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CASE REPORT

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Rapid Clearance of Lichen Planus With Topical Tapinarof

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ABSTRACT

Background: Lichen planus (LP) is an auto-inflammatory idiopathic condition affecting skin, mucous membranes, hair follicles, and nails that presents as pruritic, violaceous papules and plaques. No FDA-approved treatments exist for LP.

Case Presentation: A 70-year-old female presented with scalp pruritus and pruritic eruption on bilateral shins. She was treated for presumed seborrheic and contact dermatitis; however, after 2 months, she had no improvement. On biopsy, scalp and right shin samples were consistent with lichen planopilaris and LP, respectively. The patient was treated with hydroxychloroquine, pimecrolimus, fluocinonide, and tofacitinib 2% creams, and intralesional Kenalog. Four months later, she had complete resolution of the scalp rash, but the rash on the shins persisted. We discontinued tofacitinib, fluocinonide, and pimecrolimus and prescribed tapinarof 1% cream once daily. After 1 month, her scalp condition remained in remission, and the LP on the shins completely resolved with minimal post-inflammatory hyperpigmentation.

Conclusion: To our knowledge, this is the first report in the literature showing successful treatment of LP with tapinarof. Further studies are required to determine if tapinarof is beneficial for the treatment of lichenoid diseases such as LP, lichen planopilaris, and others.

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INTRODUCTION

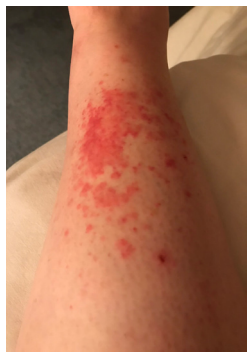
Lichen Planus (LP) is an auto-inflammatory idiopathic condition with unclear etiology, believed to be an auto-reactive response of T-cell-mediated damage after exposure to an inciting antigen. LP presents as pruritic, violaceous papules and plaques most commonly on the volar wrists, flexural surfaces, and trunk. Histologically, LP is characterized by a band-like lichenoid lymphocytic infiltrate with vacuolar destruction of the basal layer.[1] LP has no FDA-approved treatments. Typical off-label treatments are topical and systemic steroids, anti-malarials, retinoids, antibiotics, immunosuppressants, phototherapy, and recently, apremilast and oral and topical Janus kinase inhibitors.[1,2] We present the case of a 70-year-old female with a history of LP and lichen planopilaris who experienced complete resolution of LP after using tapinarof 1% topical cream.

Case Presentation

A 70-year-old female presented to the clinic with complaints of scalp pruritus and pruritic eruption on bilateral shins lasting several weeks. Her medical history was significant for acid reflux, for which she was taking famotidine.

On examination, we observed erythema and subtle perifollicular scaling at the scalp, as well as erythematous and cobble-stoning flat-topped papules coalescing into plaques on the bilateral shins. She was treated for presumed seborrheic dermatitis with ketoconazole shampoo and clobetasol solution. Fluocinonide cream was prescribed for the shins.

FIGURE 1. Initial presentation with erythematous and cobble-stoning flat-topped papules coalescing into plaques extended over the shin.



She returned after 2 months, reporting no improvement in either condition. Two biopsies were performed. The biopsy of the right temporal scalp showed hyperkeratosis of the epidermis and fibrosis with chronic inflammation centered around the hair follicles in the dermis. The biopsy of the right shin showed hyperkeratosis and hypergranulosis with a band-like lymphocytic infiltrate. These findings were consistent with lichen planopilaris and LP, respectively.

The patient was started on hydroxychloroquine 200 mg twice daily. Hepatitis C was negative. CBC and CMP were unremarkable. She denied mucus membrane or nail involvement. The scalp was treated with intralesional Kenalog 2.5 mg/mL. Pimecrolimus and compounded tofacitinib 2% creams were added.

FIGURE 2. Scalp biopsy site of erythema and subtle perifollicular scaling.

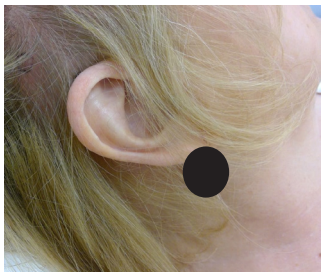
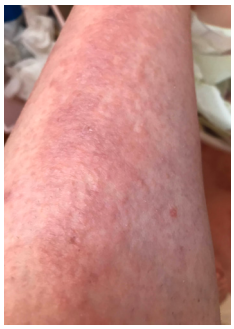


FIGURE 3. Erythematous plaques and cobble-stoning flat-topped papules on the shin at the time of the skin biopsy.



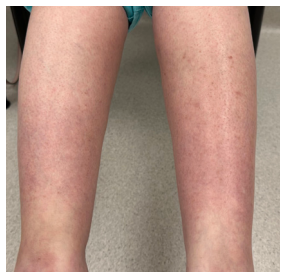
The patient returned 4-months later with complete resolution of the scalp rash; however, the rash on the shins persisted. We discontinued tofacitinib, fluocinonide, and pimecrolimus. She was given tapinarof 1% cream to use once daily. She returned 1 month later with complete resolution of her LP on the shins with minimal post-inflammatory hyperpigmentation. The scalp continued in remission without any topical treatments.

DISCUSSION

LP is a prototypical disorder of lichenoid dermatoses, such as lichen planopilaris, pityriasis lichenoides, lichen striatus, etc. It is an auto-reactive condition, believed to result from CD8+ cytotoxic T-cell-mediated reactions against basal keratinocytes, further influenced by T helper (Th) pathways, such as Th1 and Th17/interleukin (IL)-23 axis.3 LP is thought to be a response to foreign antigens such as infections (hepatitis C, human herpesviruses, COVID-19), vaccinations, contact allergens (dental metals), and medications (ACE-inhibitors, beta blockers).[1,3,4] LP is classically described as purple, planar, pruritic papules and plaques with Wickham striae in some lesions.

Several alterations in the expression of cytokines and chemokines in lesions or serum of patients with LP have been described. Serum concentrations of IL-5, IL-6, IL-8, IL-9, IL-10, IL-12, IL-17, IL-22, TNF-α, transforming growth factor-β, and interferon (IFN)-γ have been found to be elevated.[1] Increased expression

FIGURE 4. Interim clearance of lichen planus on regimen daily tapinarof 1% cream over a 1-month period.



of IFN-γ and IL-17 in LP lesions has also been described.[1] The lesions also have an increased number of mast cells, indicating that non-specific mechanisms such as mast cell degranulation and protease activation are involved in LP pathogenesis. These mechanisms may combine to cause T-cell accumulation and induce keratinocyte apoptosis.[1]

In addition, analysis of the relationship between nitrosative stress, antioxidant defense, and inflammation offers insights into the role of the nitric oxide pathway in LP pathogenesis. Increased concentrations of nitrosative stress markers, such as nitrates and nitrites, in patients with LP were positively correlated with concentrations of C-reactive protein and negatively correlated with markers of total antioxidant status, indicative of pro-inflammatory status and impaired antioxidant defense in LP.[5]

Most off-label treatments used for LP give inconsistent results. We found the use of tapinarof 1% cream led to rapid and complete clearance of LP. Tapinarof is a small-molecule therapeutic agent that uniquely activates the aryl hydrocarbon receptor (AhR) pathway to decrease oxidative stress, re-establish skin homeostasis, and decrease multiple pro-inflammatory cytokines, including several cytokines relevant in LP pathogenesis (IL-17, IL-22, IFN-γ, IL-5, IL-6, IL-8).[6,7]

AhR is a transcription factor ubiquitously expressed by epithelial and immune cell types,[8] including keratinocytes, antigen-presenting cells, Langerhans cells, mast cells, T and B cells.[7,9] AhR is believed to increase antioxidant activity via the nuclear factor erythroid 2-related factor 2 pathways, thereby decreasing oxidative stress in the skin.[9] AhR exerts numerous effects on mast cells, B cells, macrophages, antigen-presenting cells, Th1/Th2 cell balance, Th17 cells, and regulatory T cells.[10] Notably, Th17 cells express AhR at high concentrations. AhR downregulates Th17 cell differentiation as well as IL-17 and IL-22 production.[6] AhR also suppresses Th2 cell differentiation and the production of IL-4, IL-5, IL-13, and IL-31 cytokines.[7,8] AhR regulates inflammatory signals via crosstalk with other signaling pathways, such as the nuclear factor kappa B pathway,[6,7] which

controls the expressions of IL-1β, IL-6, IL-8, TNF-α, and other inflammatory genes.[6] Given involved cytokine pathways, AhR may be implicated in the modulation of the normal differentiation of Th1/Th2 cells, which explains tapinarof's efficacy in atopic dermatitis, psoriasis, and, in this case, LP. More studies are needed to evaluate tapinarof's efficacy for lichenoid dermatoses such as LP.

CONCLUSION

Our patient experienced complete remission of LP within one month of initiating treatment with topical tapinarof. To our knowledge, this is the first case report in the literature showing successful treatment of LP with tapinarof.The unique mechanism of action of tapiranof, its modulation of cytokine pathways and oxidative stress reduction, may explain its effectiveness in Th1 and Th2 predominant conditions, such as atopic dermatitis, psoriasis, and, in this case, LP.

This case report highlights the potential role tapinarof may play in the treatment of LP and other lichenoid diseases, such as lichen planopilaris, pityriasis lichenoides chronica, etc.

DISCLOSURES

A medical writing assistance grant was provided by Organon. Authors do not have any other conflicts of interest to disclose.

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