@gmail.com

Received: 09.08.2026, Revised: 11.09.2026, Accepted: 13.10.2026

ABSTRACT

Background: Lichen planus pigmentosus (LPP) is a chronic acquired pigmentary disorder with dark brown to slate-gray macules and patches, mainly affecting people with darker skin phototypes. Management is often difficult due to the persistence of pigmentation and inconsistent response to treatments available. Recently, Janus kinase (JAK) inhibitors have been of interest as potential treatment options for inflammatory dermatoses, such as LPP.

Case presentation: A 37-year-old woman, a farmer from Maharashtra, India, presented with progressive hyperpigmentation of the face and neck over a period of 3-4 years, with mild itching and burning on exposure to the sun. On clinical examination, diffuse slate-gray to blackish pigmentation was observed on the forehead, temples, cheeks, periorbital regions and neck. Histopathological examination showed superficial dermal pigmentary incontinence, melanophages and mild perivascular lymphocytic infiltrate. The clinical and histopathological findings were in favour of LPP. After pretreatment assessment, the patient received oral tofacitinib 5 mg twice daily for 3 months, along with topical sunscreen and photoprotection. Improvement was first noted at six weeks, and the pigmentation was estimated clinically to be reduced by about 60% at 3 months, the greatest improvement being noted in the periorbital and malar regions. No side effects were reported. Tofacitinib was discontinued after 3 months and there was no recurrence found during 1 month of post-treatment follow-up.

Conclusion: In this case, significant clinical response was achieved with the use of oral tofacitinib for three months in a patient with persistent LPP. The results of this case suggest that JAK inhibition could be a possible therapeutic approach in LPP, but concurrent photoprotection, the overlap of the diagnosis with other acquired pigmentary disorders, and the short follow-up period do not allow a causal interpretation.

Keywords: Lichen Planus Pigmentosus, Tofacitinib, JAK Inhibitor, Facial Hyperpigmentation, Pigmentary Incontinence, Case Report.

INTRODUCTION

Lichen planus pigmentosus (LPP) is a chronic acquired pigmentary dermatosis, considered a variant of lichen planus, which is mainly seen in persons with darker skin phototypes. It usually occurs in adulthood and is marked by progressive brown, gray-brown or slate-gray macules and patches, particularly on the face, neck and other sun-exposed areas. Pigmentation can be diffuse, reticulate, blotchy or perifollicular, and sometimes there is pruritus or burning. While systemic morbidity is usually not associated with the condition, chronic pigmentation in cosmetically visible areas may have a negative impact on psychological health and quality of life. The Indian subcontinent has been a source of clinical studies that have emphasized the variety of clinical presentations and the long duration of this disorder, making it

clinically relevant in this population with skin of colour [1,2].

The etiology and pathogenesis of LPP are not fully understood, but immune-mediated inflammation involving basal keratinocytes is thought to play a significant role. Immunohistochemical studies have shown that both CD4+ and CD8+ T lymphocytes are involved, especially in relatively early lesions, which is consistent with the involvement of cell-mediated immune responses in disease activity. A number of environmental and exogenous factors have been studied as aggravating or associated factors, such as ultraviolet radiation, cosmetic preparations and hair-coloring products. However, the relative importance of their contributions is not known and there is no consistent evidence of a causal connection [3,4]. In addition, a study evaluating contact

sensitization in patients with acquired dermal macular hyperpigmentation found that contact sensitization to hair-dye ingredients was clinically relevant in a small group of patients, underscoring the need to consider contact-related pigmentation in the differential diagnosis [5].

The histopathology of LPP can show varying degrees of basal epidermal injury, interface inflammation, dermal pigmentary incontinence and melanophages. These changes are due to inflammatory disruption of the epidermal pigmentary unit with deposition of melanin in dermal macrophages. But there is a significant clinicopathological overlap among LPP, ashly dermatosis, erythema dyschromicum perstans and pigmented contact dermatitis. Some features have been identified by comparative histopathological studies that might be helpful in favouring LPP, but no single feature is diagnostically specific [6]. In this context, the general idea of acquired dermal macular hyperpigmentation has been stressed, along with the need to consider clinical history, the distribution of the lesions, histopathology, and relevant history of exposure to make a diagnosis [7].

Treatment of LPP is especially difficult because inflammation and existing pigmentation of the skin might not be treated in the same way. Therapies have ranged from photoprotection, avoidance of suspected triggers, topical corticosteroids, calcineurin inhibitors and selected systemic therapies, with variable results. Topical tacrolimus was effective in achieving significant pigmentary improvement in a subset of treated subjects in an open-label prospective study, indicating a possible beneficial effect on cutaneous inflammation. However, the available therapeutic literature is still limited by small patient cohorts, diverse treatment protocols and a lack of long-term outcome data [8].

Increasing recognition of cytokine-mediated inflammatory mechanisms has stimulated interest in targeted immunomodulatory therapies for lichen planus and its variants. In particular, interferon-associated signaling through the Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathway has been implicated in inflammatory processes contributing to lichenoid tissue injury. Pharmacological inhibition of this pathway may therefore offer a potential approach to controlling disease-associated inflammation. An evidence-based review of JAK inhibitors in lichen planus reported encouraging preliminary

therapeutic outcomes across different clinical variants. However, the available evidence was largely derived from individual case reports and small observational studies, highlighting the need for more rigorous clinical investigations [9].

Despite advances in understanding the inflammatory mechanisms of lichen planus pigmentosus (LPP), its therapeutic management remains challenging. Syder et al. reviewed emerging treatment approaches for LPP and highlighted the limitations of existing therapies, including inconsistent clinical responses and the lack of standardized treatment protocols [10]. More recently, Didona et al. examined the expanding therapeutic applications of JAK inhibitors in lichen planus, discussing their potential role in targeting inflammatory pathways involved in disease pathogenesis [11]. Nevertheless, clinical evidence supporting systemic JAK inhibition specifically in LPP remains limited, and questions regarding treatment efficacy, optimal dosage, duration of therapy, and long-term safety remain unresolved. Against this background, we present a case of longstanding facial and cervical LPP in a 37-year-old woman who experienced substantial clinical improvement during a three-month course of oral tofacitinib combined with strict photoprotection, contributing further clinical evidence to the emerging therapeutic literature.

Case Presentation

A 37-year-old female from Amravati district, Maharashtra, India, came to the dermatology outpatient department with a complaint of gradual onset of hyperpigmentation of the face and neck for the past 3–4 years. Pigmentation first developed on the forehead and spread to the cheeks and then to the neck. The lesions were accompanied by mild pruritus and burning sensation, especially after exposure to sunlight. The patient was a farmer by profession and reported prolonged exposure to sunlight during routine agricultural work without adequate photoprotective measures. A history of hair-dye use was also obtained. There was no family history of similar pigmentary disorders. She had no known systemic comorbidities and was not taking any ongoing medications.

She had been treated by local practitioners intermittently over the previous two years and had experienced only partial symptomatic and cosmetic improvement. The patient had not received any treatment for about two months

prior to starting the current therapeutic program.

Clinical Examination

A dermatological examination showed diffuse slate-gray to blackish hyperpigmented patches across the forehead, bilateral temporal regions, cheeks, periorbital regions and neck. Pigmentation was mainly localized in the facial areas, particularly the forehead, periorbital areas and malar prominences (Figure 1A–C). The patient had Fitzpatrick skin phototype V. There was no involvement of the oral mucosa, scalp, nails, or other body sites. The chronic, gradually progressive course, distribution on the face and neck and characteristic pigmentation favoured a diagnosis of lichen planus pigmentosus (LPP).

Considering the clinical features, a provisional diagnosis of LPP was made and histopathological examination was performed for further evaluation.

Histopathological Examination

A biopsy of the skin from a recently developed hyperpigmented patch over the lateral aspect of the neck was performed.

Histopathology of hematoxylin and eosin-stained sections showed pigmentary incontinence with scattered brown pigment deposits in the superficial dermis. There was a mild perivascular lymphocytic inflammatory infiltrate (Figure 2), and pigment-laden macrophages (melanophages) were seen within the dermis.

Dermal pigmentary incontinence and melanophages indicated that there had been prior inflammatory injury to the epidermal pigmentary unit. The histopathological findings were interpreted as being compatible with LPP in conjunction with the clinical morphology, anatomical distribution and chronic progressive course.

Pretreatment Investigations

The patient underwent hematological, biochemical and infectious disease screening

prior to starting systemic therapy, to determine her eligibility for oral tofacitinib therapy.

Complete blood count showed hemoglobin level of 12.8 g/dL, total leukocyte count of 7,200 cells/mm³, and platelet count of 280,000/mm³. The total erythrocyte count was 4.3 million/mm³, the hematocrit was 38%, the mean corpuscular volume was 88 fL, the mean corpuscular hemoglobin was 29.8 pg, and the mean corpuscular hemoglobin concentration was 33.8 g/dL. Differential leukocyte count was 60% neutrophils, 30% lymphocytes, 3% eosinophils, 6% monocytes and 1% basophils. The levels of liver function parameters were within normal laboratory ranges, with total bilirubin at 0.7 mg/dL, aspartate aminotransferase (AST) at 24 U/L, alanine aminotransferase (ALT) at 22 U/L and alkaline phosphatase at 85 U/L. Serum albumin and total protein concentrations were 4.2 g/dL and 7.0 g/dL, respectively.

The blood urea was 25 mg/dL and serum creatinine was 0.8 mg/dL on renal function assessment. The serum electrolytes were normal, with sodium at 140 mEq/L, potassium at 4.1 mEq/L, and chloride at 102 mEq/L.

Baseline lipid profile was total cholesterol 175 mg/dL, low-density lipoprotein (LDL) cholesterol 95 mg/dL, high-density lipoprotein (HDL) cholesterol 55 mg/dL, and triglycerides 110 mg/dL.

Tuberculosis screening showed a negative Mantoux test (0 mm induration) and negative interferon-gamma release assay (IGRA). Chest radiography revealed no active pulmonary tuberculosis.

Viral screening results included non-reactive hepatitis B surface antigen (HBsAg), total hepatitis B core antibody (anti-HBc), hepatitis C antibody (anti-HCV), and HIV-1/2 screening. Hepatitis B surface antibody (anti-HBs) was 45 mIU/mL.

During the pretreatment assessment, no significant comorbidities, relevant contraindications or identifiable risk factors were noted to preclude the proposed treatment.

Table 1. Selected Laboratory Investigations before Initiation of Tofacitinib

Investigation	Result
Hematological parameters	
Hemoglobin	12.8 g/dL
Total RBC count	4.3 million/mm ³
Total leukocyte count	7,200 cells/mm ³
Neutrophils	60%
Lymphocytes	30%
Platelet count	280,000/mm ³

Hematocrit	38%
MCV	88 fL
MCH	29.8 pg
MCHC	33.8 g/dL
Liver function tests	
Total bilirubin	0.7 mg/dL
Direct bilirubin	0.2 mg/dL
AST	24 U/L
ALT	22 U/L
Alkaline phosphatase	85 U/L
Total protein	7.0 g/dL
Serum albumin	4.2 g/dL
Renal function and electrolytes	
Blood urea	25 mg/dL
Blood urea nitrogen	12 mg/dL
Serum creatinine	0.8 mg/dL
Serum sodium	140 mEq/L
Serum potassium	4.1 mEq/L
Lipid profile	
Total cholesterol	175 mg/dL
LDL cholesterol	95 mg/dL
HDL cholesterol	55 mg/dL
Triglycerides	110 mg/dL
Infectious disease screening	
Mantoux test	0 mm induration
IGRA	Negative
Chest radiograph	No active pulmonary tuberculosis
HBsAg	Non-reactive
Total anti-HBc	Non-reactive
Anti-HBs	45 mIU/mL
Anti-HCV antibody	Non-reactive
HIV-1/2 screening	Non-reactive

Abbreviations: RBC, red blood cell; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDL, low-density lipoprotein; HDL, high-density lipoprotein; IGRA, interferon-gamma release assay; HBsAg, hepatitis B surface antigen; anti-HBc, hepatitis B core antibody; anti-HBs, hepatitis B surface antibody; anti-HCV, hepatitis C virus antibody.

Therapeutic Intervention

Due to the longstanding and progressive pigmentation, the involvement of cosmetically important facial areas, and partial improvement with previous treatment, the use of a therapeutic regimen with oral tofacitinib was decided.

Oral tofacitinib 5 mg twice daily was administered to the patient for 3 months.

In addition to systemic therapy, she was instructed to apply topical sunscreen three

times a day and follow strict photoprotection. Special attention was given to reducing unprotected exposure to the sun while working in the fields.

The patient was counselled regarding adherence to the treatment plan and the importance of photoprotection. She reported good adherence to medication and sun protection during the three-month treatment period. The clinical response was assessed during follow-up by clinical examination of the affected areas and by comparing serial clinical photographs.

Treatment Response and Follow-Up

Pigmentation improvement was observed about 6 weeks after starting oral tofacitinib treatment. The response was characterized by progressive lightening of hyperpigmented areas, with further improvement during the remaining treatment period.

After three months, the pigmentation was estimated to be reduced by about 60%

clinically, as judged by dermatological examination and serial photographs. The greatest improvement was seen over the periorbital and malar areas, followed by the neck.

Comparison of clinical photographs taken before and after treatment showed a visible decrease in the intensity of facial pigmentation, especially in the periorbital and cheek areas, as shown in Figure 1A–F.

The patient expressed high satisfaction with the treatment results and with the improvement of her facial appearance. Her subjective assessment was in line with the improvement seen during clinical follow-up. No adverse

effects related to oral tofacitinib were reported during the three months of treatment. As part of clinical monitoring, laboratory investigations were repeated at the end of the treatment period. Given the good response and patient satisfaction, oral tofacitinib treatment was stopped after 3 months.

One month after the end of treatment, the patient was followed up. During this visit, the improvement was sustained, and there was no recurrence or worsening of pigmentation.

Clinical response was evaluated by serial photographic comparison and clinician estimation rather than a quantitative pigmentation score.

Clinical Timeline

Table 2. Chronological Summary of Disease Progression, Therapeutic Intervention and Clinical Outcomes

Time point	Clinical findings and management
3–4 years before presentation	Gradual onset of pigmentation over the forehead, followed by progressive involvement of the cheeks and neck
Previous two years	Intermittent treatment from local practitioners, with incomplete improvement
Two months before treatment	Treatment-free interval
At presentation (baseline)	Diffuse slate-gray to blackish hyperpigmentation over the forehead, temples, periorbital regions, cheeks, and neck; Fitzpatrick phototype V
Diagnostic evaluation	Skin biopsy showing superficial dermal pigmentary incontinence, melanophages, and mild perivascular lymphocytic infiltration
Pretreatment screening	Hematological, biochemical, lipid, tuberculosis, and viral screening performed
Treatment initiation (week 0)	Oral tofacitinib 5 mg twice daily; topical sunscreen three times daily and strict photoprotection
Week 6	Initial clinically noticeable reduction in pigmentation
Month 3	Approximately 60% clinician-estimated reduction in pigmentation, with maximum improvement in periorbital and malar regions; no reported adverse effects
End of month 3	Completion and discontinuation of oral tofacitinib
Month 4	One-month post-treatment follow-up demonstrating maintained improvement without recurrence

DISCUSSION

Lichen planus pigmentosus (LPP) is a chronic pigmentary dermatosis that can be affected by inflammatory activity, pigment deposition and environmental exposures. The present patient had several typical features of the disorder such as adult onset, Fitzpatrick skin phototype V, progressive slate-gray to blackish pigmentation and predominant facial and neck involvement. Mild itching and burning sensation with sun exposure suggested an associated inflammatory component. Histopathology showed superficial dermal pigmentary incontinence, melanophages and mild perivascular lymphocytic infiltration. The results

were consistent with LPP, but the lack of specifically documented interface changes and the history of exposure to hair dye should lead to consideration of overlapping acquired pigmentary conditions, such as pigmented contact dermatitis. The diagnosis was thus made from the overall clinicopathological presentation and not from a single histological feature.

LPP is still a difficult condition to manage as suppression of cutaneous inflammation does not always result in rapid resolution of established dermal pigmentation. A number of therapeutic options have been explored such as topical immunomodulators, systemic retinoids,

tranexamic acid and photoprotection, but with variable clinical results. Muthu et al. performed an open-label prospective study of 32 patients with LPP who were treated with oral isotretinoin 20 mg/day for 6 months with sunscreen. Of the 27 patients who finished treatment, 15 showed moderate improvement and seven patients showed more than 50% improvement. The investigators also noted relatively good results in patients who had a shorter disease duration and less extensive disease involvement [12]. In a second prospective study, Zenzari et al. assessed the efficacy of oral tranexamic acid in 20 patients with LPP and found that partial improvement occurred in some of their patients, thus indicating a potential therapeutic role but also highlighting the limitations of the available evidence [13]. The variable responsiveness of LPP to conventional systemic therapies as demonstrated in these studies underscores the need for continued research into alternative treatment options.

The Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathway has been identified as a biological target for lichenoid inflammation based on recent advances in understanding the biology of lichenoid inflammation. Signalling by interferon- γ is important for inflammatory interactions between epidermal keratinocytes and T lymphocytes. Kersh et al., by single-cell RNA sequencing and functional experiments, showed that CXCL9, CXCL10, and CCL19 are increased in the lesions of lichen planus and play a role in the recruitment of T cells [14]. These observations support the role of cytokine-driven cellular immunity in lichenoid inflammation. Tofacitinib inhibits JAK1- and JAK3-mediated signalling and could dampen downstream inflammatory pathways that contribute to keratinocyte damage. The molecular investigation was of lichen planus in general, however, and thus the conclusions cannot be assumed to establish an identical mechanism in LPP. In addition, anti-inflammatory effects may help to prevent further pigmentary damage without directly promoting the removal of melanin already deposited in the dermis.

Topical JAK inhibitors have given further evidence to support the study of this pathway in LPP. Cornman et al. reported a 63-year-old man with a history of poor response to multiple therapeutic agents who developed recalcitrant LPP of the face and neck. The patient was treated with topical ruxolitinib 1.5% cream twice a day and ongoing photoprotection. After

3 months, clinical stabilization and initial lightening were observed, and at 9 months, further lightening and reduction in pruritus was observed [15]. Asai et al. also reported that topical delgocitinib, another JAK-mediated signalling inhibitor, was effective in treating LPP [16]. While these observations indicate that local inhibition of JAK might be useful, their value is limited by small patient numbers, variability in treatment regimens, and the lack of controlled comparisons.

The oral tofacitinib evidence is especially applicable to the current case. Araghi et al. described a 50-year-old female patient with generalized LPP that involved large areas of the body, including the palms, and had failed to respond adequately to systemic corticosteroid therapy. Tofacitinib was given orally at 15 mg a day, and significant clinical improvement was noted during follow-up over about four months [17]. Our patient, however, showed pigmentation predominantly on the face and neck, with no involvement of the mucous membranes, nails, scalp or other skin areas. She received a lower total daily dose of tofacitinib, 10 mg, for three months. Improvement was first noticed after six weeks, and approximately 60% reduction in pigmentation was estimated clinically at the end of treatment. Although both patients demonstrated improvement during systemic JAK inhibition, differences in clinical distribution, treatment exposure and outcome assessment prevent conclusions regarding comparative efficacy or optimal dosage.

Over 3 months, she was given a lower total daily dose of tofacitinib (10 mg). Improvement was first noticed after 6 weeks and about 60% reduction in pigmentation was clinically estimated at the end of treatment. The clinical distribution, treatment exposure and evaluation of outcome in both patients make it difficult to draw conclusions about the comparative efficacy or optimal dose of systemic JAK inhibition.

Oral tofacitinib was also reported by Sinha and Sardana in a 48-year-old woman with persistent LPP who had frontal fibrosing alopecia. She had been prescribed several therapeutic interventions in the past without satisfactory improvement and was then started on oral tofacitinib 5 mg twice daily with strict photoprotection. By 6 months, disease progression had stabilized; by 12 months, substantial pigmentary improvement was observed; and by 18 months, near-complete clearance was documented. The drug was then

discontinued and the patient continued to be in remission for a year after treatment [18]. Our patient was given the same daily dose but differed in several important ways. She did not have associated cicatricial alopecia, showed appreciable improvement after six weeks and finished treatment after three months. This reduction in pigmentation was clinically significant, especially in the periorbital and malar areas, and was not seen to recur in the first month after stopping treatment. However, the brief treatment period and observation after treatment in our case do not allow evaluation of whether the response would be maintained over a period similar to the one outlined by Sinha and Sardana.

These reports collectively indicate that JAK inhibition could be a therapeutic option for certain patients with LPP, but there is limited evidence for this. In a recent systematic review, Shanshal and Uthayakumar studied the use of topical JAK inhibitors in various clinical presentations of lichen planus and found promising results in some inflammatory and follicular forms. However, evidence specifically concerning LPP was limited to case reports, and there was a need for controlled investigations and standardized outcome assessment [19]. In the current case, we add another clinical observation involving systemic, rather than topical, JAK inhibition, with clinically estimated improvement over a relatively short duration of therapy. It should not, however, be taken to mean that it is superior to other therapies or that it establishes a minimum acceptable period of treatment.

One of the factors to consider in our patient was the use of concomitant photoprotection. Her occupation was agriculture and she had not been using proper sun protection. During the treatment period she was instructed to use sunscreen three times a day and to take strict precautions against the sun. This improvement could be due to systemic immunomodulation and to less exposure to ultraviolet radiation. Thus, it is difficult to separate the therapeutic effect of tofacitinib from concurrent photoprotection. This is particularly important as reduction of aggravation by ultraviolet exposure may be beneficial for the gradual improvement of acquired pigmentary disorders. Also, the distribution and intensity of the response observed should be noted. The most marked decrease in pigmentation was seen in the periorbital and malar areas, and to a lesser extent in the neck. These areas are easily seen, and the patient was pleased with the

enhancement of facial appearance. An estimated reduction of approximately 60% was made based on clinical examination and serial photographs, but this is a clinician-assessed outcome and not a validated quantitative measure. Visual interpretation may be affected by variations in photographic illumination, positioning and reflectance of skin. Standardized imaging protocols and objective methods of pigmentary assessment should be used in future studies to enhance the reliability and comparability of treatment outcomes.

The possible benefits of systemic JAK inhibition should also be weighed against known safety risks. The risks associated with the use of tofacitinib include serious infections, laboratory abnormalities, thromboembolic events, malignancy and major adverse cardiovascular events. Ytterberg et al. reported increased incidence of major adverse cardiovascular events and malignancy in patients taking tofacitinib compared with patients taking tumour necrosis factor inhibitors in the ORAL Surveillance trial. Importantly, the patients had rheumatoid arthritis, were aged 50 years or older and had at least one additional cardiovascular risk factor [20]. The results of this study cannot be directly extrapolated to a 37-year-old woman without identified systemic comorbidities, but they do emphasize the need for careful risk assessment and treatment monitoring. In the current case, pretreatment hematological and biochemical workup, lipid profile, and infectious disease screening were done prior to starting tofacitinib. No adverse effects were reported during treatment and repeat laboratory testing was performed after 3 months. The lack of complications during a short course of treatment in one patient, however, does not prove the long-term safety of tofacitinib for LPP.

There are a number of caveats to note. First, the improvement was seen with combined therapy with oral tofacitinib and strict photoprotection, which prevented attribution of the clinical response to tofacitinib alone. Secondly, the pigmentation reduction was clinically estimated without a validated severity index, spectrophotometric analysis or a standardized colorimetric measurement. Third, the patient's treatment history was not detailed enough to establish resistance to specific conventional treatments. Fourth, the clinical and histopathological findings were in favour of LPP, but the history of hair-dye use and the nonspecific pigmentary changes on biopsy mean that there is some doubt about a possible

contact-allergic contribution. Lastly, the follow-up period of one month following the end of treatment was too short to assess the durability of remission and the potential for delayed recurrence.

However, the current case contributes to the sparse published experience of using oral tofacitinib in LPP. The main clinical finding was an approximately 60% clinician-estimated improvement in longstanding facial and cervical pigmentation after three months of oral tofacitinib 5 mg twice daily combined with concurrent photoprotection, without reported adverse effects or recurrence during the short-term follow-up period. The results indicate that JAK inhibition may be a potential therapeutic approach but should be explored further before it becomes a standard treatment. Future studies with well-defined diagnostic criteria, standardized photographic documentation, validated pigmentary outcome measures, thorough safety assessment, and longer follow-

up should be conducted to assess the efficacy, optimal treatment duration, and long-term risk-benefit profile of systemic JAK inhibitors in LPP.

CONCLUSION

We report a 37-year-old woman with longstanding, progressive facial and cervical pigmentation clinically diagnosed as lichen planus pigmentosus who experienced approximately 60% clinician-estimated improvement during three months of treatment with oral tofacitinib 5 mg twice daily and concurrent strict photoprotection.

Improvement began after six weeks, treatment was reportedly well tolerated, and no recurrence was observed during one month of post-treatment follow-up.

While the clinical response is encouraging, this isolated observation cannot establish the independent efficacy of tofacitinib or its long-term safety in LPP. Further systematic investigation is needed before it can be considered an established treatment option.

Figure Legends



Figure 1. Clinical Photographs of Lichen Planus Pigmentosus before and After Treatment. (A–C) Pretreatment Photographs Showing Diffuse Slate-Gray to Blackish Hyperpigmentation Involving the Forehead, Periorbital Regions, Temples, Cheeks and Neck. (D–F) Corresponding Photographs Obtained After Three Months of Treatment With Oral Tofacitinib 5 Mg Twice Daily and Concurrent Photoprotection, Demonstrating The Documented Clinical Improvement In Facial Pigmentation.

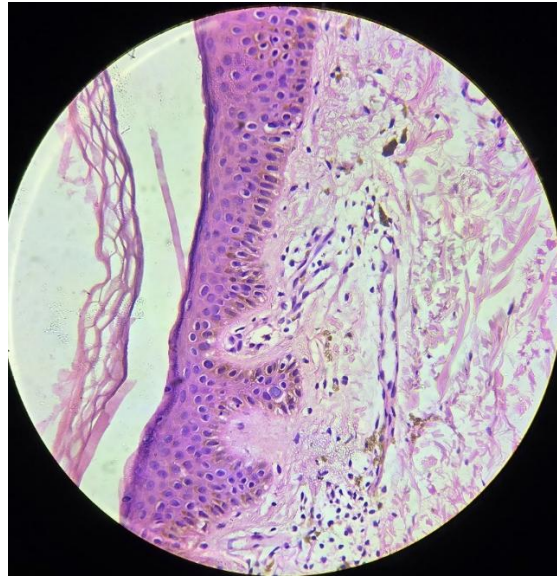


Figure 2. Histopathological Examination of a Skin Biopsy Obtained From A Recently Developed Hyperpigmented Patch Over the Lateral Neck. Scattered Brown Pigment Deposits Within The Superficial Dermis, Dermal Pigment-Laden Macrophages (Melanophages) and Mild Perivascular Lymphocytic Infiltration Were Reported (Hematoxylin And Eosin, Reported Magnification ×40).

Declaration of Patient Consent

Written informed consent for publication of the patient's clinical information and identifiable photographs was duly obtained before submission.

Conflicts of Interest

None

Funding

None

REFERENCES

1. Robles-Méndez JC, Rizo-Frías P, Herz-Ruelas ME, Pandya AG, Ocampo Candiani J. Lichen planus pigmentosus and its variants: review and update. *International Journal of Dermatology*. 2018;57(5):505-514. DOI: [10.1111/ijd.13806](https://doi.org/10.1111/ijd.13806)
2. Mendiratta V, Sanke S, Chander R. Lichen planus pigmentosus: A clinico-etiological study. *Indian Dermatology Online Journal*. 2019;10(3):288-292. DOI: [10.4103/idoj.IDOJ_253_18](https://doi.org/10.4103/idoj.IDOJ_253_18)
3. Bhat RM, Mathanda TR, Jayaprakash CS, Dandakeri S. Clinical, histopathological characteristics and immunohistochemical findings in lichen planus pigmentosus. *Indian Journal of Dermatology*. 2017;62(6):612-617. DOI: [10.4103/ijd.IJD_148_17](https://doi.org/10.4103/ijd.IJD_148_17)
4. Kumarasinghe SPW, Pandya A, Chandran V, et al. A global consensus statement on ashy dermatosis, erythema dyschromicum perstans, lichen planus pigmentosus, idiopathic eruptive macular pigmentation, and Riehl's melanosis. *International Journal of Dermatology*. 2019;58(3):263-272. DOI: [10.1111/ijd.14189](https://doi.org/10.1111/ijd.14189)
5. Bishnoi A, Vinay K, Arshdeep, et al. Contact sensitization to hair colours in acquired dermal macular hyperpigmentation: Results from a patch and photo-patch test study of 108 patients. *Journal of the European Academy of Dermatology and Venereology*. 2019;33(7):1349-1357. DOI: [10.1111/jdv.15576](https://doi.org/10.1111/jdv.15576)
6. Rutnin S, Udompanich S, Pratumchart N, Harnchoowong S, Vachiramon V. Ashy dermatosis and lichen planus pigmentosus: The histopathological differences. *BioMed Research International*. 2019;2019:5829185. DOI: [10.1155/2019/5829185](https://doi.org/10.1155/2019/5829185)
7. Sarkar R, Vinay K, Bishnoi A, et al. A Delphi consensus on the nomenclature and diagnosis of lichen planus pigmentosus and related entities. *Indian Journal of Dermatology, Venereology and Leprology*. 2023;89(1):41-46. DOI: [10.25259/IJDVL_804_2021](https://doi.org/10.25259/IJDVL_804_2021)
8. Al-Mutairi N, El-Khalawany M. Clinicopathological characteristics of lichen planus pigmentosus and its response to tacrolimus ointment: An open label, non-randomized, prospective study. *Journal of the*

- European Academy of Dermatology and Venereology*. 2010;24(5):535-540. DOI: [10.1111/j.1468-3083.2009.03460.x](https://doi.org/10.1111/j.1468-3083.2009.03460.x)
9. Abdulmula A, Bagit A, Mufti A, Yeung KCY, Yeung J. The use of Janus kinase inhibitors for lichen planus: An evidence-based review. *Journal of Cutaneous Medicine and Surgery*. 2023;27(3):271-276. DOI: [10.1177/12034754231156100](https://doi.org/10.1177/12034754231156100)
10. Syder NC, Sicco KL, Gutierrez D. Updates in therapeutics for lichen planus pigmentosus. *J Drugs Dermatol*. 2022;21(3):324-330. DOI: [10.36849/JDD.6454](https://doi.org/10.36849/JDD.6454)
11. Didona D, Caposiena Caro RD, Calabrese L, D'Onghia M, Galluccio G, Di Nicola MR, et al. Use of JAK inhibitors in lichen planus: An update. *Medicina (Kaunas)*. 2025;61(6):1056. DOI: [10.3390/medicina61061056](https://doi.org/10.3390/medicina61061056)
12. Muthu SK, Narang T, Saikia UN, Kanwar AJ, Parsad D, Dogra S. Low-dose oral isotretinoin therapy in lichen planus pigmentosus: An open-label non-randomized prospective pilot study. *Int J Dermatol*. 2016;55(9):1048-1054. DOI: [10.1111/ijd.13293](https://doi.org/10.1111/ijd.13293)
13. Zenjari L, Elfetoiki FZ, Hali F, Skalli H, Chiheb S. Oral tranexamic acid in the treatment of lichen planus pigmentosus: A prospective study of 20 cases. *Ann Dermatol Venereol*. 2020;147(12):818-822. DOI: [10.1016/j.annder.2020.06.017](https://doi.org/10.1016/j.annder.2020.06.017)
14. Kersh AE, Sati S, Huang J, Murphy C, Ahart O, Leung TH. CXCL9, CXCL10, and CCL19 synergistically recruit T lymphocytes to skin in lichen planus. *JCI Insight*. 2024;9(20). DOI: [10.1172/jci.insight.179899](https://doi.org/10.1172/jci.insight.179899)
15. Cornman HL, Wei E, Manjunath J, et al. Recalcitrant lichen planus pigmentosus treated with topical ruxolitinib. *JAAD Case Rep*. 2023;42:84-86. DOI: [10.1016/j.jdc.2023.10.012](https://doi.org/10.1016/j.jdc.2023.10.012)
16. Asai J, Ohta R, Maruyama A, Katoh N. Lichen planus pigmentosus successfully treated with topical delgocitinib. *J Dermatol*. 2024;51(7):e244. DOI: [10.1111/1346-8138.17151](https://doi.org/10.1111/1346-8138.17151)
17. Araghi F, Beyki F, Rakhshan A, MoravvejFarshi H. Generalized lichen planus pigmentosus and a new management approach. *Clin Case Rep*. 2024;12(6). DOI: [10.1002/ccr3.8829](https://doi.org/10.1002/ccr3.8829)
18. Sinha S, Sardana K. Oral tofacitinib for refractory lichen planus pigmentosus coexisting with frontal fibrosing alopecia. *Indian Dermatol Online J*. 2026;17(5):716-718. DOI: [10.4103/idoj.idoj_515_25](https://doi.org/10.4103/idoj.idoj_515_25)
19. Shanshal M, Uthayakumar A. Topical JAK inhibitors in the lichen planus spectrum: A systematic review. *Ann Dermatol Venereol*. 2026;153(3):103512. DOI: [10.1016/j.annder.2026.103512](https://doi.org/10.1016/j.annder.2026.103512)
20. Ytterberg SR, Bhatt DL, Mikuls TR, et al. Cardiovascular and cancer risk with tofacitinib in rheumatoid arthritis. *N Engl J Med*. 2022;386(4):316-326. DOI: [10.1056/NEJMoa2109927](https://doi.org/10.1056/NEJMoa2109927)