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CASE REPORT

Successful Treatment of Nail Lichen Planus by the Janus Kinase 1 Upadacitinib and Literature Review

Xingyun Zhao[1,*], Jianbo Zhong[1,*], Yanzhen Xu[2], Yingzhi Luo[1], Yunmi Qiu[1], Liming Wu[1], Ping Yang[1]

[1]Department of Dermatology, Affiliated Hangzhou First People's Hospital, School of Medicine, Westlake University, Hangzhou, Zhejiang, People's Republic of China; [2]Department of Pathology, Affiliated Hangzhou First People's Hospital, School of Medicine, Westlake University, Hangzhou, Zhejiang, People's Republic of China

*These authors contributed equally to this work

Correspondence: Ping Yang, Department of Dermatology, Affiliated Hangzhou First People's Hospital, School of Medicine, Westlake University, No. 261, Huansha Road, Hangzhou, Zhejiang, 310006, People's Republic of China, Tel/Fax +86 13515819369, Email yangping-apple@163.com

Abstract: Nail lichen planus (NLP) is a chronic inflammatory condition that can lead to considerable cosmetic and functional impairment. Failure to administer prompt and effective treatment may result in the development of permanent scarring and nail loss. The precise pathogenesis of NLP remains poorly understood, and there is currently an absence of safe and effective treatment options. Although not FDA-approved for the treatment of lichen planus, Janus kinase (JAK) inhibitors have shown considerable promise as therapeutic agents for a variety of dermatoses. This case report describes a patient with NLP who showed improvement after six months of treatment with upadacitinib, a selective JAK1 inhibitor. Changes were assessed using the Nail Lichen Planus Severity Index (NALSI) score. Following medication administration, the total score of the NALSI for the patient's nail involvement decreased from 146 to 37. However, a mild recurrence was observed following the reduction of the medication dosage (NALSI score to 47).

Keywords: upadacitinib, nail lichen planus, nail bed atrophy, inflammatory nail disease, janus kinase inhibitors, NALSI

Introduction

Lichen planus is an autoimmune inflammatory skin disorder characterized by pruritic and violaceous papules. Although it can impact any area of the body, it most frequently manifests on the extremities of middle-aged individuals. Nail involvement is observed in up to 10% of patients with lichen planus, with rare cases in which all twenty nails may be affected.[1,2] NLP can lead to thickening, atrophy, and roughening of the nail plate. In severe cases, it can result in complete destruction of the nail bed or onychomadesis (nail plate shedding).[2] Several therapeutic approaches are employed in the clinical management of NLP, including corticosteroids, immunosuppressants, retinoids, laser therapy and intralesional glucocorticoid injections. However, the efficacy of these treatments remains uncertain, and they may be associated with significant adverse effects.[3] A series of case reports have confirmed that tofacitinib (a JAK 1/2/3 inhibitor) and baricitinib (a JAK 1/2 inhibitor), are safe and effective in the treatment of NLP.[4–6] This article presents a case of successful treatment of NLP with the highly selective JAK1 inhibitor upadacitinib, which resulted in favorable therapeutic outcomes. The subsequent sections provide a detailed account of the case and treatment process.

Case Presentation

A 33-year-old female patient presented to Hangzhou First People's Hospital with a 12-year history of nail thickening and deformation affecting all fingers on both hands. The patient reported that 12 years ago, several of her nails began to exhibit deformities, accompanied by intermittent swelling and itching, and subsequently progressed to involve all nails. The patient had sought treatment at multiple institutions, receiving oral glucocorticoids, oral retinoids, and topical steroid occlusion, however, these therapies proved ineffective. This condition has significantly affected her daily life, making urgent effective treatment and intervention essential. The severity of nail lesions in patients was quantitatively evaluated by using the recently proposed

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NALSI.[7] Physical examination revealed that all fingernails and toenails were thickened and deformed, with some nails pitting. There is atrophy of the nail bed, splinter hemorrhage, and mild erythema and swelling of the proximal nail fold were observed (Figure 1, NALSI total score: 146). TMycological examination of the nails yielded negative results. Histological examination of nail biopsies revealed wedge-shaped hypergranulosis, irregular acanthosis, and lymphocytic banded infiltration within the nail matrix (Figure 2). The diagnosis of NLP was ultimately confirmed based on clinical appearance and histopathological findings. Treatment with upadacitinib, 15 mg daily, was initiated, and after 6 months, significant improvement in the appearance of the nails was noted (Figure 3, NALSI total score: 37). From the sixth month onwards, the dose was reduced to once every two days. Three months later, partial fingernail pitting was observed, along with a mild recurrence of the condition (Figure 4, NALSI total score: 47). Despite the limited recurrence, the patient remained satisfied with the disease control, so the treatment dose was maintained at 15 mg every two days. Blood test results conducted before and after upadacitinib treatment showed no significant changes in various indicators, including routine tests, coagulation function, D-dimer levels, and liver and kidney function. The patient tested negative for both hepatitis B virus screening and latent tuberculosis infection testing. The follow-up plan involves monthly re-examinations and infection/thrombosis-related testing at three-month intervals.

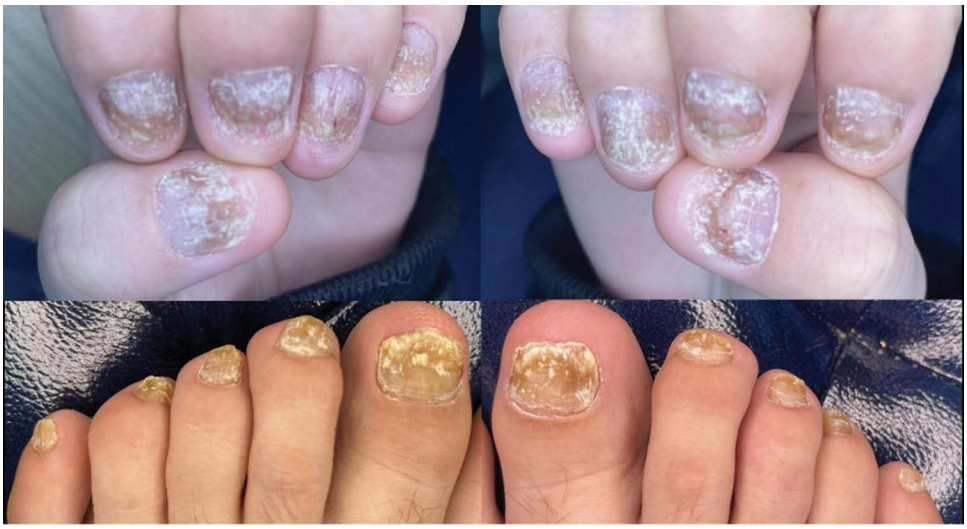

Figure 1 Clinical manifestation of twenty nails with varying degrees of thickening and deformation before upadacitinib treatment. The total NALSI score was 146.

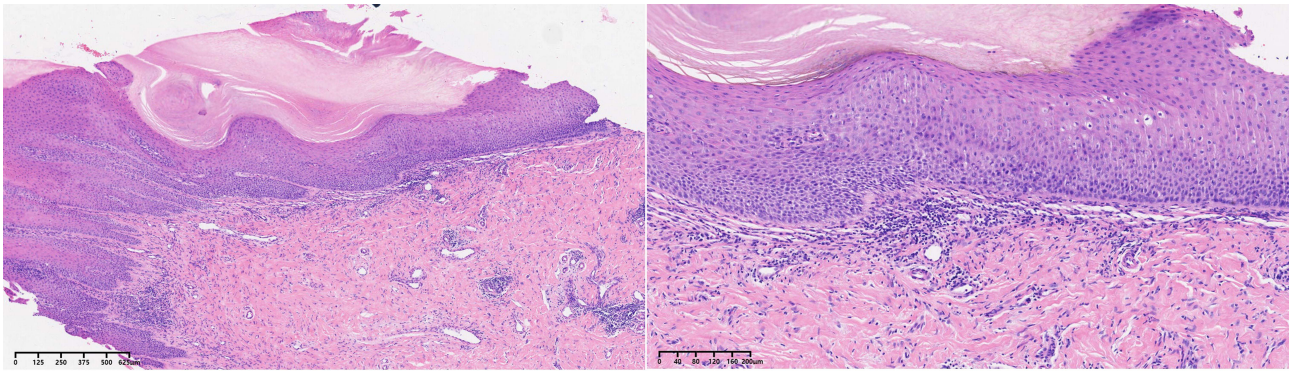

Figure 2 The biopsy specimen demonstrates a band of lymphocytic infiltration surrounding the matrix and eponychium, along with focal areas of hyperkeratosis and hypergranulosis.

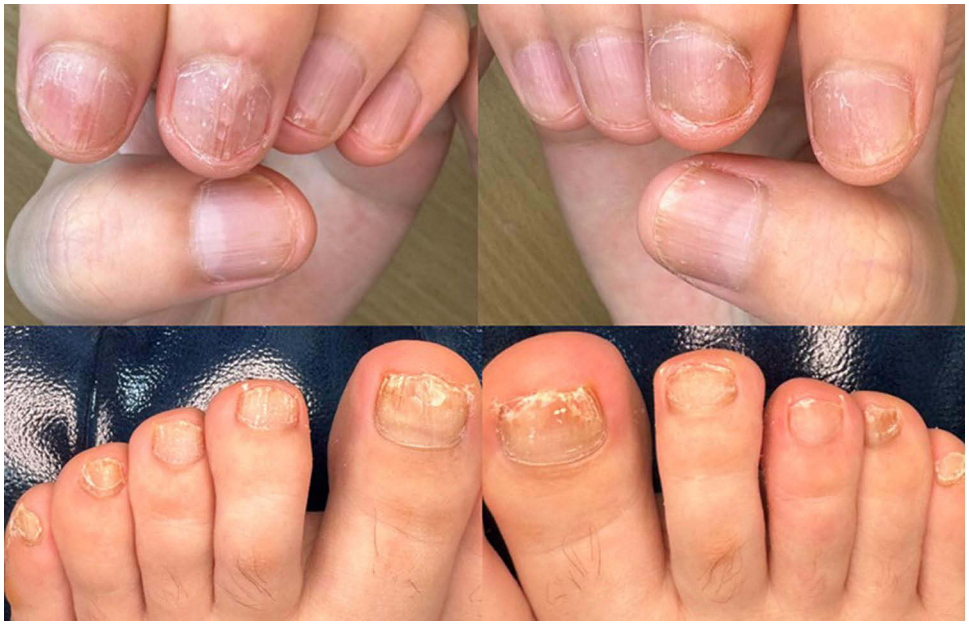

Figure 3 After 6 months of upadacitinib treatment, the nail plates showed significant improvement. The total NALSI score was 37.

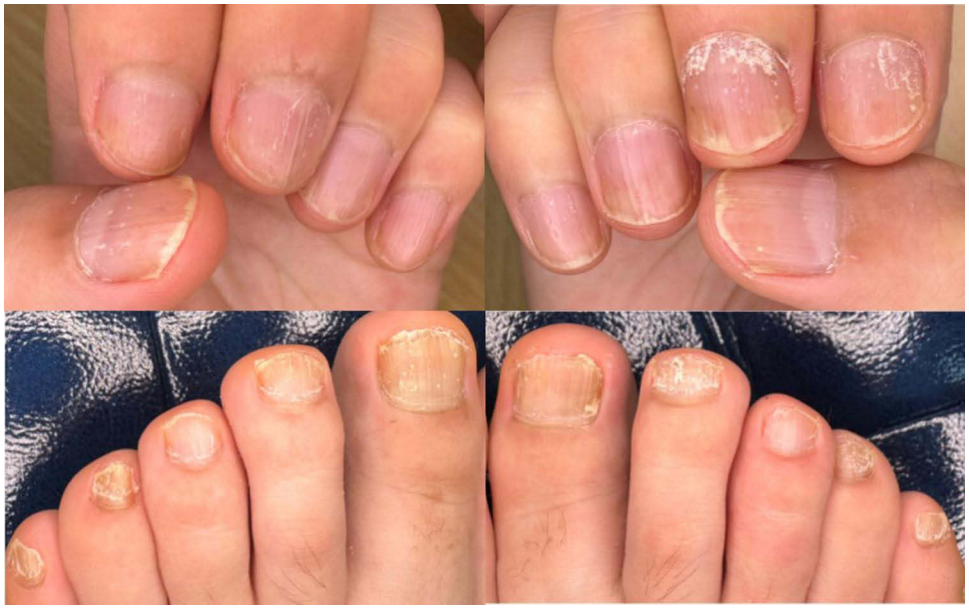

Figure 4 Upadacitinib was reduced to 15 mg every other day for 3 months, with a mild recurrence of the condition in nails. The total NALSI score was 47.

Discussion

Lichen planus is a chronic, inflammatory, autoimmune dermatosis that can result in significant functional impairment for patients.[8] It typically manifests as itchy, polygonal, flat papules and plaques, often located on the flexor surfaces of the limbs, and frequently affects the mucous membranes and nails as well.[9] Clinically, NLP manifests as longitudinal ridges and grooves, thinning, fissuring, splitting or notching of the nail plate, with the potential for pterygium formation and anonychia.[3,9] NLP can lead to significant discomfort and even permanent nail damage, underscoring the importance of prompt treatment. Several treatment options are available, with high-potency topical or intralesional corticosteroids as primary choices. Intralesional injection is a technique that involves injecting high-efficiency corticosteroids into the proximal nail fold, enabling targeted drug delivery to the nail matrix.[2] A retrospective study showed that 87.5% of

patients experienced significant improvement in nail symptoms, but common adverse reactions included injection pain and subungual hematoma. And Oral administration of alitretinoin is effective in treating NLP.[2] Research has found that 64% of patients experienced relief or significant improvement in their condition. The common adverse reaction is dry lips.[2] However, these may not suit all patients, particularly those with widespread lesions.[3]

The pathogenesis of lichen planus remains incompletely understood. Studies suggest that the condition may be triggered by keratinocytes responding to cytotoxic signals mediated by CD8+ T cells.[10] Studies have reported that cytokines are closely related to the occurrence of lichen planus, especially TNF-α, which plays a key role in the activation of Langerhans cells and the apoptosis of basal keratinocytes.[11] Increasing evidence indicates that lichen planus is a disease driven by Th1/2 cells, and the key cytokines (IFN-γ and IL-10) involved in its pathogenesis function through the JAK-STAT signaling pathway, suggesting that JAK inhibition may be a feasible therapeutic target.[11,12]

Furthermore, recent research has highlighted the potential advantages of oral JAK inhibitors for managing both cutaneous and oral lichen planus.[8,13] The patient, a nurse, experienced significant disruption to her work due to nail changes affecting both hands. Despite attempting various treatments, the disease remained inadequately controlled. We selected upadacitinib to treat our patient's NLP, given the inflammatory nature of the condition and preclinical evidence supporting the efficacy of JAK inhibition in various dermatoses, particularly lichen planus.[8]

Members of the JAK family, including JAK1, JAK2, JAK3, and TYK2, are non-receptor protein tyrosine kinases that act as intracellular second messengers. They enable cells to receive extracellular cytokine signals and regulate key biological processes.[12,14] JAK inhibitors suppress the gene transcription of pro-inflammatory cytokines by blocking the intracellular signaling pathways mediated by JAK and signal transducer and activator of transcription (STAT) proteins.[15] Upadacitinib, a selective JAK1 inhibitor, has been approved for the treatment of conditions such as ulcerative colitis, rheumatoid arthritis, psoriatic arthritis, atopic dermatitis, and ankylosing spondylitis.[12,16,17] Currently, JAK inhibitors have shown promising efficacy in treating NLP.[12] Several case reports have documented the use of JAK inhibitors have shown favorable efficacy and safety outcomes.[4–6,18–20] Currently, research has shown that upadacitinib is effective for treating oral lichen planus.[21,22] And a meta-analysis indicates that upadacitinib has a significant therapeutic effect on patients with moderate to severe atopic dermatitis.[23] There is limited literature available regarding the application of upadacitinib in the treatment of NLP. In the present case, the selective JAK1 inhibitor upadacitinib was initially used as monotherapy for NLP, resulting in a well-tolerated response. Pan-JAK inhibitors block IL-6-induced phosphorylation of STAT1, STAT3, and STAT5, preventing JAK1/JAK3 phosphorylation and downstream signaling. Tofacitinib inhibits dendritic cell antigen presentation by blocking IFN-γ signaling, reducing T lymphocyte stimulation.[12] And the effectiveness of upadacitinib in treating NLP may be attributed to its ability to reduce the function of pro-inflammatory interleukins, temporarily boost lymphocyte counts, and slightly lower immunoglobulin levels.[24] Nevertheless, the use of these drugs resulted in a greater likelihood of negative side effects, such as significant cardiovascular issues, oncological conditions, and venous thromboembolism, which prompted the FDA to issue strong black-box cautions.[25,26]

We performed an extensive search on PubMed for eligible articles about nail lichen planu from 2015 to 2025, using the following key words: "nail lichen planu"; "JAK kinase"; "treatment" and "case report". After the screening process, 6 articles met the criteria for eligibility and were included.[4–6,18–20] Table 1 presents the characteristics of patients, as well as data on treatment and response. Overall, there have been few reports of successfully treated cases of NLP.

Therefore, selective JAK inhibitors may provide a potential advantage over pan-JAK inhibitors due to their potentially improved safety profile. Based on the safety data of upadacitinib in atopic dermatitis,[27] the patient was treated with off-label upadacitinib for NLP under fully informed consent, with no adverse reactions reported during the treatment period. The patient will continue to be monitored for drug tolerability and long-term outcomes.

Our case reports suggest that upadacitinib may be both safe and effective for the treatment of NLP. However, there are several limitations, including the lack of long-term follow-up and the small number of cases. Future research should expand case analyses and clinical trials to evaluate the long-term efficacy and safety of upadacitinib in this patient population and clarify the immunological effects of JAK1 inhibition in NLP.

Table 1 JAK Inhibitors in Reported NLP Cases

Study Number	First Author (Year of Publication)	Sex/Age	Disease Duration (Years)	Concomitant Diseases	Treatment Previously of NLP	JAK Inhibitor and Treatment Protocols	Efficacy and Recurrence	Adverse Events
1	Matilde Iorizzo, Eckart Haneke (2021)[4]	F/57y	1.5y	Hypothyroidism and rheumatoid arthritis	Intralesional triamcinolone injections; oral acetonide; oral alitretinoin	Tofacitinib 5 mg BID	Consistent improvement in 6 months	None
2	Pünchera and Laffitte (2021)[5]	F/60y	1y	NA	Intramuscular triamcinolone injections; oral acitretin; oral methotrexate	Baricitinib, 4 mg QD, reduced to 2 mg QD at month 11	Complete remission in 6 months, and small recurrence after the dose reduction	None
3	He J (2023)[18]	M/30y	2y	NA	Topical glucocorticoid encapsulation therapy; oral acitretin;	Baricitinib, 4 mg QD	Completely clear after 6 months	None
4	Huang and Shi (2023)[6]	F/41y	2y	NA	Topical treatment	Tofacitinib 5 mg BID	Significant improvement in 6 months	None
5	He and Yang (2024)[19]	F/39y	1y	NA	Topical treatment	Abrocitinib 100 mg QD, reduced to QOD at month 4	Significant improvement in 6 months	None
6	Luo and Wu (2025)[20]	F/31y	3y	NA	Topical treatment	Abrocitinib 100 mg QD	Significant improvement in 6 months, and an obvious recurrence after a short discontinuation	None
7	Our case	F/30	12y	NA	Topical glucocorticoid encapsulation therapy; oral corticosteroids; oral acitretin;	Upadacitinib, 15 mg QD, reduced to 15 mg QOD at month 6	Complete remission in 6 months, and small recurrence after the dose reduction	None

Abbreviations: F, female; M, male; Y, year; NA, not applicable; NLP, nail lichen planus; BID, twice daily; QD, once daily; QOD, every other day.

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Ethical Statement and Informed Consent

Written informed consent was secured from the patient for the dissemination of their medical case details and any associated images. The ethics committee of Hangzhou First People's Hospital approved to publish the case details (2025ZN112-1).

Consent Statement

In this study, patients provided written informed consent for the publication of this case information and accompanying images.

Disclosure

The authors report no conflicts of interest in this work.

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