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Successful Treatment of Lichen Amyloidosis Using a Fixed Combination of Halobetasol-Propionate and Tazarotene Lotion

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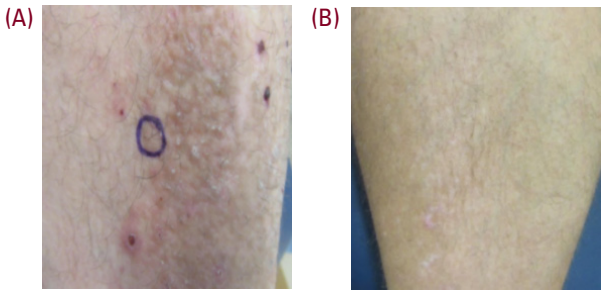
INTRODUCTION

Lichen amyloidosis is characterized by "rippled", brown, extremely pruritic plaques, typically on the lower extremities, that is often refractory to medical intervention. Here, a case of lichen amyloidosis successfully treated with a fixed combination of halobetasol-propionate 0.1% and tazarotene 0.45% lotion is presented.

A 53-year-old male with a relevant medical history for eczema presented with a pruritic rash on his left shin for several years recalcitrant to several topical corticosteroids including triamcinolone, clobetasol, and halcinonide ointments.

On physical exam, there was an ill-defined, dark brown, plaque composed of firm scaly papules on his left pretibial area (Figure 1).

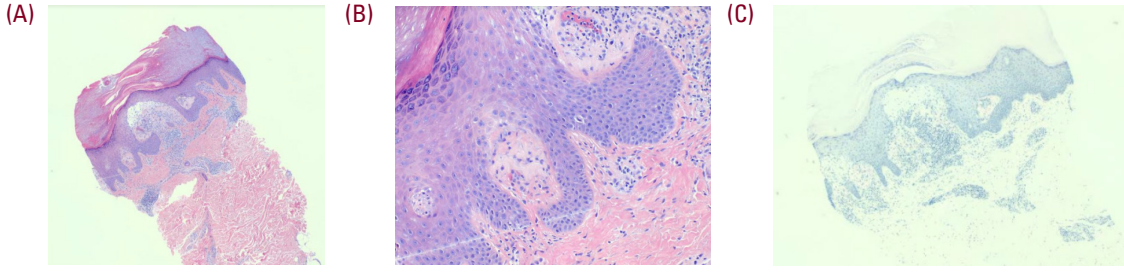
FIGURE 1. (A) Eroded pink to brown papules coalescing into a plaque on the left pretibial area. (B) Faint macular hyperpigmentation on the left pretibial area six months following initial presentation



A 3 mm punch biopsy from the affected area revealed hyperkeratosis, epidermal acanthosis, and amyloid deposits at the epidermal-dermal junction identified with Congo red stain, all consistent with lichen amyloidosis. Given the ineffectiveness of past topical corticosteroids, and the patient's inability to undergo UVB therapy due to the COVID-19 pandemic, the patient was prescribed a fixed combination of halobetasol-tazerotene lotion. Within 2 weeks of use, the patient reported substantial improvement, and returned approximately 2 months after initiating therapy. A 3 mm punch biopsy from the affected area revealed hyperkeratosis, epidermal acanthosis (Figure 2 A and B), and amyloid deposits at the epidermal-dermal junction identied with Congo red stain (Figure 2C).

Lichen amyloidosis, a form of localized cutaneous amyloidosis, is caused by keratinocyte derived amyloid deposition in the superficial dermis. An uncontrolled inflammatory response likely plays a role in the pathogenesis of this disorder. Sweat gland dysfunction may lead to leakage of sweat into the epidermal/dermal junction, which triggers inflammation, causing keratinocyte apoptosis, and amyloid formation.[1] Alternative theories suggest that cytokeratin, released from apoptotic basal keratinocytes, are phagocytized by macrophages leading to amyloid formation.[2] Given the role of immunoregulatory proteins in macrophage recruitment and migration, suppression of such proteins with topical corticosteroids could consequently decrease amyloid formation.

FIGURE 2. Lichen Amyloidosis. (A and B) Histopathology demonstrated marked hyperkeratosis, epidermal acanthosis, a superficial lymphocytic dermal infiltrate and pink amorphous globules in the superficial dermis (H&E, original magnification A. x 20 and B. x 100). (C) Amyloid deposits were confirmed with Congo red staining (original magnification x 20)



In considering retinoids in this setting, their role in the management of hyperkeratotic and hyperproliferative disorders has been well recorded. Furthermore, retinoids' anti-inflammatory potential could help augment the effect of topical corticosteroids. Retinoids may reduce inflammation through the inhibition of immunoregulatory transcription factors, proinflammatory cytokines, and leukocyte recruitment.[3] An improved anti-inflammatory response could reduce keratinocyte apoptosis and amyloid formation.

Currently, limited evidence exists supporting a definitive regimen in the management of lichen amyloidosis. Randomized controlled trials are needed to identify the appropriate treatment approach.[4] The case presented suggests that a fixed combination of halobetasol-tazerotene therapy could be an effective off-label treatment in the management of lichen amyloidosis, as well as be an acceptable candidate for future trials. The synergistic effects of a topical corticosteroid and a topical retinoid reduce inflammation, and thus, decrease amyloid production. Furthermore, the addition of a retinoid reduces hyperkeratosis and epidermal acanthosis. Future well-controlled clinical studies would be needed to fully appreciate the potential.

DISCLOSURES

The authors declare that there is no conflict of interest.

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