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Dual IL-17A/F Blockade for Acrodermatitis Continua of Hallopeau: A Clinical Response to Bimekizumab

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ABSTRACT

Acrodermatitis continua of Hallopeau (ACH) is a rare, localized variant of pustular psoriasis that primarily affects the distal digits and nail apparatus, often presenting with recurrent pustules, nail dystrophy, and significant functional impairment. Due to its rarity and chronic relapsing course, ACH is notoriously difficult to treat, and standardized treatment guidelines are lacking. We present the case of a 67-year-old male with ACH who failed multiple therapies, including corticosteroids, topical tapinarof, and oral deucravacitinib, before achieving rapid and sustained improvement with bimekizumab, a monoclonal antibody targeting interleukin-17A and IL-17F. Within one month of initiating bimekizumab, the patient experienced marked clinical improvement in both skin lesions and joint pain, with continued progress allowing him to return to work. This case highlights the potential utility of dual IL-17A/F inhibition in neutrophil-dominant pustular conditions such as ACH. As more case reports document favorable outcomes, bimekizumab may emerge as a valuable treatment option for patients with refractory ACH, offering targeted cytokine blockade in a condition with few effective therapies.

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INTRODUCTION

Acrodermatitis continua of Hallopeau (ACH) is a rare, localized variant of pustular psoriasis. Fewer than 200 cases have been reported in the literature. It is characterized by recurrent, tender, sterile pustules with underlying erythema affecting the distal digits, typically with consistent involvement of the nail apparatus.[1] Complications can include onychodystrophy leading to anonychia, as well as osteolysis of the distal phalanges.[1] The condition follows a chronic, relapsing course and is often recalcitrant to conventional therapy. Management is particularly challenging due to the lack of standardized treatment guidelines, largely stemming from the rarity of ACH.

While several case reports have demonstrated success with therapies commonly used in plaque psoriasis,[1] an increasing number of cases have reported promising responses to bimekizumab.[2-4] Here, we present a case of a 67-year-old male with ACH who responded to bimekizumab after failing treatment with deucravacitinib. This case adds to the growing body of evidence supporting bimekizumab,a dual IL-17A and IL-17F inhibitor, as a therapeutic option for ACH.

CASE PRESENTATION

A 67-year-old male presented in 2024 with a several-month history of pain, inflammation, and a rash involving the distal digits of both hands and his left foot. He reported progressive difficulty performing manual work as a motorcycle mechanic due to swelling and joint discomfort localized to the fingertips. Initial treatment with doxycycline, mupirocin, and oral terbinafine was ineffective.

His past medical history was significant for type 2 diabetes mellitus and hypertension. Current medications included dapagliflozin, pantoprazole, lisinopril, and extended-release saxagliptin/metformin.

On physical examination, several fingers and the lateral toes of the left foot exhibited erythematous, scaling plaques with nail dystrophy and subungual pustules. Nail involvement was prominent, with multiple fingernails and the lateral two toenails of the left foot affected.

A shave biopsy of an active lesion on his left index finger was performed, and laboratory testing, including a comprehensive

FIGURE 1. Baseline presentation of right thumb following treatment failure with deucravacitinib and topical tapinarof. Note coalescent, subungual pustules, nail dystrophy, and surrounding erythema.

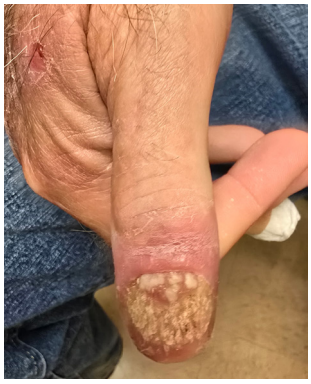
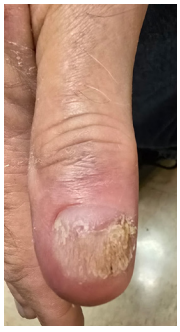


FIGURE 2. Right thumb one month after initiation of bimekizumab. Marked resolution of erythema and clearance of pustules are observed, with residual nail dystrophy remaining.



metabolic panel, lipid panel, and tuberculosis test, was performed and found to be unremarkable. The patient was started empirically on a prednisone taper.

Histopathologic examination revealed subcorneal and intracorneal aggregates of neutrophils with parakeratosis, absence of the granular layer, and spongiosis. The findings were consistent with pustular psoriasis. Special stains, including PAS, GMS, HSV I/II, and *T. pallidum*, were all negative.

Despite partial symptomatic improvement with corticosteroids, symptoms quickly recurred following completion of the steroid taper. A trial of topical tapinarof and oral deucravacitinib was initiated. At follow-up one month later, the patient reported no perceived improvement and continued to experience functional impairment. He continued to remain on his trial of topical tapinarof and deucravacitinib at that time.

In the setting of worsening symptoms, failure of both topical and oral therapies, and prominent subungual pustulosis (Figure 1), the patient was transitioned from deucravacitinib to bimekizumab, a monoclonal antibody targeting interleukin-17A and 17F. The patient continued to use topical tapinarof as adjunctive therapy.

At one-month follow-up, the patient reported marked improvement in both his skin (Figure 2) and joint pain. After two months of therapy, he reported significant functional gains and had resumed work as a mechanic. Continued clinical improvement was noted through subsequent visits, and the patient successfully transitioned to the maintenance dosing phase of bimekizumab (every 8 weeks) after 16 weeks of therapy.

DISCUSSION

Acrodermatitis continua of Hallopeau (ACH) is rare and notoriously difficult to treat, largely due to its chronic relapsing nature and the absence of standardized treatment guidelines. Historically, therapies have been adapted from treatment regimens for other forms of psoriasis, including topical and systemic corticosteroids, vitamin D analogs, fluorouracil, calcineurin inhibitors, coal tar preparations, systemic retinoids, cyclosporine, methotrexate, and phototherapy.[1] Similarly, in our case, the patient was initially managed with corticosteroids, topical tapinarof, and deucravacitinib, none of which resulted in sustained clinical improvement. These negative outcomes are consistent with prior reports of therapeutic resistance in ACH and underscore the pressing need for more effective, targeted options.

In ACH, dysregulation of IL-36, Th17 cells, neutrophils, and keratinocytes leads to elevated IL-17 cytokine levels, which in turn drive neutrophil recruitment.[2] Consequently, IL-17-targeted therapies have emerged as promising options in the management of pustular psoriasis subtypes, including ACH. Bimekizumab, a monoclonal antibody that inhibits both IL-17A and IL-17F, may offer superior efficacy compared to IL-17A inhibition alone. Our patient's rapid improvement after transitioning to bimekizumab supports the notion that dual cytokine blockade may be particularly effective in neutrophil-dominant disease presentations such as ACH. Experimental studies have demonstrated its ability to normalize IL-36 expression, a key inflammatory driver in pustular psoriasis, to levels seen in non-lesional skin.[4] These findings provide a biologic rationale for the growing number of case reports supporting bimekizumab as a therapeutic option in pustular variants of psoriasis.

As biologics have become increasingly central in the treatment of psoriasis and its subtypes, attention has shifted toward their role in managing ACH. A review by Wang et al of published pustular psoriasis cases found that anti-TNF-α agents were the most frequently reported, with IL-12/23 and IL-17A inhibitors also demonstrating efficacy.[5] Notably, our case is among the few to describe successful treatment of ACH with bimekizumab, a dual IL-17A/F inhibitor that was not included in that review. This gap

highlights the need for continued reporting of successful cases, particularly those involving biologics with broader cytokine blockade.

Our case adds to this emerging literature, demonstrating rapid and sustained improvement of ACH with bimekizumab in a patient who failed both corticosteroids and deucravacitinib. It further supports the expanding role of dual IL-17A/F inhibition as a viable treatment strategy for refractory cases of ACH.

CONCLUSION

This case contributes to the expanding evidence base supporting bimekizumab as a treatment for ACH. As our understanding of pustular psoriasis pathophysiology deepens, targeted biologics with broader cytokine inhibition may offer new hope for patients with treatment-resistant forms of the disease. Ongoing documentation and investigation will be crucial in guiding future therapeutic strategies for this rare and challenging condition.

DISCLOSURES

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