

have found a statistically significant prevalence of diabetes mellitus⁶; and f) repeated trauma (Koebner phenomenon).³

The isomorphic response, also known as the Koebner phenomenon, consists of the appearance of typical lesions of a certain dermatosis in areas of healthy skin that have previously been subjected to different kinds of trauma. They are divided into 4 groups:

1. Category I: a true Koebner phenomenon, only seen with psoriasis, vitiligo, and lichen planus.
2. Category II: a pseudo-Koebner, which includes warts, molluscum contagiosum, and pyoderma gangrenosum.
3. Category III: dermatoses with occasional lesions in areas of trauma, such as Kaposi sarcoma, Darier disease, and erythema multiforme.
4. Category IV: A doubtful isomorphic phenomenon, which appears in diseases such as pemphigus vulgaris, eczema, or lichen nitidus.⁷

Lichen sclerosus et atrophicus is included in category III of the Koebner phenomenon. It has been associated with UV radiation, ionizing radiation, burns, venous hypertension (related to varicose veins), vulvovaginitis, pellagra,⁸ vaccines,⁹ repeated pressure, friction from clothing, trauma, and traumatic and surgical scars.¹⁰ Lichen sclerosus et atrophicus developing on herpes zoster scars (isotopic response) could be included in this group.⁵

In conclusion, the reason for reporting this case was to describe a patient suffering from perianal lichen sclerosus et atrophicus who developed lesions at the site of insulin injections as a result of a Koebner phenomenon, an association that we have not found elsewhere in the literature.

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Progressive Macular Hypomelanosis Successfully Treated with Topical Clindamycin and Benzoyl Peroxide

Hipomelanosis macular progresiva resuelta con peróxido de benzoilo y clindamicina tópicos

To the Editor:

Progressive macular hypomelanosis is an acquired skin pigmentation disorder that was initially thought to be related to racial characteristics, but is currently more often associated with the presence of *Propionibacterium acnes*.

A 23-year-old woman was seen for the progressive appearance over a 5-year period of asymptomatic whitish lesions on the trunk (Figures 1A and 2). The lesions were poorly defined, nondesquamating, hypopigmented macules tending to become confluent. The macules were initially located in the center of the chest but had spread centrifugally to affect the neck and the most proximal regions of the upper limbs. The lesions did not improve in summer, but rather tended to stand out more noticeably against the adjacent skin.

The patient had no history of atopic dermatitis or other eczematous disorders, and had never reported pruritus or desquamation of the lesions.

After other causes of acquired hypopigmentation, such as resolving pityriasis alba or pityriasis versicolor, had been ruled out clinically, the patient was diagnosed with

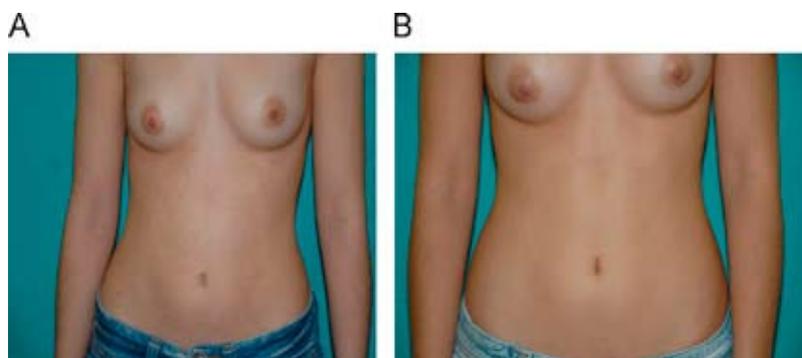


Figure 1 A, First patient before treatment. B, Complete resolution in the same patient 2 months after the application of a clindamycin and benzoyl peroxide gel.



Figure 2 First patient, detail view.

progressive macular hypomelanosis. Initially treatment was given with oral minocycline (100 mg/d) and topical clindamycin. When the lesions had not improved after 2 months on this regimen, the patient was switched to topical therapy with a combination formulation of clindamycin and benzoyl peroxide in gel. This new regimen led to the complete and permanent resolution of the lesions within 2 months (Figure 1B).

During the same period, a 20-year-old woman consulted for the same alterations (Figure 3). She was also diagnosed with progressive macular hypomelanosis but, unlike the earlier patient, was prescribed topical treatment with clindamycin without benzoyl peroxide from the outset. As in the earlier case, complete resolution was achieved.

Progressive macular hypomelanosis is an acquired skin pigmentation disorder found predominantly in adolescents and young adults. It is characterized by a symmetrical distribution of whitish macules that show progressive growth. Clinically, these lesions are very similar to those found in pityriasis alba and pityriasis versicolor during the resolution phase.¹ However, unlike those lesions, they are not associated with atopic dermatitis and there is no desquamation. This type of hypomelanosis usually affects the trunk, although the lesions may spread to the neck, face, buttocks, and proximal regions of the limbs.

Because it was first reported in areas of the Caribbean and because ultrastructural examination revealed stage II and III melanosomes (characteristic of white skin) in the hypopigmented areas and stage IV melanosomes (characteristic of black skin) in the areas of normal pigmentation,¹ it was originally thought that the etiology and pathogenesis of this condition were related to genetic and race-specific abnormalities.²⁻⁶ However, an infectious etiology was considered more probable after positive cultures for *P. acnes* were obtained from the areas of hypopigmentation and perifollicular red fluorescence was observed under Wood light in a dark room.⁷ In support of this hypothesis, some authors have proposed that *P. acnes* may produce a factor that interferes with melanogenesis, thereby giving rise to areas of hypopigmentation.⁸

The clinical course of the lesions is variable. While some authors think that they tend to stabilize and undergo spontaneous regression over a period of 2 to 5 years,⁶ others have suggested that the normal course is one of slow progression of the lesions over time.⁷

From among the available treatments, the following have not been shown to be effective: psoralen plus UV light therapy, systemic antifungal agents, and topical corticosteroids.⁹ In some cases, physicians have reported a good response to treatment with narrowband UV-B light.¹⁰ However, the therapy that appears to obtain the best results, including permanent repigmentation, is the combination of UV-A plus topical bactericidal therapy (benzoyl peroxide 5% and clindamycin 1%)⁹ or oral therapy with tetracyclines.¹

We believe that progressive macular hypomelanosis is more common in clinical practice than reports in the medical literature would suggest. The condition tends to be underdiagnosed because it is often confused with other disorders characterized by a very similar clinical picture, such as resolving pityriasis alba or pityriasis versicolor; however, unlike those 2 conditions, it resolves completely with topical and/or systemic antibiotic treatment. Based on descriptions in the literature, perifollicular fluorescence could aid diagnosis. We present 2 cases of patients with progressive macular hypomelanosis whose lesions resolved completely after 2 months of topical bactericidal treatment.

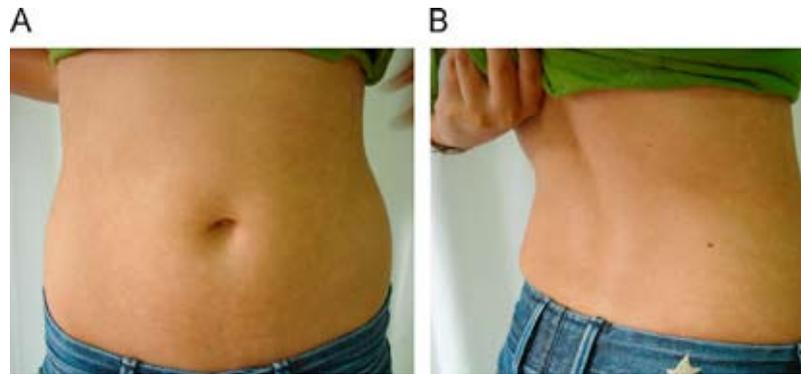


Figure 3 Hypopigmented lesions in the second patient. A, on the abdomen. B, on the flanks and in the lumbar region.

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Mixed Intraepithelial Lesion of the Vulva

Lesión intraepitelial combinada de vulva

To the Editor:

The vulva, as a site of contact with viral agents such as the human papilloma virus, may develop intraepithelial neoplasms such as Bowen disease (BD).¹ It can also be affected by extramammary Paget disease (EPD), given the high density of apocrine glands in the anogenital region.² There is also the possibility of a melanotic lesion developing on the vulva, or, more rarely, an intraepidermal adnexal tumor.

A mixed intraepithelial BD and EPD lesion of the vulva,³⁻⁷ as in the case we describe below, is rare.

We report the case of an 89-year-old woman, with an indurated lesion of the vulva affecting the left labia

majora and labia minora. The patient had no past history of relevance. A left hemivulvectomy was performed and our laboratory received a surgical specimen measuring 10 cm by 9 cm, with a maximum depth of 3 cm. A hardening of the tissue was evident in the area of the left labia majora and labia minora, occupying an area measuring 5 cm by 3.5 cm. On section, the lesion was found to be up to 9 mm in depth.

Microscopically, there were 2 clearly distinct epidermal areas. One area showed a marked distortion of the epidermal architecture, it not being possible to identify any of the normal epidermal layers because they had been replaced by clearly atypical cells with dense eosinophilic cytoplasm and a number of intercellular bridges (Figure 1). The lesion did not infiltrate the dermis at any point. The other area had a completely different appearance; maturation of the epithelium was unaltered, but there were scattered atypical cells with clear vacuolated cytoplasm (with mucin) and very prominent nucleoli (Figure 2).