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Letter

Striae: a potential precipitating factor for Köebner phenomenon in psoriasis?

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Abstract

The Köebner phenomenon (isomorphic response) is defined as the development of typical lesions of a dermatosis that occur in areas of trauma in previously uninvolved skin. Rare reports have suggested that striae are a form of injury to the skin that can result in koebnerization of psoriasis, vitiligo, and lichen planus. We herein report a 27-year-old female patient with plaque psoriasis who developed psoriatic lesions along striae distensae.

Keywords: Köebner phenomenon, striae, psoriasis.

Case Synopsis

The Köebner phenomenon (KP), the isomorphic response, was first described in 1872 by Heinrich Köebner in a patient with psoriasis [1]. It is one of the most widely known phenomena in dermatology. KP refers to the development of isomorphic lesions in the traumatized uninvolved skin of patients who have certain skin diseases including psoriasis, vitiligo, and lichen planus (LP) [2]. In psoriasis, the reported incidence of KP varies from 11 to 75%. Koebnerization may be induced by various forms of trauma or external mechanical stimuli to the skin. These injuries are not only types that penetrate the epidermis and dermis, but also some types of non-penetrating blunt trauma, such as stretching, friction, compression, and vibration [2]. The Köebner response may also follow allergic or irritant reactions and some therapies or dermatoses. Rare reports have suggested that striae distensae and striae gravidarum can potentially induce KP in psoriasis, vitiligo, and LP (**Table 1**) [3-7].

Table 1. Studies supporting the role of striae distensae and striae gravidarum as potential precipitating factors for Köebner phenomenon in vitiligo, psoriasis, and lichen planus.

Study (year) [Ref.]	Patients (N)	Gender/ age (y)	Cause(s) of striae	Disease koebnerized
Lahiri & Sengupta (1996) [3]	1	M/28	Weight variation	Vitiligo
Lahiri & Malakar (2004) [4]	1	F/24	Pregnancy	Lichen planus
Verma (2009) [5]	4	F/27	Pregnancy	Vitiligo
		F/18*	Height/weight variation	Vitiligo
		F/?	Weight variation	Vitiligo
		F/?	Weight variation	Vitiligo
Verma (2009) [6]	5	F/18*	Height/weight variation	Vitiligo
		F/?	Weight variation	Vitiligo
		M/22	Weight variation	Psoriasis
		F/17	Height variation	Psoriasis
		F/33	Pregnancy	Lichen planus
Iftikhar et al. (2009) [7]	1	M/14	Height variation (puberty); use of systemic steroids (?)	Vitiligo
Our study	1	F/27	Weight variation; use of topical steroids	Psoriasis

*Same patient.

We present the case of a 27-year-old woman with obesity (93 kg; BMI: 31 kg/m²) and plaque psoriasis from the age of 10 years. At age 16 she developed hypertension that was treated with amlodipine 10 mg/day. Her psoriasis had previously been treated with topical steroids, vitamin D3 analogues, bethametasone/calcipotriol cream, emollients, several cycles of PUVA and UVB phototherapy, cyclosporine, and methotrexate, with unsatisfactory control. Since 2009, she has been receiving intravenous infliximab 5mg/kg at 0, 2, and 6 weeks (induction period), and every 8 weeks thereafter. As a result of her pre-infusion relapses, the interval between administrations was reduced to 7 weeks and methotrexate (7.5-15 mg/week) was added, with control of the disease. Since adolescence, she had tried several diet and weight loss programs without effective results. Because she experienced several weight changes during this period, she developed prominent striae distensae on the abdomen, lumbar area, and thighs. These measured up to 2 cm in width and 20 cm in length. Additionally, the prolonged use of topical steroids may have contributed to the striae development. At her last visit she complained about the appearance of psoriatic lesions appearing in the week before her infliximab infusion, mainly over the trunk. Upon physical examination, erythematous, scaly papules and plaques, measuring up to 2 cm in diameter were observed on the abdomen and flanks, precisely over the striae (**Figure 1**). Despite this relapse, the patient was satisfied with the results and no treatment other than the application of emollients and bethametasone/calcipotriol ointment was advised.

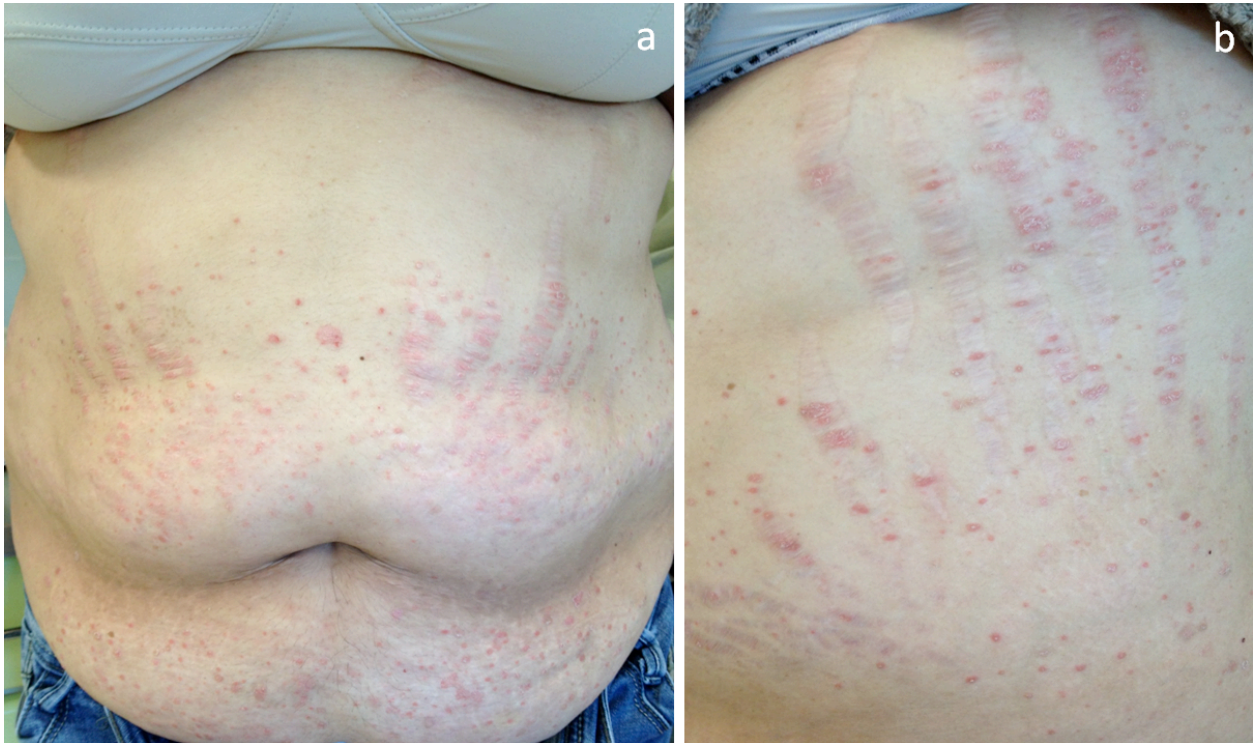


Figure 1. Clinical appearance of the patient. Note the presence of psoriatic lesions located along the stretch marks.

In agreement with prior reports from Verma [6], our case suggests that striae should be added to the list of potential inducers of KP in psoriasis. Distension of the skin and striae formation seem to act as a form of non-penetrating physical trauma, similar to a scar, which is a well-known site for the development of psoriasis. In fact, Arem and Kisher [8] have proposed that stretch marks are a form of dermal scarring in which the dermal collagen ruptures. The pathological changes of striae essentially occur in the components of the extracellular matrix, including fibrillin, elastin, and collagen, whereas the overlying epidermis remains unaltered except for some thinning and flattening [9,10]. Although there are no exact explanations for striae-induced KP, Verma [5] suggested some possible mechanisms, including imperceptible microscopic trauma to the epidermis during striae formation and alterations in dermal vascularity. Mast cell infiltrates, CD4+ T cells, and locally produced adhesion molecules may be involved.

Taking into account the higher prevalence and incidence of obesity in psoriatic patients [11] and the prolonged use of topical corticosteroids among these patients, is not surprising that striae distensae are a common finding in the psoriasis population. Therefore, although rare, striae-induced koebnerization may be more common in psoriasis than reported in the literature. More studies are needed for a complete understanding of this interesting entity.

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