

Improvement of Low-Dose Abrocitinib-Resistant Lichen Amyloidosis with Dupilumab: Two Case Reports

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Abstract: Lichen amyloidosis (LA), a form of primary localized cutaneous amyloidosis, is often refractory to conventional therapies. We report two LA patients who showed inadequate response to 100mg abrocitinib daily but achieved significant improvement with dupilumab. A 29-year-old woman and a 31-year-old man with chronic, pruritic LA lesions experienced rapid itch relief with abrocitinib but minimal skin improvement after 4–6 months, along with adverse effects, such as diarrhea, acneiform eruptions, and herpes simplex reactivation. After switching to dupilumab, both patients maintained pruritus control and exhibited marked lesion regression, with no side effects. While abrocitinib effectively alleviated itch, dupilumab demonstrated superior lesion resolution, possibly due to its dual inhibition of IL-4 and IL-13 signaling, which may more comprehensively address the underlying inflammation in LA. This is the first report of dupilumab's efficacy after low dose abrocitinib failure in LA, suggesting its potential as a therapeutic option for refractory cases. Further studies are needed to confirm these findings.

Keywords: lichen amyloidosis, cutaneous amyloidosis, dupilumab, JAK inhibitors, treatment

Introduction

Lichen amyloidosis (LA) is a form of primary localized cutaneous amyloidosis (PLCA) characterized by hyperkeratotic, pruritic papules, often localized to the shins, arms, or back. The pathogenesis of PLCA remains unclear, possibly involving environmental and genetic factors. Chronic irritation, itching and scratching can lead to epidermal damage and degradation of keratinocytes. Dermal macrophages and fibroblasts convert keratin peptide into amyloid fibrils, resulting in amyloid protein deposition within dermal papillae.¹ Interleukin –13 (IL-31), an itch-inducing cytokine secreted by multiple immune cell types,² has been increasingly implicated in the pathogenesis of PLCA. The increased epidermal expression of oncostatin M receptor (OSMR) and IL-31R in PLCA patients may also contribute to disease pathogenesis by enhancing cutaneous nerve sensitivity.³ A recent case report by Nakagawa et al (2025) also highlighted the efficacy of anti-IL-31 antibody in LA, reinforcing the role of IL-31.⁴

Despite various treatment modalities, including topical corticosteroids, oral antihistamines, cyclosporine, retinoic acid, surgery and as well as CO2 laser therapy, none of these treatments have shown satisfactory efficacy and all of them have shown high rates of recurrence.⁵

Dupilumab, a monoclonal antibody targeting IL-4 and IL-13 signaling, has demonstrated efficacy in pruritic dermatoses with a favorable safety profile.⁶ Several case reports have already documented its success in LA. Meanwhile, Janus kinase inhibitors (JAKis) have gained traction in dermatology due to their broad anti-inflammatory effects. Abrocitinib is an oral small-molecule JAK1 inhibitor used for treating moderate-to-severe atopic dermatitis (AD). While its primary use focuses on AD, emerging evidence and clinical reports suggest its efficacy in other dermatological conditions, including vitiligo, prurigo nodularis, and hand eczema.⁷ Its therapeutic potential in these disorders stems from

its selective inhibition of JAK1-mediated signaling, which plays a key role in inflammatory and immune-driven skin diseases.

We present two cases of LA patients who exhibited inadequate responses to low-dose abrocitinib but achieved marked clinical improvement after subsequently transitioning to dupilumab, underscoring the novel therapeutic sequence and its potential mechanistic implications.

Case Reports

This Retrospective Study Was Approved by the Ethics Committee of Peking University People's Hospital (2023PHB296-001)

Case 1

A 29-year-old woman with a 5-year history of pruritic papules on her forehead, occipital region, neck, back and chest (Figure 1A). Lesions had progressively worsened despite topical corticosteroids and oral antihistamines. She had a history of neurodermatitis. Physical examination revealed numerous brownish papules on the affected areas. Laboratory findings included normal IgE levels (11.63 IU/mL) and eosinophils (0.4%,0.15/mL). Dermoscopy revealed white scar-like centers with peripheral brown pigmentation. The Investigator's Global Assessment (IGA) score was 5. The Visual Analog Scale score (VAS) in pruritus was 7. Photography was performed using a smartphone (iPhone 13 Pro, Apple Inc, Cupertino, California, USA) set at a fixed distance from the patient's affected skin before each treatment session and at each follow-up visit. Abrocitinib (100 mg daily) was initiated. We followed up with her 2 weeks after the start of treatment and then every 4 weeks thereafter. By Week 2, her VAS score decreased to 2, but she developed diarrhea. At Week 4, VAS score further decreased to 1, yet acneiform eruptions emerged. Over six months, pruritus remained controlled, but skin lesions showed minimal improvement (Figure 1B) with an IGA score of 4. She also experienced persistent acne and menstrual irregularities. Due to suboptimal therapeutic response and adverse effects, abrocitinib treatment was discontinued. We subsequently performed local dermabrasion on the patient's upper back and then initiated dupilumab therapy with a 600 mg loading dose followed by 300 mg administered subcutaneously every two weeks. By week 4, folliculitis resolved, and pruritus remained minimal. At 6 months, lesions had significantly regressed (Figure 1C) and the IGA score was 2, with sustained symptom control and no adverse events.

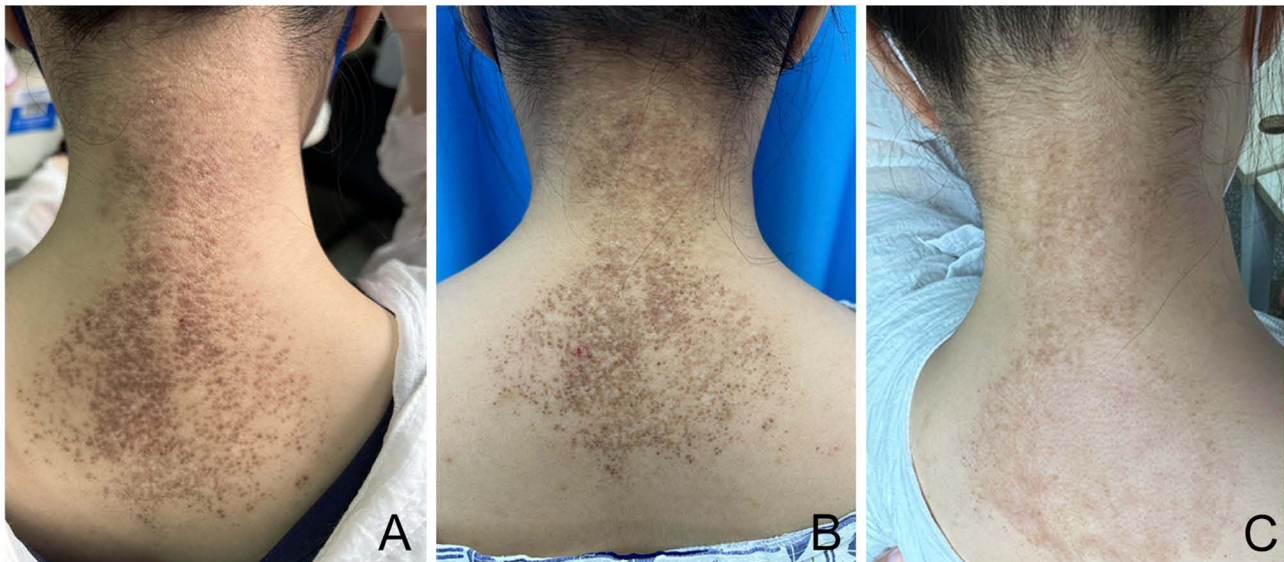


Figure 1 (A) Baseline lesions: multiple hyperpigmented, dark brown papules on the neck and back. (B) After 6 months of abrocitinib: minimal improvement in papular lesions with persistent hyperpigmentation. (C) After 6 months of dupilumab: near-complete resolution of papules, with residual light brown postinflammatory hyperpigmentation.

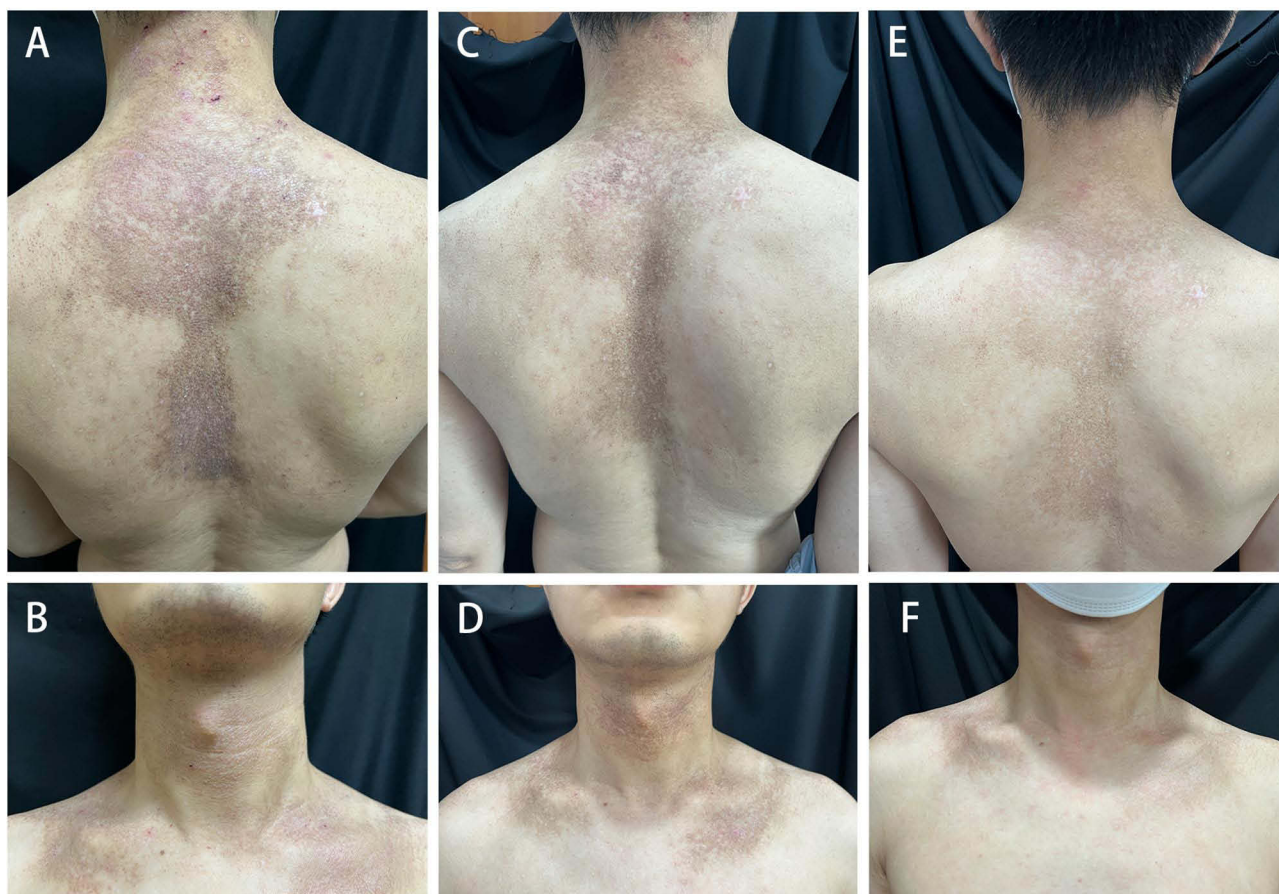


Figure 2 (A and B) Baseline lesions: numerous lichenified brown papules with erythema, excoriations, hemorrhagic crusts on the neck and back. **(C and D)** After 4 months of abrocitinib: reduced excoriations and erythema; lichenification partially persisted. **(E and F)**. After 4 months of dupilumab: flattening of papules and marked reduction in lichenification, with residual faint brown hyperpigmentation.

Case 2

A 31-year-old man presented with an 8-year history of refractory pruritic papules on his neck, chest and back (**Figure 2A and B**), unresponsive to topical corticosteroids. Physical examination revealed numerous brownish papules, lichenification, and crusting on his back. Laboratory tests showed elevated IgE levels (158 IU/mL) and eosinophils at 3.5%. A prior biopsy confirmed cutaneous amyloidosis. His baseline IGA score was 5 and VAS score was 5. We followed up with him 2 weeks after the start of treatment and then every 4 weeks thereafter. Abrocitinib (100 mg daily) reduced his VAS score to 2 by week 2. However, he contracted COVID-19 at 4 weeks. By 4 months, lichenification persisted and the IGA score was 4, with herpes simplex reactivation (**Figure 2C and D**). Due to inadequate lesion response and herpes reactivation, he was transitioned to dupilumab therapy. By 4 months, he had achieved sustained pruritus relief and significant lesion resolution, with an IGA score of 2. Residual hyperpigmentation was present (**Figure 2E and F**).

Discussion

In both presented cases, patients experienced rapid pruritus relief within two weeks of low-dose abrocitinib (100 mg daily) initiation, which was sustained throughout treatment. However, no significant improvement in cutaneous lesions was observed after 4–6 months of therapy, and its use was associated with adverse events such as diarrhea, acneiform eruptions, and herpes simplex reactivation. Following transition to dupilumab, patients not only maintained itch control and demonstrated marked lesion resolution, but also exhibited a favorable safety profile with no side effects reported. Notably, in Case 1, the patient underwent local dermabrasion on the upper back before starting dupilumab. While this

constitutes a confounding factor, improvement was also observed in non-dermabraded areas, supporting a possible systemic effect of dupilumab. Nonetheless, this limitation should be acknowledged.

While multiple case reports have demonstrated dupilumab's efficacy in LA,^{8,9} evidence for JAKi remains limited to isolated reports of improvement with baricitinib and tofacitinib.^{10,11} Dupilumab, a monoclonal antibody that inhibits IL-4 and IL-13 signaling via blockade of the IL-4 receptor α subunit, demonstrates high efficacy in reducing inflammatory lesions and pruritus, with a well-established long-term safety profile that necessitates no routine laboratory monitoring. Its most frequently observed adverse events include conjunctivitis and injection-site reactions. In contrast, JAKis, such as abrocitinib and upadacitinib, are oral small molecules that provide a broader suppression of cytokine signaling via the JAK-STAT pathway (particularly JAK1).¹² This broader mechanism translates to a notably rapid onset of action, with some head-to-head trials suggesting superior short-term efficacy in itch relief compared to dupilumab.¹³ However, this class carries a boxed warning for serious infections, thrombosis, malignancy, and major adverse cardiovascular events, mandating baseline and periodic laboratory monitoring.

In the two LA patients, 100 mg abrocitinib treatment provided rapid and sustained pruritus relief but led to insignificant rash improvement over 4–6 months, with mild adverse events observed during this period. In contrast, subsequent transition to dupilumab maintained itch control while achieving significant lesion resolution, also with a favorable safety profile. This differential response may suggest that while low-dose abrocitinib may be sufficient for symptomatic pruritus management, it appears inadequate for achieving meaningful lesion resolution in LA. Dupilumab's superior lesion resolution in LA compared to abrocitinib's more rapid antipruritic effects may be explained by either abrocitinib's incomplete blockade of IL-31 signaling pathways or dupilumab's additional mechanisms beyond IL-4/IL-13 inhibition.

To our knowledge, this is the first report of successful dupilumab use after low-dose abrocitinib failure in LA. Our findings may provide novel insights into both the pathogenesis and therapeutic approaches for LA.

Conclusion

Dupilumab demonstrated sustained pruritus control and significant lesion improvement in two LA patients refractory to low-dose abrocitinib, with a favorable safety profile. Larger controlled studies are warranted to validate these observations and establish dupilumab as a therapeutic option for refractory LA.

Ethics Approval

Reviewed and approved by the Ethics Committee of Peking University People's Hospital (2023PHB296-001). Institutional approval was not required to publish the case details. Written consent was obtained from both patients for publication.

Funding

This work was supported by the National Natural Science Foundation of China (No. 82373504 and 82103750).

Disclosure

The authors report no conflicts of interest in this work.

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