Table 1 Description of the Published Cases of Erythema Multiforme-like Bullous Pemphigoid.

	Sex	Age, y	Suspected Etiologic Factor	Site of the Lesions	Mucosal Involvement	Involvement of the Palms and Soles
Alian et al.[4]	Female	36	Orf virus	Trunk, limbs	No	No
Park et al.[5]	Female	80	Amlodipine	Trunk, limbs	No	No
Hirano et al.[6]	Female	80	Furosemide	Face, neck, flexor surface of the limbs, trunk	No	No
Hayakawa and Shiohara[7]	Male	72	None	Extensor surface of the limbs, back, buttocks	No	No
Mehravaran et al.[8]	Female	73	Citalopram Thioridazine Flupentixol	Flexor surface of the limbs, neckline	No	Yes
Alcalay et al.[9]	Male Male Female	23 16 50	Amoxicillin Penicillin G procaine Penicillin	Face, neck, trunk, limbs	Yes (oral, nasal, anogenital, ocular)	Yes

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Sclerosing Nevus With Pseudomelanomatous Features: A Case Report☆

Nevus esclerosante con rasgos seudomelanomatosos

To the Editor:

In 2008, Giuseppe Fabrizi et al.[1] were the first to describe a subgroup of lesions with distinct histopathologic characteristics among all the melanocytic nevi excised for clinical regression; these lesions were given the name of sclerosing nevus with pseudomelanomatous features (SNPF).

We present the case of a 44-year-old woman who was seen for persistent pruritus in the area of a nevus on her back. The variegate maculopapular lesion measured approximately 1 cm in diameter and had irregular borders. On dermoscopy, an atypical globular pattern and a negative network were observed, with red, white, and occasional bluish areas (Fig. 1).

The lesion was evaluated using reflectance confocal microscopy (RCM), which showed marked destructuring of the epidermis, with irregularly shaped keratinocytes instead of the typical honeycomb or cobbled appearance, the presence of pagetoid cells with a multifocal distribution and dendritic morphology, a loss of bright rings, the absence of a defined pattern at the dermoepidermal junction, the

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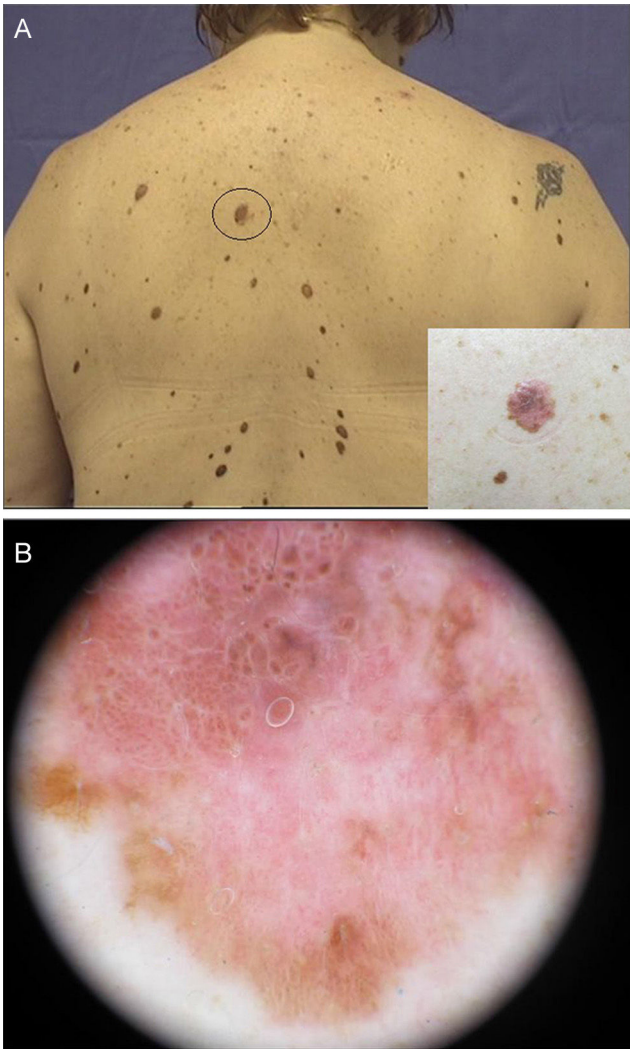

Figure 1 Clinical and dermoscopic appearance. A, Variegate pigmented lesion with irregular borders measuring approximately 1 cm in diameter. B, Atypical globular pattern, with a red, white, and blue negative network.

presence of atypical cells forming junctional and dermal nests, and a dense inflammatory infiltrate with fibrosis and abundant melanophages (Fig. 2). With a suspected diagnosis of melanoma, the lesion was excised.

Histopathology revealed an atypical proliferation of melanocytes at the dermoepidermal junction, with occasional pagetoid spread in the epidermis, an area of scar tissue, melanocytic nests with a morphology similar to that described at the dermoepidermal junction, and a residual nevus with a congenital pattern, both peripherally and deep to the scar tissue. The cells were not frankly atypical and, after detailed examination, no mitotic figures were identified (Fig. 3). The proliferation index was very low and was limited practically to the junctional component. The cells of the irregular nests and of the residual nevus component expressed Melan-A and p16, with a loss of expression of HMB-45 in the dermal component. The lesion did not reach the borders of resection. This histologic image was consistent with a sclerosing nevus with pseudomelanomatous features. With this diagnosis, no additional treatment was performed, and the patient remains on follow-up.

SNPF, a recently described clinical and pathologic entity, is also known as nevus with florid fibroplasia.[2] It is considered to mimic melanoma both clinically and histologically.[3] Etiologically, this lesion appears to be a benign melanocytic nevus that becomes involved in a process of fibrosis combined with a pseudomelanomatous proliferation. It typically arises in young individuals, mainly on the back, particularly in the area of the scapula. This site is thought to be affected because of almost imperceptible microtrauma or inflammatory changes in the region, such as the chronic friction of clothing, sunburn, seborrheic eczema, or acne. However, other authors consider that this type of fibrosis may be something intrinsic to the maturation process of a dysplastic nevus.[2]

Dermoscopically it is characterized by signs of regression, affecting 10% to 50% of the lesion, in the form of white and blue scars, and an absence of other specific signs of melanoma.[4]

RCM is a noninvasive technique with a resolution very similar to conventional histology. It provides horizontal images

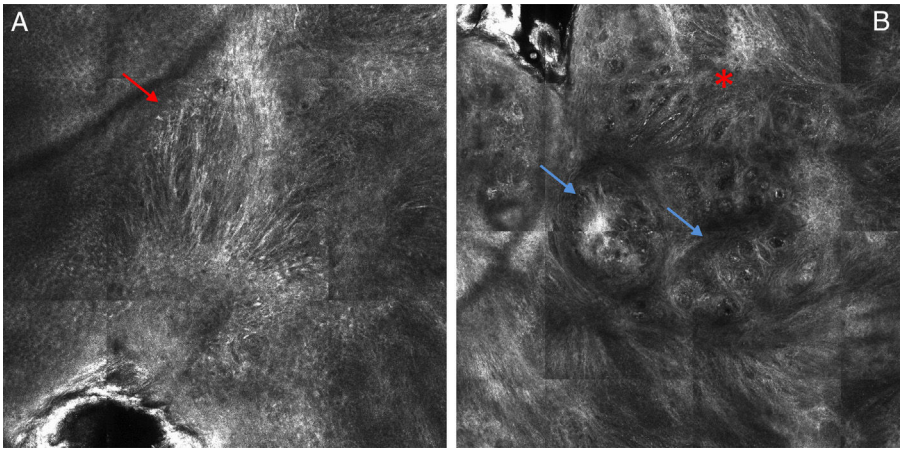

Figure 2 Confocal microscopy. A, Focus of abundant dendritic cells in the epidermis (red arrow). B, Areas of poorly defined papillas, with atypical junctional and dermal nests (blue arrows), areas of fibrosis, and an inflammatory infiltrate (asterisk).

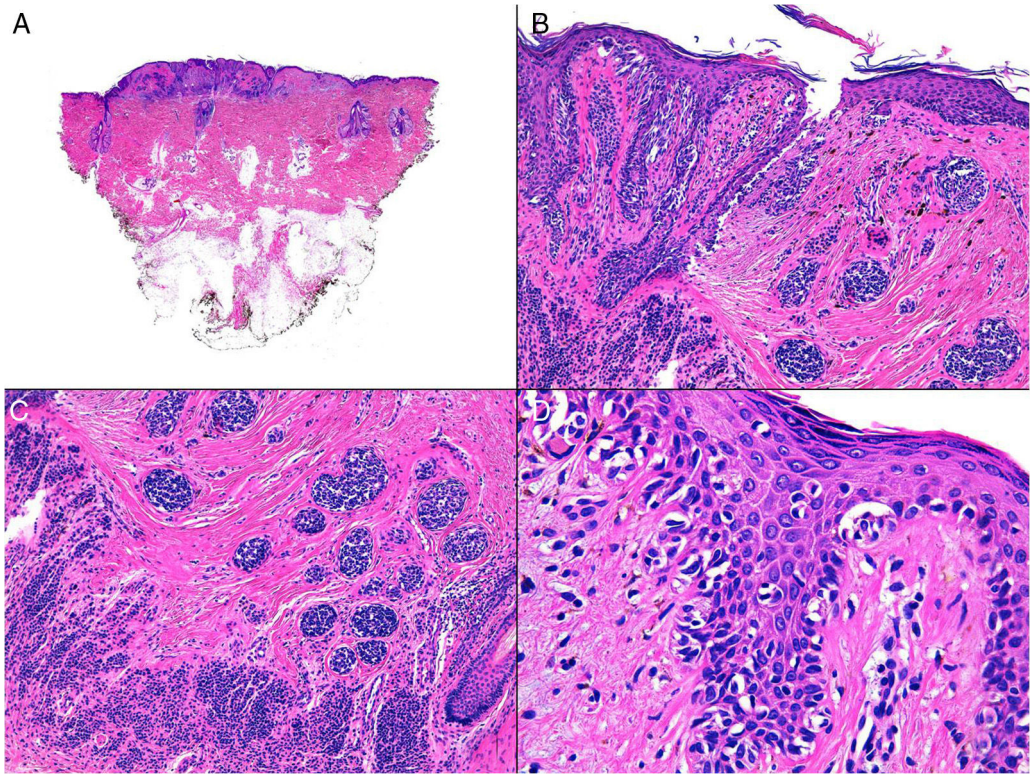

Figure 3 Histology. A, Lesion with a 3-zone pattern. Hematoxylin and eosin (H&E), low-power view. B and C, Proliferation of atypical melanocytes at the dermoepidermal junction with an area of scarring, nests of atypical melanocytes, and a congenital-type residual nevus both peripheral and deep to the scar. H&E, original magnification ×100. D, Scattered areas of pagetoid spread of melanocytic cells in other areas of the epidermis. H&E, original magnification ×400.

and can be considered an intermediate diagnostic method between dermoscopy and histopathology, frequently avoiding unnecessary surgical excisions. However, the presence of cellular atypia on RCM study in these cases means that a diagnosis of melanoma cannot be ruled out. As is to be expected, abundant melanophages and collagen bundles are also observed.[5]

Histologically there are 3 zones: an atypical proliferation of melanocytes at the dermoepidermal junction, with lentiginous hyperplasia, and confluent junctional nests with occasional pagetoid spread; a significant area of dermal sclerosis that contains irregular nests of atypical melanocytes; and a congenital-type residual nevus adjacent to the deep surface of the scar.[1] The low level of cellular atypia and the absence of mitoses, cell necrosis, or spreading dermal nodules differentiates this lesion from regressing melanoma. In addition, SNPF usually has an ''ordered'' pattern of fibrosis, with homogeneous bundles of parallel eosinophilic collagen fibers closely related to the epidermis. In contrast, a regressing melanoma is characterized by fibrosis that is often paler (perhaps because of edema), formed of more irregular collagen bundles, and the presence of melanophages. The diagnostic criteria of Fabrizi et al. probably make it possible to differentiate SNPF from a regressing melanoma. However, atypia in the junctional region associated with pagetoid spread makes diagnosis of this entity a question of the quantitative presence of morphological criteria, and a degree of interobserver variability between pathologists should therefore be expected.[3]

Taking into account the benign biological behavior of this entity, some authors consider that a more conservative approach could be warranted in melanocytic lesions showing regression on the convex area of the back.[4] Those authors propose observation. However, other authors recommend surgical excision, as SNPF mimics melanoma clinically, dermoscopically, and on RCM.[5] Only histology can give us the definitive diagnosis.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Allergic Contact Dermatitis Due to Capryloyl Salicylic Acid☆

Dermatitis alérgica de contacto por ácido capriloil salicílico

To the Editor:

When cosmetic allergy is suspected, it is essential to include the personal care products used by the patient in the patch tests as not only will this facilitate a more exact diagnosis, but also it may make it possible to identify new allergens. The collaboration of the pharmaceutical and cosmetics industries is fundamental to this approach, as it is those companies that will have to supply us with the components of the specific products. We present a case of allergic contact dermatitis to capryloyl salicylic acid present in an antiwrinkle cream. A 40-year-old woman consulted for a pruritic erythematous rash that arose on her face 10 days after starting to apply an antiwrinkle cream (Revitalift Laser X3 Day Cream by L'Oreal). The condition resolved after interruption of the use of the cream and a week of treatment with a topical corticosteroid. Patch testing was performed using the standard series of the Spanish Contact Dermatitis and Skin Allergy Research Group (GEIDAC), a cosmetics series (Chemotechnique, Sweden), and the specific cream used by the patient, with readings taken at 48 and 96 hours, in accordance with the recommendations of the European Society of Contact Dermatitis (ESCD). In the final reading at 96 hours, a positive reaction (++) was observed to the specific cream used by the patient and negative reactions to the other allergens in the standard and cosmetics series. We contacted the company that marketed the cream involved and they provided us with the 27 ingredients of the cream, thus enabling us to complete the study. A positive allergic reaction (++) to 1% capryloyl salicylic acid in alcohol was observed in the readings taken at 48 and 96 hours (Fig. 1). This same substance showed no allergic responses after patch testing in 15 healthy controls.

Capryloyl salicylic acid (5-capryloyl salicylic acid, CAS no. 78418-01-6, also known as 2-hydroxy-5-octanoylbenzoic acid) is a lipophilic derivative of salicylic acid that promotes epidermal renewal, stimulates collagen formation, combats chronic actinic photodamage,[1] and increases skin resilience to UV radiation. It is attributed comedolytic,[2] antiacne,[3] antibacterial, and anti-inflammatory[4] properties. This substance is widely used, mainly by the French cosmetics industry, and it can be included in facial emollients, antiaging formulations, products for the treatment of acne, sunscreens, and facial hygiene products.[5]

De Groot et al.[5] recently published the first 2 cases of contact allergy to capryloyl salicylic acid, using 1% capryloyl salicylic acid in alcohol in the patch testing of their patients. According to those authors, the allergenicity of this substance may derive more from the salicylic or benzoic part of the molecule that from the caprylic acid fraction. However, Roberts et al.[6] concluded that 5-capryloyl salicylic acid was probably not the agent responsible for the allergy of the patients described by de Groot et al.; in their opinion, the allergy was due to its 3-capryloyl salicylic acid isomer, a contaminant of 5-capryloyl salicylic acid.

We have described a third case of allergic contact dermatitis to capryloyl salicylic acid, the diagnosis of which was made possible by patch testing the ingredients provided by the company marketing the cream used by the patient, as these substances are not included in other test series. The description of further cases in which capryloyl salicylic acid is implicated will clarify the allergenic potential of this substance.

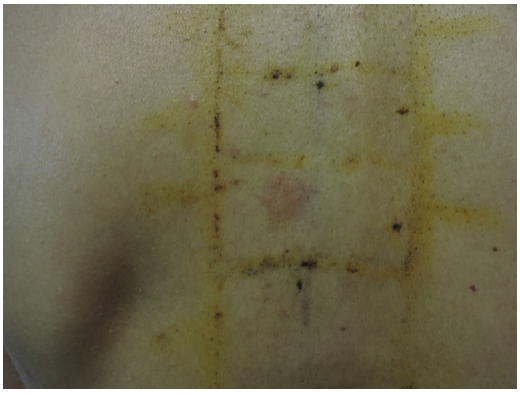

Figure 1 Positive allergic reaction to capryloyl salicylic acid.

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