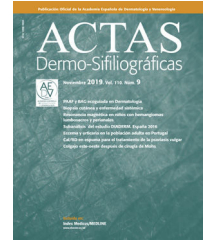




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RESIDENT'S FORUM

[Translated article] RF - Should Striae be Considered a Predisposing Factor for Koebner Phenomenon in Psoriasis? ☆



FR - ¿Deben ser consideradas las estrías un factor predisponente de Koebner en psoriasis?

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Psoriasis;
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PALABRAS CLAVE

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Koebner phenomenon is defined by the appearance of lesions corresponding to a given dermatosis in an area of previously healthy skin that has been subjected to trauma. It was first described in a patient with psoriasis by Heinrich Koebner in 1872.¹ Depending on the series, its incidence ranges from 11% to 75%.

In 1990 Boy and Nelder² proposed a system to classify the different Koebner phenomena, which are divided into 4 categories: true Koebner; pseudo-Koebner; occasional traumatic localization of lesions; and questionable trauma-induced processes. Only 3 diseases are included in the first category: vitiligo, psoriasis, and lichen planus.

We recently treated a 44-year-old man, with no personal history of interest, who had psoriasis for more than 30 years and was referred to our unit with ostraceous, erythematous–desquamative plaques in the axillae and groin (psoriasis area and severity index [PASI], 2.4; affected body surface area [BSA], 1.5) located on striae caused by multiple cycles of high-potency topical corticosteroid treatment (Fig. 1). Based on this characteristic location we investigated whether these striae should be considered a type of Koebner phenomenon.

We have only found 3 reports in the literature of striae (due to topical use of high-potency steroids and changes in weight and height) as a cause of Koebnerization.^{3,4}

The different forms of trauma that can cause true Koebnerization include mosquito bites, burns, abrasions, and grafts. The pathological changes of striae occur mainly in the extracellular components of the matrix (fibrillin, elastin, and collagen), resulting in flattening of the epidermis without modification of its structure. The process involves mechanical factors (microtrauma), and the underlying molecular mechanisms appear to involve mast cells and their inflammatory derivatives (tryptase and interleukin [IL]

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Figure 1 Ostraceous erythematous–desquamative plaques on distension striae in both groins.

6, IL-8, IL-17, IL-36 γ), and increased expression of nerve growth factor (NGF) and vascular endothelial growth factor (VEGF).⁵

The striae in our patient were mainly secondary to alterations in skin distensibility caused by weight change (i.e. true traumatic striae). Skin atrophy would be the expected local skin change in our patient after intensive use of high-potency corticosteroids. However, we believe it is important to consider these striae as a potential cause of Koebnerization in patients with psoriasis.

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