

Clinical Image

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Lichen planus pigmentosus inversus: a rare clinical variant

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A 71-year-old Hispanic woman presented with an asymptomatic rash on her axillae and neck that had progressively developed over the 2 years before she presented in January 2022. She denied any changes in hygiene products, tight or restrictive clothing, and a history of previous contact allergies, and she denied any previous facial involvement. Her medical history was unremarkable for hepatitis or diabetes. Previous treatment with ketoconazole and triamcinolone from her primary care physician provided no improvement. A review of systems was unremarkable. The physical examination demonstrated hyperpigmented violaceous papules and plaques with central atrophy and fine scale in her axillary vaults, with similar appearing plaques affecting her lateral neck following lines of skin cleavage (Figures 1 and 2). No oral or nail involvement was noted. A shave biopsy was performed demonstrating increased epidermal hyperkeratosis, hypergranulosis, marked pigmentary incontinence, and superficial perivascular inflammation. Clinicopathologic correlation supported a diagnosis of lichen planus pigmentosus inversus (LPPI). The patient was prescribed topical tacrolimus ointment to apply daily but was lost to follow-up.

LPPI is a distinct variant of lichen planus pigmentosus (LPP) presenting as gray to brown macules, papules, and plaques favoring the intertriginous regions of the body [1]. As first described by Pock et al. [2], this clinical entity presented in White patients affecting flexural zones with significant sparing of sun-exposed areas. Our case highlights the unique presentation in skin of color involved in LPPI. In darker skin colors, the primary lesions appear dark brown, gray, or a blue-gray. In contrast, these lesions may appear dark red or violaceous in lighter skin tones. The



Figure 1: Hyperpigmented violaceous papules and plaques with central atrophy and fine scale of the right axillary vault.

pathogenesis of LPPI is comparable to LPP with cytotoxic effects of CD8⁺ T lymphocytes targeting basal keratinocytes [3]. A biopsy is warranted for diagnosis because this must be histopathologically correlated to differentiate between other clinical entities: contact dermatitis, erythrasma, acanthosis nigricans, axillary granular parakeratosis, and erythema dyschromicum perstans (EDP) [4, 5]. Among these entities, EDP or ashly dermatosis can be difficult to clinically distinguish from LPPI. EDP presents as ill-defined slate gray macules or patches favoring a symmetrical distribution of the trunk and proximal extremities. Ultimately, histology can help differentiate LPPI vs. other clinical mimickers with the following features: epidermal hyperkeratosis, hypergranulosis, focal lichenoid dermatitis, perifollicular involvement, moderate to severe inflammatory

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Figure 2: Hyperpigmented violaceous papules and plaques with central atrophy and fine scale of the left axillary vault.

cell infiltration, and pigmentary incontinence [5]. Given the distribution and histopathologic findings, EDP was ruled out. Topical and oral corticosteroids may be utilized for therapy, but some authors deemed this unsuccessful [1]. Topical tacrolimus may be prescribed as a preferred agent because of its minimal side-effect profile and improved clinical outcomes [1, 6].

Informed consent: The patient described in this report provided informed consent.

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