Dermatology Reports
https://journals.pagepress.net/dr

eISSN 2036-7406

Please cite this article as:

Giorgio CM, Balato A, Argenziano G, et al. Tildrakizumab in the treatment of plaque psoriasis in an HIV+ patient: a case report and literature review of anti-interleukin drugs. *Dermatol Rep 2026 [Epub Ahead of Print] doi: 10.4081/dr.2026.10666*

Received: 26 October 2025; Accepted: 7 June 2026.

Complete remission of keratosis lichenoides chronica with abrocitinib

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Key words: abrocitinib; Nekam's disease; management; keratosis lichenoides chronica.

Contributions: Caterina Mariarosaria Giorgio, Anna Balato, Giuseppe Argenziano: conception and design of study; Caterina Mariarosaria Giorgio, Anna Balato, Paolino Franzese, Elisabetta Fulgione, Maria Maddalena Nicoletti, Gaetano Licata: acquisition of data; Caterina Maria Rosaria Giorgio, Anna Balato, Gaetano Licata: analysis and/or interpretation of data, drafting the manuscript. All authors revised the manuscript critically for the validity of scientific content and certify that they have participated sufficiently in the work to take public responsibility for the content.

Conflict of interest: the authors have no conflict of interest to declare.

Ethics approval and consent to participate: not required.

Consent for publication: the patient in this manuscript has given written informed consent to publication of her case details and any accompanying images.

Availability of data and materials: all data are included in this published article.

Funding: none.

Dear Editor,

Keratosis lichenoides chronica (KLC), also known as Nekam's disease, is a rare and recalcitrant lichenoid dermatosis. It manifests with keratotic violaceous papules arranged in linear or reticulated patterns and typically follows a chronic and disabling course. Therapeutic guidelines are lacking, and conventional treatments – including corticosteroids, retinoids, immunosuppressants, and phototherapy – often fail to provide sustained benefit.[1] Given its rarity, each therapeutic observation carries significant relevance. We describe the case of a 41-year-old woman with a 6-year history of persistent violaceous papules on both pretibial regions. Multiple therapies, including oral cyclosporine administered continuously for 12 months, intralesional triamcinolone injections every 3 weeks for approximately 6 months, and several cycles of systemic and topical corticosteroids as well as acitretin. None of these approaches resulted in clinical remission or even partial improvement. The chronicity of the lesions and the absence of even partial or transient regression underscore the resistance of this patient's disease to established therapies. Clinical examination showed hyperkeratotic papules arranged in cord-like patterns. Dermoscopy displayed thick white-to-yellow hyperkeratotic scales, follicular plugging with keratotic collars, and a distinctive reticulated and linear arrangement over a violaceous background (Figure 1).[2] Histopathological examination of an incisional biopsy confirmed epidermal hyperplasia with marked hyperkeratosis, focal basal vacuolar alteration, superficial dermal fibrosis with congested capillaries, scattered lymphomononuclear infiltrate, melanophages, and small milium-like cysts, consistent with the diagnosis of keratosis lichenoides striata, a localized variant of KLC. Given disease severity and refractoriness, we initiated off-label therapy with abrocitinib, an oral selective Janus kinase (JAK)1 inhibitor currently approved for moderate-to-severe atopic dermatitis, at 100 mg daily. The rationale was based on the JAK-signal transducer and activator of transcription (STAT) pathway involvement in lichenoid inflammation. Cytokines such as interferon-γ, interleukin-6, and interleukin-15 rely on JAK1-dependent signaling. Abrocitinib, by selectively blocking JAK1, interrupts these proinflammatory pathways while sparing other kinases, theoretically reducing both the intensity of inflammation and the long-term burden of hyperkeratosis. Recent evidence has also documented the efficacy of another JAK1 inhibitor, upadacitinib, in the treatment of KLC.[3] The expanding body of evidence on JAK inhibitors in cutaneous autoimmune and inflammatory disorders, including lichen planus and dermatomyositis, provided further support for this off-label intervention.[4] After one month of therapy with abrocitinib 100 mg daily, a striking flattening of the hyperkeratotic cords was observed, accompanied by a rapid decline in pruritus (Figure 2 A-C). At 6 months, the patient achieved complete and sustained remission, with the disappearance of dermoscopic hyperkeratotic ridges and background violaceous

pigmentation (Figure 2 B-D). No adverse events occurred, and serial laboratory tests remained normal. While spontaneous fluctuations have occasionally been described in KLC, the long-standing persistence of this patient's lesions and the documented inefficacy of multiple systemic and local treatments render spontaneous resolution improbable. The correlation between clinical improvement and dermoscopic normalization further supports a true therapeutic effect of abrocitinib. Although this observation cannot establish generalizable efficacy, it provides important proof-of-concept for JAK1 inhibition in KLC. Potential criticisms may concern the safety of long-term JAK inhibition and the off-label nature of the treatment. In our case, rigorous monitoring revealed no safety signals, but vigilance remains mandatory in view of potential hematologic, metabolic, or thromboembolic risks described with JAK inhibitors. Regarding the off-label indication, the exceptional refractoriness of the disease and the absence of approved alternatives justify the therapeutic attempt under careful supervision. In conclusion, this case represents, to our knowledge, the first documentation of complete and sustained remission of KLC with abrocitinib. The convergence of clinical, dermoscopic, and histopathological findings highlights both diagnostic precision and the utility of dermoscopy in therapeutic monitoring. More broadly, the case suggests that selective JAK1 blockade may interrupt the inflammatory circuitry underlying this rare and devastating dermatosis, offering a paradigm shift for patients previously considered untreatable. Future prospective studies are warranted to determine whether abrocitinib and related agents could inaugurate a new era in the management of Nekam's disease.

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Figure 1. A, **B**) Clinical presentation before treatment: erythematous-violaceous infiltrated plaques on the lower leg with arcuate/annular configurations, superficial scaling, and excoriations. **C**, **D**) Dermoscopy before abrocitinib: diffuse erythematous background, fine whitish scales, dotted and irregular linear vessels, and focal erosions.

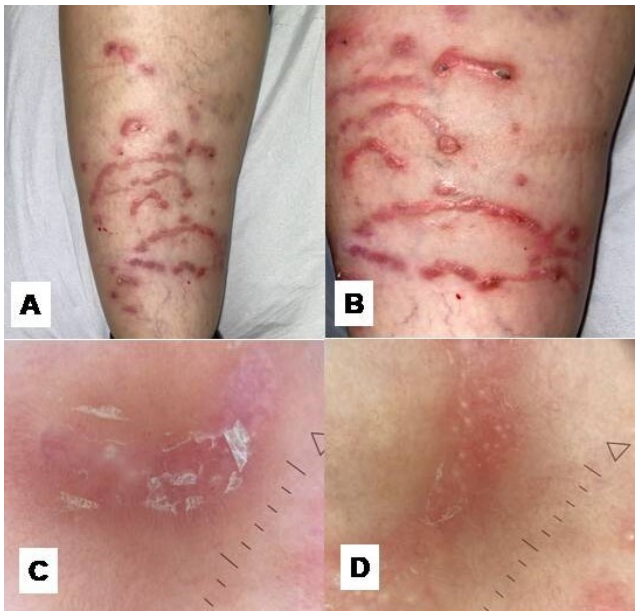

Figure 2. A) Partial clinical resolution after one month of abrocitinib. **B**) Complete clinical resolution with residual post-inflammatory hyperpigmentation after 6 months. **C**) Dermoscopic improvement after one month, with reduced erythema and vessels. **D**) Complete dermoscopic resolution after 6 months.