

SKINimages

Hypertrophic Lichen Planus Due to Koebnerization After Percutaneous Vascular Procedure

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A 65-year-old male with a history of cutaneous lichen planus (LP) over 30 years prior presented 1 month post percutaneous vascular procedure with pruritic lesions on the left lower leg. On examination, lesions were hyperpigmented, hypertrophic violaceous papules localized at the sites of the percutaneous access points (**Figure 1**). Biopsy of a lesion on the lower extremity revealed hyperkeratosis, hypergranulosis, thickening of the spinous cell layer, and a dense, band-like infiltrate of lymphoid cells with melanophages at the dermal-epidermal interface. Basal cell vacuolopathy, colloid bodies, melanin pigment incontinence, and a hypertrophic epidermis were also noted (**Figure 2**). The patient was diagnosed with hypertrophic lichen planus (HLP) triggered by his recent vascular access procedure. The patient was treated with a combination betamethasone and tazarotene cream once daily with resolution after 2 months of application.

This case represents a unique presentation of HLP resulting after a percutaneous vascular access procedure via koebnerization. LP is an inflammatory papulosquamous disorder classically characterized by pruritic, polygonal, violaceous, papules or plaques on the skin associated with a superficial white reticular pattern (i.e. Wickham striae).^{1,2} Other less common morphologic variants of LP include, but are not limited to, the annular, atrophic, ulcerative, bullous, erythrodermic, and hypertrophic forms.² As such, it is important for practitioners to retain a high degree of clinical suspicion for this disease process. Its pathogenesis is not completely understood but is thought to be due to an immune mediated autonomic response to antigenic triggers.^{1,2} Hypertrophic lichen planus (HLP), also referred to as lichen planus verrucous or hyperkeratosis, presents as firm, pruritic, discolored papules, plaques, or nodules on an erythematous base which may merge and appear as a verrucous or hyperkeratotic

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Figure 1. Verrucous polygonal violaceous plaques at sites of percutaneous vascular access.

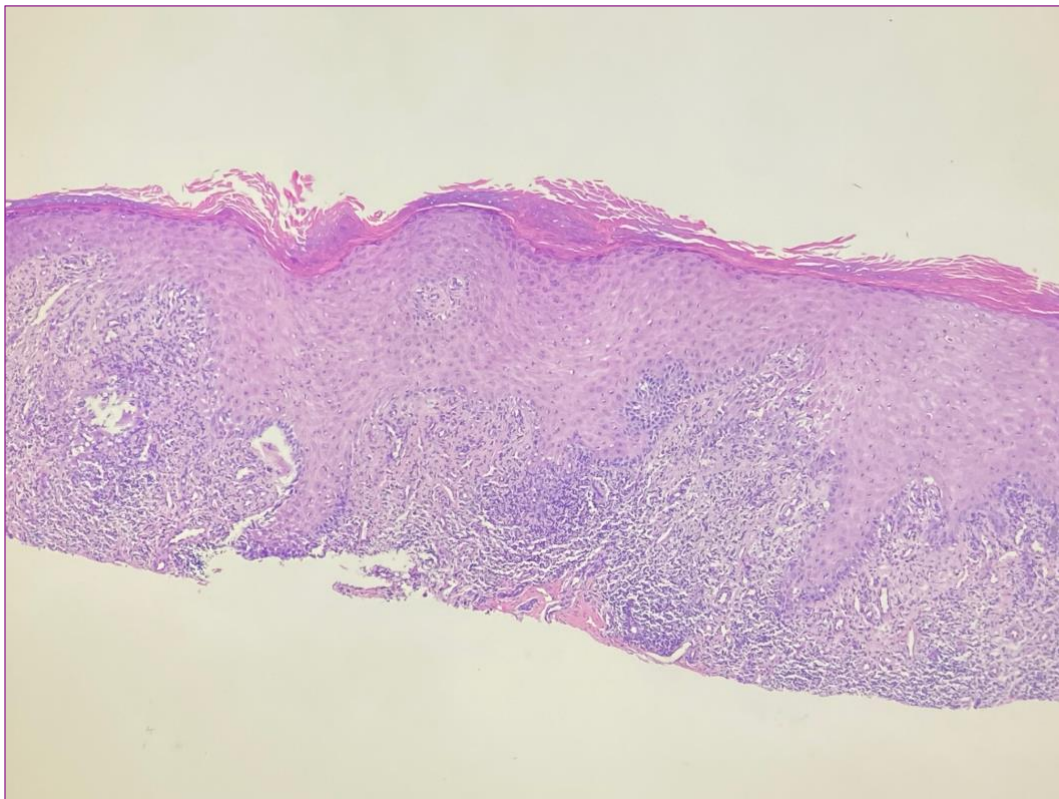


Figure 2. Representative hematoxylin and eosin stain of lesions skin.

lesion.² The Koebner phenomenon (KP) is a well-known occurrence in LP and involves the appearance of lesions at sites of cutaneous injury, trauma, stress, or stimulation.³ True KP occurs in psoriasis, vitiligo, and lichen planus.³ The incubation period varies both between patients and within the same patient depending upon the location, type, severity, and timing of injury.³ Its pathogenesis in LP is not completely understood, but has been attributed to mechano-transduction.³ Transient receptor potential ankyrin 1 (TRPA1) is a mechanosensory receptor which has been shown to be upregulated in oral lichen planus lesions.⁴ These lesions have a predilection for sites that experience mechanical trauma.⁵ Its role in modulating KP in other types of lichen planus has not been defined, however, this would be an interesting focus for future research.

The patient's history regarding recent procedures was integral in narrowing the diagnosis in this case. Although koebnerization is known to be associated with LP, the formation of HLP lesions following a vascular access procedure is not well documented in the literature. In this case, the patient's procedure triggered his quiescent LP via koebnerization. This case highlights the heterogeneous nature of LP presentation and emphasizes diagnostic tools and patient history in aiding dermatologists in making a timely, accurate diagnosis. It also highlights the importance of considering LP in the differential after a vascular procedure.

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