Multiple milia formation in blistering diseases

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Background

Milia are superficial keratinous cysts, clinically seen as pearly white, dome-shaped lesions 1 to 2 mm in diameter. Milia are associated with diseases that cause subepidermal blistering, such as hereditary forms of epidermolysis bullosa, epidermolysis bullosa acquisita, bullous pemphigoid, bullous lichen planus, and porphyria cutanea tarda. Multiple eruptive milia are rare and more extensive in number than primary milia.

Objective

The objective of this study was to search the literature for cases of blistering diseases with multiple milia formation, especially in areas of the skin where there was no evidence of blistering or trauma, and review the interpretations of their pathogenesis.

Methods

We performed a literature search with the terms multiple / numerous milia and bullous diseases /blistering diseases / pemphigoid / epidermolysis / lichen planus.

Results

Very few studies have investigated the origin of milia. Primary milia are thought to originate from the sebaceous collar of vellus hairs (lower infundibulum), whereas secondary milia are believed to derive from eccrine ducts more commonly than from overlying epidermis, hair follicles, or sebaceous ducts. Milia secondary to blisters or traumas are speculated to be produced through the regeneration process of disrupted sweat glands or hair follicles. Immunological predisposition, aberrant interaction between the hemidesmosomes, and the extracellular matrix components beneath the hemidesmosomes have been described with regard to the formation of numerous milia during recovery, especially in cases of bullous pemphigoid with IgG autoantibodies to LAD-1 and/or the recombinant protein of BP180 C-terminal domain. Multiple milia could be a primary manifestation of dystrophic epidermolysis bullosa in skin areas without evidence of blistering.

Conclusion

The exact etiology of multiple milia remains unknown. Immunologic predisposition and improper interaction between hemidesmosomes and extracellular matrix components are speculated in the formation of milia during recovery from bullous lesions in blistering diseases. Still, further studies on the triggering mechanisms of keratinocyte dysfunction in cases of multiple milia formation without evidence of prior blistering are needed.

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Neuropeptidergic acral dysesthesia

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Background

Neuropeptidergic acral dysesthesia (NAD) was described for the first time in 1993 as an entity characterized by the increased number of neuropeptidergic fibers and high levels of neuropeptide storage in the affected acral skin. Clinical symptoms completely disappear with capsaicin therapy.

Case report

A 56-year-old Caucasian male patient was examined at our Institute of Dermatology in Florence, Italy, with an 18-year history of intractable itching and burning discomfort on the dorsum of the hands and feet. All local and systemic treatments with potent corticosteroids, antihistamines, and antiinflamatory agents did not bring any benefit to the patient. Systemic antidepressants and antipsychotic drugs were tried by psychiatrists without results. At the inspection visit, the patient showed normal skin (according to patient age) on the dorsum of the hands and feet. Upon closer inspection, long-lasting linear and non-linear irregular scars were observed (see pictures 1 and 2). The patient underwent blood analysis for chronic inflammatory and autoimmune skin diseases, which gave negative or normal results. A psychiatric evaluation was not contributory. An allergologic investigation, including patch and prick tests and photo-patch test, gave negative results. According to similar cases first described in 1993, we treated the patient with capsaicin cream 0.1% for 3 days and 1% capsaicin cream for the following 30 days (6 times/day). After 7 days of initial burning discomfort, all symptoms completely disappeared. After 33 days, the treatment with capsaicin cream was interrupted. The patient reported a symptom-free period of 9 days, and immediately after a recurrence of the burning and itching discomfort in the lesional areas appeared. Local treatment with capsaicin cream 1% again induced total remission of the symptoms.

Conclusion

We diagnosed this case as a typical case of NAD based on to the clinical presentation and response to local vanilloid/capsaicin cream treatment. NAD is considered a form of localized abnormal functioning of A delta and C neuropeptidergic fibers of the skin, which appear sensitive only to vanilloid/capsaicin local treatment.

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Hospital-based prospective study of the clinical and epidemiologic profile of adult female acne in Lagos, Nigeria

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Background

Adult female acne vulgaris is increasingly becoming a common presentation in dermatology clinics. Adult female acne is classified as either late onset, persistent acne or return acne with a mixed pattern of inflammation in most women.