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Not all scabies come with a… burrow

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Dear Editor,

Bullous scabies is one of the most rare variant of scabies. Scabies is an itchy skin disease caused by *Sarcoptes scabies var. hominis*. Recently, it was included in the World Health Organization roadmap for neglected tropical diseases from 2021 to 2030. The prevalence of scabies is very high in poor populations causing elevated mortality rate. Data regarding prevalence reported about 200 million cases of scabies worldwide in 2015.[1]

The infestation comes from direct skin to skin contact, including sexual contact or as indirect transmission *via* devices like towels, bed lines and clothes. The survival of the acarus is related to environmental factors such as temperature and humidity.[2]

The contact with the skin triggers the secretion of saliva, which breaks down the stratum corneum. The mites then move in a slow, tortoise-like pattern, creating characteristic tunnels known as *cunicoli*. Female mites subsequently lay eggs, and the life cycle of the mites lasts approximately 4 to 6 weeks. Pathogenetic mechanism are not specifically known; we know that host response is related to both immediate type-I response followed by type-IV hypersensitivity causing both inflammatory and allergic response with itch that occurs specifically at night.[3]

Macroscopically, lesions occur within 4 to 8 weeks after primary infestation by *Scarcoptes mites*. Clinical presentation is both characterized by *cuniculi* (ordinary scabies), nodular eczematous lesions (nodular scabies), highly irritating flaccid or tense bullae (bullous scabies) and crusts adherent to underlying tissues (crusted scabies). The bullous variant is more rare and a recent literature review on PubMed from 2001 to 2021 only identified 28 case all over the word.[2]

Different diagnosis includes immune bullous pathologies such us pemphigus and pemphigoids; it is often mandatory to perform systemic auto-antibodies to exclude pemphigoid (anti-BP180 and anti-BP230) and pemphigus (anti-DG1 and anti-DG3) and local biopsy with immunofluorescence (scabies is positive for IgM and C3 instead of immune bullous pathologies that show positivity for IgG, C3, and local antibodies such as in blood examination).

First-line treatment includes permethrin 5% cream, oral ivermectin (200 micrograms/kg), or benzyl benzoate 10-25% lotion. Recent studies confirm that mites can develop resistance to topical permethrin or systemic ivermectin due to mutations in drug receptors. Environmental interventions are crucial for eradicating mites, with high humidity being a key factor in killing scabies mites.[1]

A 73-year-old man entered the emergency and the dermatology departments with a diffuse papular rash associated with bullae (both tense and flaccid ones) localized predominantly in brachial, abdominal, inguinal, and gluteal areas (at time 0). The patient reported intense itching, particularly at night; he showed diffuse scratching lesions all over the body. Medical history only showed biliary

calculosis with normal biliary acid in blood and no systemic pathology; he referred only recent episode of typical scabies 4 months before, resolved after both systemic and topical therapy.

When he entered the dermatological department dermoscopy tests were negative for typical signs of scabies, blood examination was negative for both routine and specific exams for immune bullous pathologies. Recent treatment with systemic steroids was performed in another hospital (suspecting pemphigoid) and resulted in only temporary improvement, without clinical resolution. A cutaneous biopsy was necessary to perform differential diagnosis.

Histological examination revealed blisters with dermo-epidermal detachment, angiomatous-like hyperplasia of the papillary dermis, and associated lymphocytic inflammation extending into the medium-deep dermis, while the deeper dermal layers remained within normal limits. Immunofluorescence testing showed negative results for IgG, IgA, and C4, in contrast to the typical positivity for IgM and C3, with focal sub-epidermal positivity. Testing for both local and systemic BP180 and BP230 antibodies was negative, effectively excluding the diagnosis of pemphigoid. Consequently, both systemic and topical steroid treatments were ineffective.

Subsequently, the pruritic symptomatology was also observed in other members of the family in whom burrows were highlighted with dermoscopical examinations. Upon further careful clinical and dermoscopic examination rare burrows were highlighted even in the patient.

The diagnosis of scabies was straightforward, and the patient, along with their family members, was treated with systemic ivermectin (200 μg/kg orally) and topical benzyl benzoate 25% lotion, applied in two sessions. Clinical resolution was achieved after administering the treatment on days 0, 7, and 14. Furthermore, the patient did not experience any new blisters within three months following the therapy.

The diagnosis of bullous scabies represents a clinical challenge for dermatologists. It represents a rare variant of scabies, but incidence is going to increase rapidly in poor populations. Our patient presents an atypical case, where the typical signs of scabies were absent. A thorough clinical and anamnesic evaluation of family members with similar symptoms was essential to reach the correct diagnosis.

A cutaneous biopsy can be useful primarily for excluding the diagnosis of autoimmune bullous diseases, especially in cases where C3 and IgM are positive, while IgG and immunofluorescence (anti-BP180 and BP230 antibodies) are negative.

We can conclude that “not all scabies come with a… burrow” deliberately inspired by a famous and traditional Italian saying.

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Figure 1. Clinical presentation at time 0 before therapeutic goals.