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Case Report

Scabies Norvegica in infant

Mergita Ferizi¹, Antigona Gercari² and Mybera Ferizi^{2*}

¹Center of Medical Health in Prishtina, Kosovo

²University Clinical Center of Kosova, Department of Dermatology Prishtina, Kosovo

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*Corresponding author: Mybera Ferizi, University Clinical Center of Kosovo, Department of Dermatology, Rr. Spitalit p.n. 10000, Pristina, Kosovo, E-mail: mybera.ferizi@uni-pr.edu

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Abstract

Introduction: Crusted scabies are a rare manifestation of scabies characterized by uncontrolled proliferation of mites in the skin. Crusted scabies are a severe variant of highly contagious scabies. Various cutaneous, neurologic and immunologic diseases have been described to predispose to crusted scabies.

Case presentation: The presented case is a 7-month-old male. He was hospitalized at the Clinic of Dermatovenerology, due to hyperkeratotic changes in the palms and food, papules throughout the body, and nail changes all of these accompanied by scratching. Initially, it was diagnosed as atopic dermatitis, hyperkeratosis palmoplantaris, and onychomycosis mani. The changes had started to appear five months ago. Also, the mother of the child presents the papules throughout the body, also associated with itching as classical scabies.

Conclusion: Crusted scabies are a severe variant of highly contagious scabies. Is known the failure of the immune system to suppress the proliferation of the mite is an important cause of crusted scabies development. Crusted scabies usually do not present as an acute eruption as in classical scabies. The eruption is slow in onset and insidious in progression. In the present case, we learned that in the stimulation of the condition, there was a major role in the application of topically fluoride steroids.

Introduction

Scabies is an infestation of the top layer of skin caused by the parasite, *Sarcoptes scabiei*, often called scabies or mites. The mite is an obligate parasite that lives in burrowed tunnels in the stratum corneum [1]. In the case of crusted scabies, the number of mites is astronomical because of uncontrolled proliferation [1].

The failure of the immune system to suppress the proliferation of the mite is an important cause of crusted scabies development. Crusted scabies typically develop in patients with a defective T-cell immune response or decreased cutaneous sensation and reduced ability to mechanically debride the mites [2]. Crusted scabies are characterized by hyperkeratosis and crusting of the skin due to the profuse proliferation of mites resulting from an altered host response to the infestation [3].

Various cutaneous, neurologic and immunologic diseases have been described to predispose to crusted scabies [2]. There is a wide range of presentations of Norwegian scabies in people with HIV with lesions ranging from thick, crusted plaques to red papules to psoriasiform plaques to hyperkeratotic yellow papules [2,3]. The lesions in Norwegian scabies are classically distributed on the extremities but are frequently found on the back, face, scalp, and around the nail folds [4]. As Norwegian scabies are extremely infectious, early diagnosis is paramount to allow prompt therapeutic interventions and infection control [5].

Recent studies have shown that skin-homing cytotoxic T cells contribute to an imbalanced inflammatory response in the dermis of crusted scabies lesional skin. Transmission via fomites is more commonly seen in crusted scabies [2]. The immediate environment of a patient with crusted scabies is heavily infected with mites [1].

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Patients with crusted scabies are capable of triggering an epidemic of scabies [6]. Diagnosis is based on clinical findings and the demonstration of the mite. Microscopic examination of the skin scrapings from the patient is essential to demonstrate the mites [6]. The specimen from the crusted lesion is scraped with a blunt scalpel and placed on a glass slide. A drop of mineral oil and a cover slip are placed on it. The microscopic examination reveals numerous mites, eggs, and mite feces (scybala) [7].

Children are an underappreciated source of infection [8], for a number of reasons: scabies infections in children are often not detected early [9] or treated thoroughly enough [10], probably involve a greater number of mites and are more likely to be passed on through close physical contact with other persons [11].

Hyperkeratosis of the skin, which is a prominent feature in crusted scabies, is probably related to increased levels of interleukin-4 [12]. Crusted scabies also develop in Australian aborigines with normal immunity [13].

Case presentation

The presented case is a 7-month-old male. He was brought to the outpatient department in the Dermatological Clinical Center by his mother with a five-month history of the skin condition had worsened rapidly and there were extensive, generalized, thick, hyperkeratotic, crusting, yellowish papule lesions that eventually disseminated across the palms and foot, papules throughout the body and nail changes all of these accompanied by scratching (Figures 1–10).

The child had been born after a full-term normal pregnancy and was still breastfed. From the mother, we learn that the mother and other family members have changes in the skin associated with itching. The baby's treatment has been done with various lotions for atopic dermatitis (corticosteroid



Figures 1,2: Physical findings: fissured hyperkeratotic lesions on both hands



Figure 3: Papulle in the face.



Figure 4: Hyperkeratotic lesions on both feet.



Figure 5: Papulles in the child's corpus.



Figure 6: Nails infected with parasites.



Figure 7: Multiple Papulles in the child's corpus.

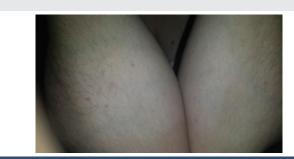


Figure 8: Papullae in the hands of the mother.

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ointment) and the condition is worsening, and were forced to lie in a research clinic. The mother and other family members have used therapy for scabies, but the baby does not. And the changes have improved but they have emerged again.

Based on the heteroanamnesis from her mother, clinical presentation, the microscopic examination reveals numerous mites, eggs and mite feces (scybala) is diagnostic of scabies (Figure 11).

The patient was initially diagnosed with atopic dermatitis, psoriasis, ichthyosis, seborrchoeic dermatitis, erythroderma, or Langerhans cell histiocytosis. However, to dermatologists' doubts, crusts were assembled at 10% KOH preparation and observed on low and high-energy targets where it was diagnosed with Norwegian scabies. Laboratory tests were at normal limits. Test for HIV infections is negative for child and mother.

The treatment is based on Sulfur ointments or benzyl benzoate, which are often used in the developing world due to their low cost [6], 10% sulfur solutions have been shown to be effective [14], and sulfur ointments are typically used for at least a week [6].

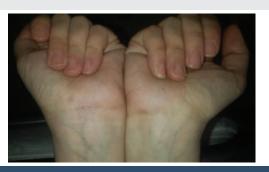


Figure 9: Papulles in the hands of the mother.



Figure 10: Papulles in the corpus of the mother.



Figure 11: Microscopic examination: low-power microscopic visualization of the mite Sarcoptes scabiei.

Discussion

Scabies are one of the three most common skin disorders in children, along with tinea and pyoderma. The disease may be transmitted from objects but is most often transmitted by direct skin-to-skin contact, with a higher risk with prolonged contact. Crusted scabies, formerly known as Norwegian scabies, is a more severe form of the infection often associated with immunosuppression Acropustulosis [4], or blisters and pustules on the palms and soles of the feet, are characteristic symptoms of scabies in infants [5]. The laboratory diagnosis of Norwegian scabies is simple, but clinical suspicion is required on the part of attending healthcare workers. Evidence has demonstrated the association between inappropriate use of topical corticosteroids and localized decreased cell-mediated immune responses, promoting the spread and proliferation of the mite in this kind of scabies [10,11]. On non-medical advice, our patient applied topical corticosteroids for seven weeks to treat pruritus, which exacerbated the mite infestation.

Conclusion

Crusted Scabies is the least frequent presentation of scabies, and there are few reports in the national and international literature. There is relatively little worldwide epidemiological data available concerning this specific infection type. It is necessary to suspect this presentation form of the disease and establish a timely diagnosis to detect and treat, as far as possible, associated comorbidities to avoid complications and reduce morbidity and mortality.

Authors' contributions

MF, AG, and MF were involved in the diagnosis, and literature review and helped draft the manuscript. All the authors read and approved the final manuscript.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Acknowledgement

The authors acknowledge the patient for providing consent for publishing this case report.

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