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CASE REPORT

# Effectiveness of Abrocitinib in Atopic Dermatitis Presenting with Hand–Foot Eczema and Nail Dystrophy: A Case Report

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**Abstract:** Eczema-associated nail dystrophy is an uncommon but clinically relevant manifestation of atopic dermatitis (AD), which can lead to substantial structural and functional impairment and impose significant therapeutic challenges. However, evidence regarding systemic treatment for AD-related nail involvement remains limited. We report a case of AD presenting predominantly with refractory hand and foot eczema and marked nail dystrophy who achieved notable improvement following treatment with the selective Janus kinase 1 (JAK1) inhibitor abrocitinib, with cutaneous symptoms improving within several weeks after dose escalation and both fingernail and toenail abnormalities showing gradual structural recovery over a 12-month follow-up. Furthermore, the treatment was well tolerated throughout the follow-up period. This case suggests that abrocitinib may represent a promising therapeutic option for eczema-associated nail changes in patients with AD.

**Keywords:** atopic dermatitis, nail dystrophy, abrocitinib, Janus kinase 1 inhibitor

## Introduction

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder characterized by eczematous lesions and pruritus.[1] Chronic hand and foot eczema is one of the common clinical phenotypes of AD and often follows a more persistent and refractory course due to continuous environmental exposure and inherent barrier fragility, which may markedly impair daily hand function.[2] Persistent eczematous inflammation may also involve the nail folds and distal phalanges, resulting in eczematous nail dystrophy, considerable functional and cosmetic impairment, and significantly lower quality of life.[3] Immune dysregulation plays a central role in the pathogenesis of AD, with type 2 helper T (Th2) cytokines, including interleukin (IL)-4, IL-13, and IL-22(3), mediating inflammation at least in part through the Janus kinase 1 (JAK1) signaling pathway.[4] Aberrant activation of this pathway contributes to epidermal barrier dysfunction and chronic inflammation.[5] Current evidence suggests that nail abnormalities in AD are largely associated with involvement of the nail unit, particularly the nail matrix and periungual[6] inflammation. In the context of cytokine-driven immune dysregulation, the proliferative and differentiation programs of nail unit keratinocytes may be disrupted, thereby impairing normal nail plate formation.[4] Meanwhile, downstream STAT signaling mediated by JAK1, especially STAT3, is closely involved in keratinocyte proliferation and differentiation. On this basis, it is conceivable that selective JAK1 inhibition may exert a beneficial effect on eczema-associated nail changes by suppressing cytokine-driven inflammation and partially correcting abnormal keratinization within the nail unit.[7] Abrocitinib, a selective JAK1 inhibitor, has achieved rapid and substantial efficacy in patients with moderate-to-severe AD by blocking JAK1-mediated cytokine signaling.[4,8] Nevertheless, there are limited data specifically regarding the use of JAK1 inhibitors for AD-associated nail dystrophy, and no standardized therapeutic approach has been established so far. Here, we report a rare case of AD presenting predominantly with chronic hand and foot eczema and marked nail dystrophy. The patient

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exhibited sustained and significant improvement after treatment with abrocitinib, highlighting the role of selective JAK1 inhibition in this challenging clinical phenotype.

## Case Presentation

We report a 30-year-old woman who presented with a one-year history of recurrent eczematous lesions affecting both hands and feet, accompanied by progressive nail changes. The disease course was chronic and relapsing, predominantly affecting acral areas, with frequent and refractory flares. She has been diagnosed with eczema, tinea pedis, and onychomycosis. Previous treatments, including oral antihistamines, itraconazole, multiple topical corticosteroids, and antifungal agents, led to limited and unsustained improvement. The patient also mentioned a history of allergic rhinitis, suggesting an underlying atopic diathesis. Physical examination revealed extensive eczematous dermatitis of the hands and feet, characterized by erythema, vesiculation, fissuring, and marked pruritus. Nail involvement was prominent, with onycholysis, nail plate thickening, and surface roughness affecting the left great toenail and the left middle and little fingernails (Figure 1A). Serial clinical photographs illustrating the clinical course of the cutaneous and nail lesions are presented in Figure 1A–E. Laboratory examinations showed normal routine biochemical tests, thyroid function tests, and autoimmune-related parameters, and fungal examinations were negative. Serum total IgE was mildly elevated (135.4

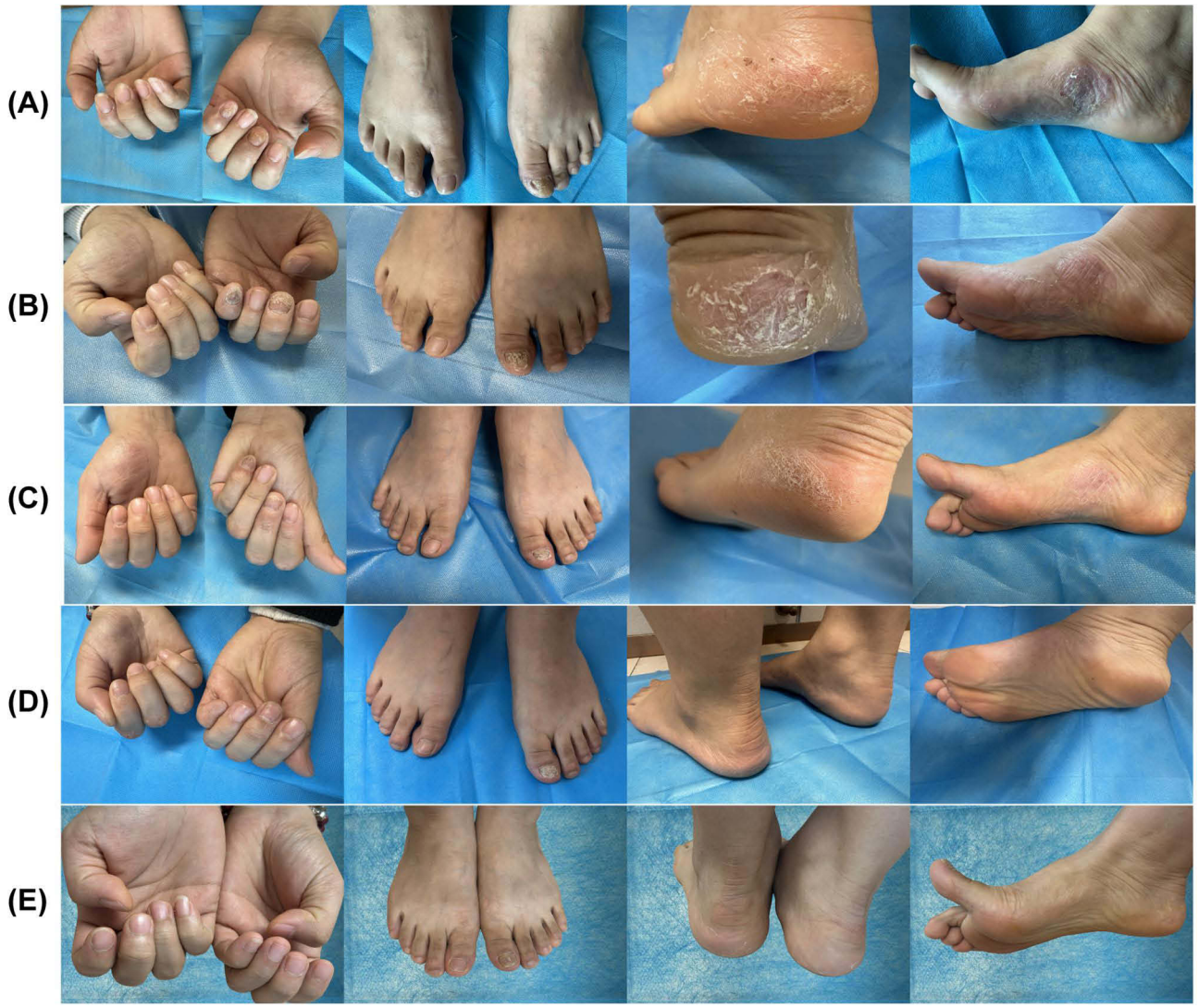


**Figure 1** The clinical course of hand and foot eczema with associated nail dystrophy during treatment with abrocitinib. Serial clinical images showing progression over time. (**A**) Baseline; (**B**) 6 weeks; (**C**) 4 months; (**D**) 8 months; (**E**) 12 months.

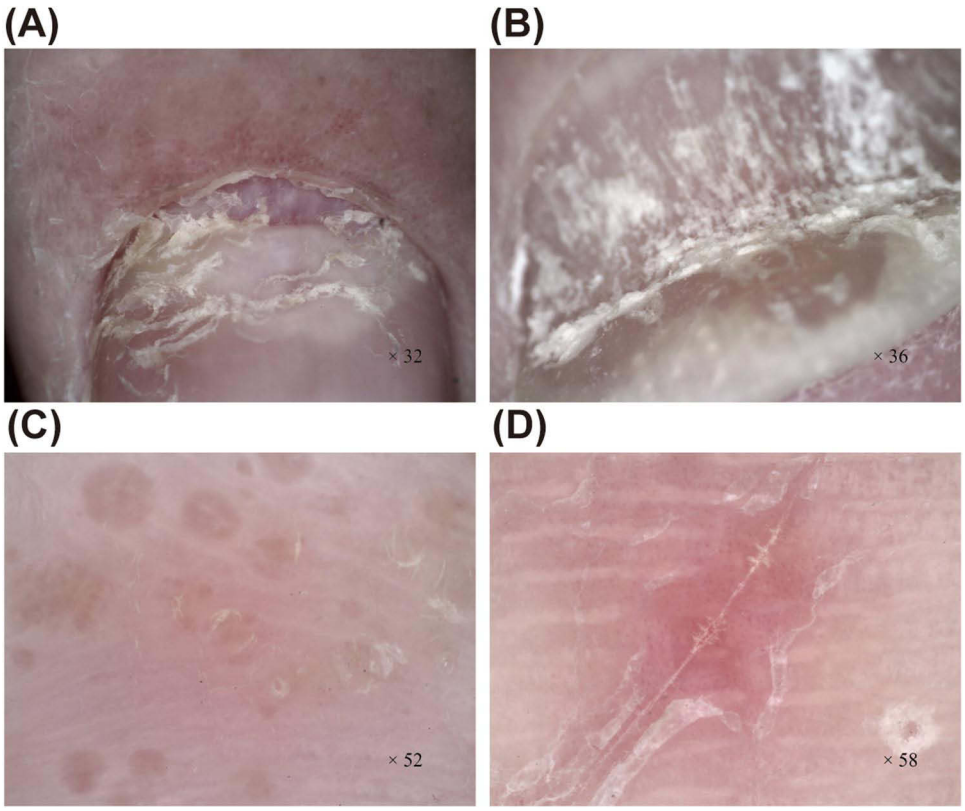


**Figure 2** Baseline dermoscopic images before treatment with abrocitinib. Dermoscopic images obtained before treatment showing the involvement of the fingernail (**A**), toenail (**B**), palm (**C**), and sole (**D**).

kIU/L). Based on clinical presentation and laboratory findings, onychomycosis, psoriasis, lichen planus, and other infectious or autoimmune disorders of nails were excluded. Baseline dermoscopic examination of the involved fingernails, toenails, palms, and soles was conducted, with the results suggesting an eczematous process (Figure 2A–D). Histopathological examination and patch testing were not performed. Considering the long-standing relapsing course, severe pruritus, substantial disease burden, and the atopic background, the patient was diagnosed with AD presenting predominantly with hand and foot eczema, complicated by eczema-associated nail dystrophy. Baseline disease assessment indicated high disease activity (a SCORAD score of 47, a peak pruritus numerical rating scale [PP-NRS] score of 9, and a sleep disturbance score of 9), reflecting marked inflammation and significantly impaired quality of life. These validated clinical indices were longitudinally assessed at multiple follow-up time points to quantitatively evaluate treatment response (Figure 3). Given the absence of a standardized, validated nail-specific scoring system for atopic dermatitis-related nail changes, nail changes were assessed based on serial clinical observations and photographic documentation during follow-up.

Given the chronic refractory course, inadequate response to conventional treatments, and involvement of acral skin and nails, areas known to respond poorly to topical treatment, systemic targeted therapy was initiated. The patient received oral abrocitinib 100 mg once daily, with no meaningful clinical improvement after four weeks. Severe pruritus of the hands and feet still persisted, accompanied by a marked thickening of the left middle and little fingernails (Figure 1B). Subsequently, the dose of abrocitinib was increased to 200 mg once daily, in accordance with standard clinical practice based on treatment response. Approximately two weeks after dose escalation, the patient reported marked relief of pruritus and substantial improvement in heel and plantar eczema, while nail lesions improved more slowly. At this stage, only minimal early changes were detected in toenail appearance, consistent with the relatively slow regenerative kinetics of the nail unit (Figure 1C). Both cutaneous and nail manifestations improved during follow-up. By month 4, hand and foot eczema had almost completely recovered, and nail and toenail morphology gradually improved, evidenced by reduced onycholysis and a smoother nail

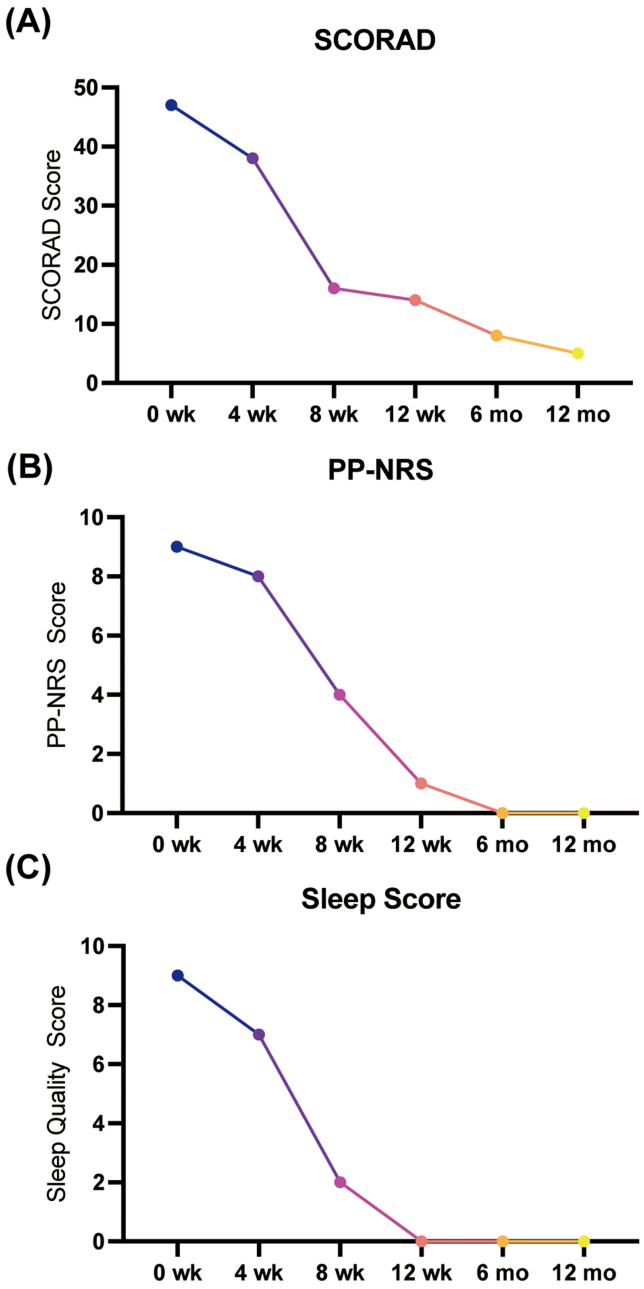


**Figure 3** Changes in clinical scores during treatment with abrocitinib. Serial line graphs showing the reduction in disease severity and improvement in quality of life over time. (**A**) SCORAD score; (**B**) PP-NRS pruritus score; (**C**) Sleep quality score. Follow-up time points: baseline (0 wk), 4 weeks, 8 weeks, 12 weeks, 6 months, and 12 months.

surface (Figure 1D). The dose of abrocitinib was subsequently tapered to 100 mg once daily for maintenance therapy. At the 12-month follow-up, complete remission of hand and foot eczema was maintained, and the fingernails and toenails showed marked structural recovery. Only mild residual thickening of the left great toenail was noted (Figure 1E). No adverse events were observed throughout the treatment and follow-up period. No concomitant systemic or topical anti-inflammatory therapies were administered during the treatment period, and abrocitinib was used as monotherapy, given the limited efficacy of prior conventional topical treatments and the intention to more clearly evaluate the therapeutic response to abrocitinib.


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## Discussion

Nail involvement in AD, although less common than cutaneous manifestations, has increasingly been linked to persistent inflammation and higher disease activity.[3] Chronic periungual inflammation and repeated mechanical stress may contribute to secondary nail dystrophy in patients with AD presenting predominantly with hand-foot eczema. This clinical manifestation is frequently overlooked in routine clinical practice, and standardized therapeutic strategies are currently lacking.[6,9] Conventional treatments, including topical corticosteroids, calcineurin inhibitors, keratolytics, and systemic immunosuppressants, often yield limited benefit when inflammatory processes involve the nail matrix.[10] With the development of targeted therapeutic agents, nail involvement in inflammatory dermatoses has received increasing attention. Previous reports have emphasized the complexity of treating nail involvement in atopic dermatitis. While dupilumab can improve eczematous nail changes in some patients, it may paradoxically induce new-onset nail dystrophy,[11] and this study indicates that periungual eczematous inflammation can lead to nonspecific nail matrix damage and impaired normal nail formation, and that targeted therapy may alter the local cytokine milieu in a complex manner, sometimes resulting in transient worsening followed by gradual improvemen.[11] In contrast, accumulating evidence suggests that JAK inhibitors may exert a more direct and consistent anti-inflammatory effect on nail involvement. For example, upadacitinib has been reported to improve hand eczema and associated nail dystrophy within 1–3 months, supporting the role of JAK/STAT pathway inhibition in regulating inflammation within the nail unit.[12] Recent reports further indicate that JAK1 inhibitors, including abrocitinib, may also be effective for other inflammatory nail diseases such as nail lichen planus, reinforcing the potential role of JAK1-targeted therapy in nail disorders.[13,14] However, clinical studies specifically assessing the use of abrocitinib for nail dystrophy associated with AD remain extremely limited.

In the present case, treatment with abrocitinib led to rapid improvement in pruritus and hand-foot eczema, followed by gradual recovery of nail morphology over the next months. This temporal sequence, consisting of early improvement in cutaneous symptoms preceding nail structural restoration, aligns with the relatively slow growth and regenerative dynamics of the nail unit. Based on the available literature, one plausible explanation is that abrocitinib first reduced JAK1-mediated inflammatory signaling within the skin and periungual tissues, leading to early relief of pruritus and eczema, whereas normalization of nail morphology required a longer interval because the previously inflamed and functionally disrupted nail matrix had to regenerate over time. In this context, the delayed improvement of the nail plate is more likely to reflect structural regrowth rather than delayed anti-inflammatory activity.[11,12] These observations suggest that sustained suppression of periungual inflammation and cytokine-mediated matrix injury may be critical for achieving structural nail repair. Notably, despite the well-recognized therapeutic challenges associated with acral and nail involvement, this patient exhibited substantial improvement in both fingernails and toenails. No treatment-related adverse events were observed, supporting the clinical value of abrocitinib in this context.

In conclusion, this case provides additional evidence supporting the role of selective JAK1 inhibitors in the management of AD-associated nail involvement. Consistent with previous reports of JAK1 inhibitors and in contrast to the paradoxical effects observed with dupilumab, our findings further support the potential role of selective JAK1 inhibition in AD-associated nail involvement. In addition, our case provides a longer longitudinal observation period, demonstrating that although cutaneous inflammation improved relatively early, visible restoration of nail structure continued over subsequent months and was sustained at 12 months. This temporal pattern further supports the concept that successful treatment of AD-associated nail dystrophy may depend not only on suppression of inflammation but also on sufficient time for nail unit regeneration. The limitations of this report include its single-case design and the absence of standardized nail-specific outcome measures. Nevertheless, this case highlights an underrecognized clinical phenotype of AD and suggests that abrocitinib may represent a valuable therapeutic option for selected patients suffering from atopic dermatitis–associated nail dystrophy. Future larger cohorts and standardized assessment tools are warranted to better define treatment efficacy and optimize management strategies for nail diseases subsequent to AD.

## Ethics Statement

Institutional approval was not required for publication of this case report in accordance with the policies of the Affiliated Hospital of Southwest Medical University. Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images.

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## Disclosure

The authors declare no conflicts of interest regarding this work.

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