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RESEARCH ARTICLE

PEMPHIGUS VULGARIS COMPLICATED BY KELOID SCARS

Belcadi Jihane MD¹, Oulad Ali Sara MD¹, Benaich Dounia MD², Znati Kaoutar MD², Ismaili Nadia MD¹, Benzekri Laila, MD¹, Senouci Karima MD¹ and Meziane Marieme MD¹

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Rabat, Morocco.

- 1. From the Department of Dermatology. and
- 2. Department of Histopathology, Mohammed V University in Rabat, Ibn Sina University Hospital, Morocco.

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Abstract

Pemphigus vulgaris is an autoimmune bullous disease characterized by the formation of intraepidermal bullae⁵. These bullae eventually rupture, leaving erosions that rapidly re-epithelialize, sometimes into hyperpigmented macules and exceptionally into keloid scars. We report the case of a patient with pemphigus vulgaris who developed keloid scars.

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Introduction:-

Pemphigus vulgaris is an autoimmune bullous disease characterized by the formation of intraepidermal bullae⁵. These bullae eventually rupture, leaving erosions that rapidly re-epithelialize, sometimes into hyperpigmented macules and exceptionally into keloid scars.

We report the case of a patient with pemphigus vulgaris who developed keloid scars.

Observation:-

A 53-year-old female patient with a history of a pemphigus vulgaris outbreak in May 2020, involving 30% of the skin surface, with post-bullous erosions covered with yellowish crusts affecting the face, trunk, buttocks and thighs. Erosive and fumigating cheilitis and multiple endo-jugal erosions were also present. Cultures revealed the presence of Staphylococcus aureus and appropriate antibiotic therapy was initiated.

The patient was treated with corticosteroid therapy at a rate of 1 mg/kg/d with good evolution and complete reepithelialization of the lesions after 6 weeks.

Two years later, after stopping the corticosteroid therapy, the patient reconsulted for unsightly and pruritic scars.

The examination revealed erythematous, sclerotic, poorly limited plaques in a phototype 3 patient, spread over the previous sites of pemphigus, namely the sub-breast area, the back and the buttocks (Figure 1). In addition, no other healing abnormalities were observed, i.e., no milium grains.

The patient was put on very strong class dermocorticoids with good evolution.

Corresponding Author:- Belcadi Jihane

Address:- Department of Dermatology Mohammed V University in Rabat, Ibn Sina University Hospital, Morocco.



Figure 1:- Extensive postbullous erosions of the gluteal region (A) that evolved into keloid scarring (B).

Discussion:-

The formation of keloid scars following pemphigus is exceptional.

Because the process leading to deep pemphigus is largely confined to the epidermis, lesions usually evolve as hyperpigmented macules but without scarring. Only 3 case reports of keloids forming after pemphigus vulgaris have been reported.

In 1997, Khanna and al.³ described keloid scars arising on uninfected pemphigus vulgaris erosions. Later, Sako and Workwick² and Nguyen and al.¹ reported on two patients who developed keloids shortly after the onset of the disease against a background of bacterial skin infection for the first case and herpes for the second, sometimes even on sites not affected by the disease. However, the paucity of cases of keloids following pemphigus vulgaris flares contrasts with the frequency of superinfections which are common in pemphigus, suggesting the presence of other unknown factors.

In our patient, we assume that the initial delay in healing favored by cachexia and hypoprotidemia coupled with the secondary infection of her pemphigus erosions led to dermal lesions, triggering a keloid reaction⁴ in this patient who, due to her phototype and the location of her lesions, was not predisposed to develop them.

Conclusion:-

The cause of keloid scar development in our patient was probably bacterial infection of her pemphigus lesions. It is plausible that superinfection led to intradermal inflammation and the formation of these scars.

This case demonstrates that, despite appropriate treatment, deep pemphigus can lead to extensive scarring in some patients, hence the interest in investigating and treating any superinfection occurring during this bullous dermatosis.

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